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A central focus of dental anthropological study over the last century or so has involved metric and non-metric analysis of the features of human teeth (Scott and Turner, 1977). The crowns of upper molar teeth have four main cusps and these are termed the paracone (mesiobuccal), protocone (mesiolingual), metacone (distobuccal), and hypocone (distolingual). Hypocone expression, like other non-metric dental crown traits, is generally scored by comparison with standardised plaques (Turner et al., 2001). These plaques aid visual assessment of presence and degree of expression. In the context of phylogeny, dental characters are associated with functional demands and dietary adaptations but also reflect the developmental processes controlling morphogenesis.

The field theory that was proposed by Butler (1939) and adapted by Dahlberg (1945) in an attempt to account for the common features of teeth within a class, postulated that the most mesial tooth in each morphological class is usually the most stable phenotypically. Osborn (1978), in his clone theory, proposed that a single clone of pre-programmed cells led to the development of all teeth within a particular class. Both of these theories provide insights into the mechanisms that may be involved in patterning within the human dentition. Recent progress in studying these mechanisms at a molecular level indicates the involvement of homeobox-containing genes (Mitsiadis and Smith, 2006). Recently, Mitsiadis and Smith (2006) and Townsend et al. (2008) have proposed a new genetic developmental model for teeth that incorporates the field, clone and homeobox code theories.

Strong genetic influence on hypocone expression of permanent maxillary molars in South Australian twins

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ABSTRACT An understanding of the role of genetic influences on dental traits is important in the areas of forensic odontology, human evolution and population variation. The aims of this study were: to calculate the frequency of occurrence and degree of expression of hypocones on permanent maxillary first and second molars in a sample of South Australian twins; to compare trait expression between males and females; to compare concordance rates for trait expression between monozygotic (MZ) and dizygotic (DZ) twins; and to fit genetic models to the data derived from twins and determine heritability estimates. Using stone dental casts, hypocone expression was scored on maxillary permanent first and second molars of 45 MZ twin pairs and 43 DZ pairs. Degrees of expression were scored from absence, through minor wrinkles or ridges, to very large cusps (score 0 – 5) using the standardized method of Turner et al. (1991). Hypocones were found to be more common and larger on first molars than second molars and there was a tendency for them to be larger in males although this was not statistically significant. No significant differences in occurrence or expression were noted between antimeres, with fewer differences observed between first than second molars. The percentage concordance for expression in MZ twin pairs was higher than in DZ twin pairs indicating a genetic influence determining the variation observed in hypocone expression. The most parsimonious model to explain observed variation was an AE model, incorporating additive genetic and unique environmental effects. Narrow-sense heritability estimates for both the first and second molars were high indicating that a large portion of the phenotypic variation could be explained by additive genetic effects. The greater range of phenotypic expression shown by the second molars compared with the first molars may reflect a common genetic liability that is modulated by differences in tooth size, location and/or developmental timing between these teeth. Dental Anthropology 2009;22(1):1-7.
enamel knots is believed to be influenced by members of the fibroblast growth factor family. Genes involved in cusp development appear to be the same among all the individual cusps, with no particular gene for a single cusp, which means that at the level of molecular signaling, all the cusps are alike. A patterned cascade mode of cusp spacing may promote the evolution of new cusps and individual teeth may differ only in the timing of cusp initiation (Jernvall, 2000). As the secondary enamel knot program is repeated for every cusp, any small difference in cusp spacing will have a cumulative effect on later-developing cusps (Jernvall, 2000). Reflecting this concept, studies have shown that hypocones show the greatest variation in size of all upper maxillary molar cusps in hominoid primates and in humans (Jernvall and Jung, 2000; Kondo et al., 2005).

Insight into the relative contributions of genetic and environmental factors to human tooth development can be gained from twin studies involving the comparison of monozygotic (MZ) and dizygotic (DZ) twin pairs. Differences between MZ twin pairs can be expected to be of similar magnitude to the minor left right differences that may be observed in singletons, whereas the differences between DZ pairs are similar to those seen in siblings (Kabban et al., 2001).

The purpose of this study was to investigate size variability of the hypocone of permanent maxillary first and second molars in a sample of South Australian twins. The specific hypotheses that were tested were:
• That hypocones occur more frequently and are larger in first molars compared with second molars
• That hypocones occur more frequently and are larger in males than females
• That hypocone expression is symmetrical between antimeric teeth
• That monozygotic twin pairs exhibit a higher degree of concordance for hypocone trait expression than dizygotic twin pairs, indicating a genetic contribution to observed variation.

MATERIALS AND METHODS

From a collection of dental casts of over 600 twin pairs, 45 MZ and 43 DZ pairs were selected. The twins were all of European ancestry and were aged between 10 and 46 years. Only individuals with permanent maxillary first and second molars present on both left and right sides were included. Subjects selected did not have any extensive restorations and their casts were not damaged. The study was approved by the Committee on the Ethics of Human Experimentation, University of Adelaide (Approval No. H/07/84) as part of an ongoing study of the teeth of Australian twins.

Hypocones were scored on right and left maxillary first and second molars using Turner’s ASU classification system (Arizona State University System, Plaque 8) (Scott and Turner, 1997) with 6 grades of expression. Score 0 represented absence of a cusp, score 1 indicated a ridge or wrinkle present at the cusp site, score 2 was a faint cuspule, score 3 was a small cusp, score 4 was a large cusp and score 5 was a very large cusp. The casts were examined under a magnifying light and the degree of expression was determined by reference to a plaster replica of the scoring plaque. Assessments were made for all subjects on two separate occasions so that concordance rates between determinations could be calculated. A second observer scored 30 randomly selected casts for determination of inter-examiner reliability.

Statistical analysis was carried out using SPSS for Windows®. Frequencies were calculated for right and left side teeth, and for males and females. Associations between sides, first and second molars, and sexes were tested using chi-square tests. Statistical significance was set at an alpha of 0.05.

As a preliminary assessment of possible genetic influence on phenotypic expression, concordances rates were calculated for MZ and DZ pairs for all hypocone expressions. Structural equation modelling was then undertaken using the software package Mx (Neale et al., 2006). Mx is a structural equation modelling package, flexible enough to fit a variety of mathematical applications. At its heart is a matrix algebra processor. There are many built-in fit functions to enable structural equation modelling (SEM) and other experiments in matrix algebra and statistical modelling, including facilities for maximum likelihood estimation of parameters from missing data structures, under normal theory. Complex ‘non-standard’ models are easy to specify. For further general applicability, it allows users to define their own fit functions, and optimization may be performed subject to linear and nonlinear equality or boundary constraints.

Mx can be used to apply structural equation models to variance-covariance matrices derived from monozygotic (MZ) and dizygotic (DZ) twin data. This method is particularly well-suited for continuously distributed data. However, SEM methodology can be extended to dichotomous and ordinal twin data by substituting the tetrachoric or polychoric correlation matrix (Pearson, 1901) for the variance-covariance matrix (Neale and Cardon, 1992). Use of SEM methodology for ordinal data is dependent on the assumption of an underlying continuous liability distribution that is bivariate normal. That is, it is assumed that categories are formed by imposing thresholds on a continuous liability distribution (Falconer, 1965; Reich et al., 1972).

Four sources of variation: A, additive genetic variance; D, non-additive genetic variance; C, common [shared] environmental variance; and E, unique [non-shared] environmental variance were modelled for twin pairs. A represents the additive effects of the alleles at a locus, whilst D refers to intralocus gene interactions.
C affects twin similarity regardless of zygosity, whereas E only represents unique effects contributing to within-pair differences.

Implicit in the model-fitting were the normal assumptions of the twin method: that mating was random; that trait-related, shared environmental influences on MZ and DZ twins were equal; and that there was no GxE interaction or gene-environment covariation (Jinks and Fulker, 1970). Since fitting models with four parameters to data from a classical twin study (MZ and DZ twins reared together) results in an under-identification model (Grayson, 1989; Hewitt, 1989; Dempsey et al., 1999), subsets of three or fewer parameters were chosen.

Rectangular files of raw ordinal data were prepared as described by Neale et al. (2006) and utilized directly for univariate analyses of ordinal data, maximising the likelihood under a bivariate normal distribution model. For right and left first molars, scores of 3 or less were combined into a single category (i.e., $\leq 3$) as only one tooth was scored less than a 3. Starting values for model thresholds were estimated from raw frequencies.

When analysing raw data, there is no direct measure of goodness of model fit. Instead, nested sub-models can be compared by examining the log of the likelihood function ($\log L$). Nested model differences in $-2\log L$ are distributed asymptotically as a $\chi^2$, with degrees of freedom equal to the differences in free parameters between nested sub-models (e.g., $ACE$ vs $AE = 1$ df). Initially, a Cholesky decomposition of the data was undertaken to produce a saturated model fit against which to test goodness-of-fit of nested sub-models. Where models were not nested (i.e., $ACE$ vs $ADE$), the relative magnitude of the log of the likelihood was used to indicate the parsimony of each model. The general approach was that of accepting a more complex model only when a simpler one had failed. Path coefficients (a, c, e) were estimated. Heterogeneity of causes of variation between sexes was also evaluated.

Narrow-sense heritability estimates ($h^2$) were calculated from the ratio of genetic variation (A) to total phenotypic variation (A+C+D+E) in the best-fitting model. Values of heritability estimates near 1 indicate that most of the phenotypic variation can be explained by additive genetic effects whereas values near zero indicate that environmental effects account for most of the variation in the phenotype.

### RESULTS

Concordance between the first and second sets of scores was 98% and there was no indication of systematic methodological errors. Inter-examiner concordance was 72% and the discrepancies found were generally of the order of plus or minus one category.

Hypocones were present on all permanent first molars and on a high proportion of second molars as demonstrated in Table 1. Pronounced expressions of hypocones were noted on first molars, with only one individual having a score of less than 3, and a high proportion of score 4 or 5. The second molars demonstrated more variation in hypocone expression. Subsequent genetic analysis treated hypocone expression on first molars as an ordinal trait with fewer categories than the second molars, yielding significantly lower power than the model for second molars, and consequently broader confidence intervals for parameter estimates.

Females had more pronounced expressions of score 4 and 5 on first molars, whereas the second molars showed more variability. Only 2% of females showed scores of

### TABLE 1. Expression of hypocone trait in males and females (one member of each twin pair)$^1$

| Score | Males | | | | | Females | | | | |
|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
|       | First molar | | Second molar | | | First molar | | Second molar | |
|       | Right | Left | Right | Left | | Right | Left | Right | Left |
|       | n | % | n | % | | n | % | n | % | | n | % | n | % | | n | % |
| 0     | 0 | 0.0 | 0 | 0.0 | 4 | 11.0 | 3 | 8.0 | 0 | 0.0 | 0 | 0.0 | 5 | 9.6 | 9 | 17.3 |
| 1     | 0 | 0.0 | 0 | 0.0 | 8 | 22.0 | 5 | 14.0 | 1 | 1.9 | 0 | 0.0 | 15 | 28.8 | 8 | 15.4 |
| 2     | 0 | 0.0 | 0 | 0.0 | 4 | 11.0 | 4 | 11.0 | 0 | 0.0 | 0 | 0.0 | 6 | 11.5 | 7 | 13.5 |
| 3     | 0 | 0.0 | 0 | 0.0 | 9 | 25.0 | 12 | 33.0 | 3 | 5.8 | 3 | 5.8 | 15 | 28.5 | 17 | 32.7 |
| 4     | 19 | 53.0 | 19 | 53.0 | 9 | 25.0 | 10 | 28.0 | 28 | 53.8 | 29 | 55.8 | 10 | 19.2 | 10 | 19.2 |
| 5     | 17 | 47.0 | 17 | 47.0 | 2 | 6.0 | 2 | 6.0 | 20 | 38.5 | 20 | 38.5 | 1 | 1.9 | 1 | 1.9 |

$^1 n = 88$
5 on both upper right and left second molars. Males showed a higher percentage of score 5 than females, with 47% of males showing score 5 compared to 38% of females. Females showed a higher percentage of score 4 and below. There was a tendency for male hypocones to be larger but this was not statistically significant.

In first molars, 98% concordance in expression between antimeric teeth was noted. The only example of marked asymmetry was one individual with score 1 on the left first molar and score 4 on the right first molar, as shown in Figure 1. In second molars, the concordance rate for antimeres was 74%.

The hypocone expression of first molars compared with second molars was examined in 88 individuals. One member from each of the 88 twin pairs (i.e. Twin A) was included in this analysis. As seen in Table 2, almost all of the scores for the maxillary right first molar were larger than those for the right second molar, except for five subjects—three had a score of 5 on both first and second molars, one had a score of 4 on the first molar and score 4 on the second, and one had score 3 on the first molar and score 4 on the second. When examining the maxillary left molars, again, most of the scores on the first molar were larger than those on the second molar except for four individuals—three had a score of 5 on both first and second molars and one had a score of 4.

**TABLE 2: Expression of hypocone trait on maxillary right first and second molars of individuals (one member of each twin pair)**

<table>
<thead>
<tr>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>First molars</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>17</td>
<td>5</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>8</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>17</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>47</td>
<td>37</td>
<td>88</td>
<td></td>
</tr>
</tbody>
</table>

^1n= 88
4 on both first and second molars. A typical example of reduction in cusp size from the first to second molar is shown in Figure 2.

MZ twin pairs exhibited a higher concordance rate for corresponding tooth comparisons than DZ twin pairs, as shown in Table 3. Percentage concordance for the first molars between MZ twin pairs was 80% whilst that for DZ twin pairs was 67%. The concordance rate noted for the first molars was higher than that for the second molars, with the percentage concordance for the second molars being 65% for MZ twin pairs and 22% for DZ twin pairs.

An AE model was the most parsimonious for all variables. There was no significant heterogeneity between sexes for variance components for hypocone score. The final models represent pooled data from both sexes. As an example, Table 4 presents the pooled model structure and statistics for hypocone scoring of the upper right first molars only.

Table 5 presents narrow-sense heritabilities for hypocone score variability in the sample. All values were high and ranged between 0.87 and 0.93. The second molars had slightly higher estimates than the first molars.

**DISCUSSION**

In this study, the 6-grade scoring method developed by Turner et al. (1991) for classifying hypocone expression was shown to be relatively easy to use and to provide consistent results. The intra-observer reliability of 98% was slightly higher than that of Takahashi et al. (2007) who reported a concordance rate of 92% for scoring categories of hypocone expression on two separate occasions. The inter-observer assessment study showed a concordance of 70-74% which was similar to that found by Nichol and Turner (1986) who recorded concordance between observers of 70-75% for ranked traits (error rate 25-30%).

The relative sizes of the cusps tended to correspond with phylogenetic and ontogenic timing of cusp formation. Apart from one case, the hypocone was shown to be reduced from the maxillary first to second molar. The one exception could possibly be due to different crown morphology making scoring difficult. The overall results were consistent with previous cusp area studies (Nichol and Turner, 1986; Macho and Moggi-Cecchi, 1992; Takahashi et al., 2007) and support the morphogenic field concept of Dahlberg (1945).

As reported by Takahashi et al. (2007) this study did not show any statistically significant difference in the occurrence of hypocones between males and females. However, this may be a reflection on the categorical system of classification used, which gives little information about actual size variation. This study did, however, show a tendency for higher frequencies of larger hypocone expressions in males than females. Kondo et al. (2005) also reported that larger distal cusps were found in males rather than females. Hence, it is

### Table 3. Percentage concordance observed for hypocone trait expression in DZ and MZ twin pairs

<table>
<thead>
<tr>
<th>Tooth</th>
<th>Right M1</th>
<th>Right M2</th>
<th>Left M1</th>
<th>Left M2</th>
</tr>
</thead>
<tbody>
<tr>
<td>DZ</td>
<td>67.4%</td>
<td>25.6%</td>
<td>67.4%</td>
<td>18.6%</td>
</tr>
<tr>
<td>MZ</td>
<td>80.0%</td>
<td>62.2%</td>
<td>80.0%</td>
<td>68.9%</td>
</tr>
</tbody>
</table>

### Table 4. Genetic model structure and associated statistics for hypocone scores on the maxillary right first molar

<table>
<thead>
<tr>
<th>Parameterization</th>
<th>n</th>
<th>Parameters</th>
<th>-2 Log Likelihood</th>
<th>df</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE</td>
<td>176</td>
<td>7</td>
<td>255.9</td>
<td>170</td>
<td>-84</td>
</tr>
<tr>
<td>ADE</td>
<td>176</td>
<td>7</td>
<td>256.0</td>
<td>170</td>
<td>-84</td>
</tr>
<tr>
<td>AE</td>
<td>176</td>
<td>6</td>
<td>256.0</td>
<td>171</td>
<td>-86</td>
</tr>
<tr>
<td>CE</td>
<td>176</td>
<td>6</td>
<td>260.8</td>
<td>171</td>
<td>-81</td>
</tr>
<tr>
<td>E</td>
<td>176</td>
<td>5</td>
<td>286.1</td>
<td>172</td>
<td>-58</td>
</tr>
</tbody>
</table>

1 Abbreviations: n = sample size; df = degrees of freedom; AIC = Akaike’s Information Criterion
likely that there is some influence of the sex chromosomes on the hypocone trait.

Hypocone expression was shown to be symmetrical between antimeric first molars except for one instance where the expression on the left was greater than the right, i.e. score 4 compared with score 1. A number of researchers have suggested that both sides of the dental arch are under the influence of common genetic factors (Potter et al., 1976; Baume and Crawford, 1980). The findings of this study would support this hypothesis but the observed asymmetry in one individual would suggest environmental factors can lead to antimeric asymmetry.

The fact that this study shows a lower rate of concordance for hypocone expression between right and left maxillary second molars than first molars relates well to the schedule of tooth development and the theory that there is an association between early crown formation and low morphological variation of the first molar (Macho and Cecchi, 1992). These findings support the contention that certain teeth in the dentition, generally the earlier-developing members of each tooth class, are under stronger genetic control than later-forming teeth that are more subject to environmental influences (Corruccini and Potter, 1981).

It is assumed that MZ twins share 100% of their genes but the similarities between them can be due partly to shared pre- and post-natal environment (Scott and Turner, 1997). Common environment is perfectly correlated between twins in both zygosity groups whereas unique or non-shared environment only contributes to differences between twins. If genes are responsible for the expression of a trait, then a higher concordance of expression between MZ twin pairs would be expected compared with that seen between DZ twin pairs. This is what was noted in this study. However, although the concordance rate of expression of hypooones in MZ twin pairs was higher than that in DZ twin pairs, the concordance was not 100%.

Narrow-sense heritability estimates indicate the proportion of the phenotypic variation attributable to additive genetic effects. Narrow-sense heritability is a measure of the degree to which individual phenotypes are determined by genes passed from parents to offspring, expressed as the ratio of the additive genetic variance to the total phenotypic variance. The high heritability estimates noted in this study suggest that most of the variation in expression of hypooones is due to genetic influences but environmental factors can still contribute to the observed variation. Hypoones were universally present on first molars; the second molars demonstrated a greater range of phenotypic expression than the first molars, with absence noted in some individuals. This may suggest that there is a common genetic liability for hypocone expression on both the first and second molars, which is modulated by differences in size, location and/or developmental timing events between these teeth.

In studies aimed at disclosing patterns in estimates of heritabilities, it has generally been assumed that the highest heritability will be displayed by the key tooth in each morphogenetic field (Townsend et al., 2008). This was not noted in this study; in fact, slightly higher values were achieved by the second molar compared with the first. It is considered that the longer a cusp remains in its soft tissue stage prior to mineralisation the more likely phenotypic variation will occur since odontogenesis involves a series of complicated epigenetic and morphogenic events. (Townsend et al., 2008). Due to the relatively small variation in hypocone size on the first molars, only three categories were analysed (score 3 and below, score 4 and score 5), whereas when looking at second molars all six categories were considered. This difference in the categories of expression analysed between the first and second molars may have influenced the heritability estimates, contributing to the lower scores noted for the first molars.

CONCLUSIONS

The hypotheses of this study were generally supported in that:
1. Hypocone expression was more common and larger in maxillary first molars than second molars.
2. Although sexual dimorphism was not statistically significant, there was a trend for males to have larger scores than females.
3. The expression of hypocones was symmetrical between antimeric teeth, with the concordance rates between sides being higher in first molars than second molars.
4. Monozygotic twin pairs exhibited a higher concordance rate hypocone expression than dizygotic twin pairs.

The results of model fitting and calculation of heritability estimates indicated that genetic factors exert a strong influence on hypocone expression in human maxillary molar teeth but environmental factors can also contribute to observed variance.

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LITERATURE CITED


Patterns of Hypodontia among Third Molars in Contemporary American Adolescents

Edward F. Harris

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ABSTRACT: Third molars (M3s) are congenitally absent (hypodontic) more frequently than any other tooth type. Causes of this enhanced variability are poorly understood, but the potential range of absence—from none through four M3s per person—provides the opportunity to examine the permutations of missing M3s within and among ethnic groups. Teenage samples of two overlapping populations (1,100 American whites; 600 American blacks) were studied here, with radiographic confirmation of each tooth’s presence in the jaws. Roughly 15% of these people are missing at least one M3, but only about 2% of this sample is hypodontic for all four molars. The frequency and severity of missing M3s are significantly higher in whites than blacks. Within individuals, correspondence of occurrence is much higher within than between the jaws, but all combinations of M3 hypodontia are positive and significant statistically—implying common underlying developmental influences. While various sorts of data support a genetic influence on the risk of M3 hypodontia, patterns of inheritance suggest a multifactorial rather than a single-gene mode of inheritance. Several researchers have promoted a polygenic threshold model, and the history and application of this model are discussed. Dental Anthropology 2009;22(1):8-17.
germs. While it is a graphic metaphor to suppose that a formative tooth bud might be “choked” out of existence due to inadequate bony support, there are no data to support this. Instead, cytokines from the dental follicle attract osteoclasts during normal development (Marks and Cahill, 1983), and these clast cells progressively enlarge the surrounding tooth crypt to accommodate the developing tooth (Carlson, 1944). This is readily seen (and palpable) in infants, where the buccolingual diameters of the primary tooth crypts have enlarged well beyond the incipient bony ridges, and the surfaces of the ridges are scalloped due to these out-pouchings (e.g., van der Linden and Duterloo 1976). The emergence of teeth into a tight-fitting arcade of teeth as seen in the adult is not indicative of the three-dimensional arrangement of tooth crypts—plus the temporal span during which different teeth form. For example, the canine abuts against the lateral incisor in the adult, but (A) the lateral incisor forms much earlier, when there is plenty of room in the supporting jaws, and (B) when the canines do form, their positions are far apical of the other teeth.

Molecular biology now makes it clear that a tooth will fail to develop if there is no ectodermal signal to stimulate a site along the underlying mesenchyme to initiate tooth formation (Kollar and Baird, 1970a,b). This cause of hypodontia seems primarily genetic in nature, but failure of formation also can be affected by the environment. Suggestions from animal studies are that tooth buds that fail to reach a critical size will resorb—resulting in hypodontia rather than continuing to develop. Likewise, environmental stressors acting at the critical early stages of formation can simply kill off a tooth bud. Teratogenic drug actions and irradiation are well-studied examples of this (Bruce, 1950; Kaste et al., 1998). Yet a third mechanism involves a genetic interruption of the cascade of molecular signals leading to tooth formation. This is obvious in the edentates (e.g., armadillos, anteaters; Todd, 1918) where there is no tooth formation, but development ceases early in the bell stage. This interruption also accounts for the “missing teeth” (absence of lateral incisors, canines, and premolars) that is characteristic of mice and other rodents. (See review by Peterkova et al. 2006.) The extreme example of this inhibition of tooth development probably is in birds (the class Aves), where all modern birds are tooth-less but tooth formation can be reintroduced experimentally (Chen et al., 2000; Mitsiadis et al., 2006). At an allelic level, it is conceivable that this sort of interruption of molecular events accounts for the variable frequencies of tooth agenesis in humans (e.g., Matalova et al., 2008).

Numerous clinical and physical anthropological studies have reported on the frequencies of missing M3s in humans. The purpose of the present study is to investigate the pattern of missing M3s in some detail within and among individuals in population samples. That is, there are 4 M3s distributed as left-right pairs in the two dental arches, and the issue is how hypodontia is distributed among these 4 sites. This study is restricted to M3s, though there are evident associations among tooth types (Davies, 1968; Khalaf et al., 2005; Harris and Clark, 2008). As pertinent examples, Alvesalo and Portin (1969) and Woolf (1971), among others, have documented that the maxillary lateral incisor is more often affected (diminished size, pegged, absent) in individuals with hypodontic M3s versus those with developmentally intact dentitions; hypodontia is not an isolated phenomenon, even among tooth types that form at quite different ages.

MATERIALS AND METHODS

Panoramic radiographs (van der Linden and Duterloo, 1976) of 1,700 adolescents were studied. Most (1,100) were American whites, and the rest were American blacks (600), all from clinical records at the College of Dentistry, University of Tennessee, Memphis. Subjects were selected with radiographs taken between 12 and 18 years of age. These adolescents were old enough that their M3s would have begun mineralization if they were going to form (Rantanen, 1967; Harris, 2007), but the adolescents were young enough to well remember having any M3s extracted. It seems obvious that hypodontia has to be documented radiographically, especially for M3s that commonly form but do not erupt into the oral cavity. Sample sizes vary among the statistical tests described here because not every tooth’s existence could be documented because of radiographic issues.

One intent was to estimate the background frequencies of M3 hypodontia in these two ethnic groups, so subjects with a recognized craniofacial syndrome, including facial clefts, were omitted since they have characteristic—often elevated—patterns of hypodontia (e.g., Schalk-van der Weide, 1992; Ranta, 1983; Harris and Hullings, 1990).

Tooth formation can be viewed as a dichotomous event—a tooth has either developed or it is absent. With potentially one M3 in each quadrant, there are 16 permutations of hypodontia. Expansion of the binomial shows that there are five M3 groupings, namely (A) all 4 M3s present, (B) four arrangements with 1 tooth missing, (C) 6 arrangements with just 2 teeth missing, (D) 4 arrangements of 3 teeth missing, and (E) one situation where all 4 M3s are hypodontic. In other words, the 16 permutations are arranged in the familiar ratios of 1:4:6:4:1.

Statistical tests relied on chi-square analysis. Statistics were performed using JMP 7.0 (SAS, Cary, NC). The kappa statistic was calculated as the measure of association (Fisher and van Belle, 1993).
RESULTS

The observed frequency of M3 hypodontia for the total sample (Table 1) shows that the distribution is far from random. Despite common perceptions that hypodontia of M3 is common, most people experience development of all 4 M3s (86.8%; 1449/1670), whereas congenital absence of all 4 M3s occurred in just 1.6% of the cases. Fig. 1 shows that the distribution of severity (i.e., number of congenitally absent M3s) approximates the right-end of a normal distribution, where the frequency decreases as the number of missing M3s increases. The perception that M3s frequently are absent is strongly influenced by the widespread prophylactic extraction of M3s in the late teens (e.g., Eklund and Pittman, 2001).

Black-White differences

American blacks and whites have been admixing for centuries, though admixture estimates are lower in the Southeast than elsewhere in the nation because of harsher social and legal proscriptions (Williamson, 1980; Davis, 1991). Blacks have larger and morphologically more complex teeth (Richardson and Malhotra, 1975; Irish, 1997), and, evidently in an associated manner, discernibly lower frequencies of hypodontia (Harris and Clark, 2008). Stanley Garn contended in several of his publications (notably 1977) that tooth size, morphology, tempo of formation, and occurrence (in contrast to congenital absence) are positively intercorrelated features of a common underlying theme in tooth formation, not isolated phenomena—and that these features differ among tooth types controlled, at a primary level, by a tooth’s position in its morphogenetic field.

Table 2 shows the distributions in each of the arches (sexes pooled). In both jaws, whites have highly significantly higher frequencies of M3 hypodontia, and the source of the significance is primarily due to deficits of bilateral absence in blacks compared to whites (as assessed from the cell chi squares).

Little is known about hypodontia in other, non-Caucasian races; most work has been done on peoples of European extraction where frequencies and the patterning of hypodontia among tooth types probably is not representative of all groups. Röse (1906) and Hrdlička (1921) each collated data from large series of peoples of diverse races—but with ill-defined criteria and without the benefit of radiography to confirm congenital absence. Still, differences in the frequencies of hypodontia are evident in these early studies. Population differences in trait frequencies are prima facie evidence for a genetic influence on the risk of hypodontia.

Sexual dimorphism

The data in Table 1 were dichotomized into cases
without M3 hypodontia and cases missing one or more M3s. This showed that hypodontia is significantly more common in girls than boys in whites ($\chi^2 = 5.3; \text{df} = 1; P = 0.02$). The source of significance (based on cell chi squares) is primarily due to the comparative deficit of hypodontia in males. 14% of males exhibit agenesis of one or more M3s, compared to 19% of females. The overall frequency is appreciably lower in American blacks (ca. 6% vs. about 16% in whites) and, with the smaller sample size of 600, the sex difference is not significant here ($\chi^2 = 1.8; \text{df} = 1; P = 0.1850$). If the present frequencies hold, a sample size roughly three times larger (ca. 2,000) would be needed to achieve statistical significance in blacks.

In addition to the greater frequencies of M3 hypodontia in females, Fig. 2 shows that severity—as measured by the number of missing M3s—also is greater in females than males. This shift towards greater expression in females is more obvious in whites because of their greater incidence of M3 hypodontia overall.

### Arcade effects

There are positive, statistically significant associations for M3 hypodontia between all four M3s taken pairwise; the matrix of kappa correlations (Table 3) based on the total sample shows that left-right symmetry is highest (kappa ~ 0.7) within each arch, and the inter-arch associations are appreciably lower (kappa ~ 0.3), but correlations within and between hemispheres seem equivalent. Hierarchically, the symmetry between sides is much higher than between arches, but whether the association between the arches is taken between the same or opposite quadrants seems immaterial.

A related point is that asymmetric occurrence is relatively uncommon. M3 status in one quadrant strongly predicts the same status in the antimeric site. This is anticipated since our understanding is that the same genotype affects tooth development in the left and right quadrants, with effectively the same environment in each to achieve a tooth’s phenotype. Dental researchers have sought evidence for laterality or sidedness, primarily using data on crown dimensions. Documentations of laterality are few and scattered among samples (e.g., Harris, 1992; Townsend et al., 1999). The bulk of left-right asymmetry is expressed as random (fluctuating) asymmetry, at least with regard to size.

Figure 3 arborizes the frequencies of hypodontia by tooth type and, thereby, shows the dependencies (statistical associations) between the arches. An obvious “dose-dependent” relationship from among several of the associations is this: When both upper molars are congenitally absent, just 50% of the two mandibular molar molars are present. When just one upper molar is present, the frequency of the two lower molars being

### Table 2. Frequencies of M3 hypodontia in American Blacks and Whites

<table>
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<th></th>
<th>Whites</th>
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<tbody>
<tr>
<td></td>
<td>Both Absent</td>
<td>Both One Absent</td>
<td>Both Present</td>
<td>Both Absent</td>
<td>Both One Absent</td>
<td>Both Present</td>
<td>Chi-squared</td>
<td></td>
<td></td>
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<tr>
<td>Maxilla</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
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<td>n</td>
<td>%</td>
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<td>%</td>
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<td>%</td>
</tr>
<tr>
<td>Both</td>
<td>5.0</td>
<td>56</td>
<td>4.0</td>
<td>45</td>
<td>90.9</td>
<td>1010</td>
<td>1.7</td>
<td>10</td>
<td>3.2</td>
<td>19</td>
<td>95.1</td>
<td>561</td>
<td>2</td>
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<td>Mandible</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
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<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Both</td>
<td>4.5</td>
<td>74</td>
<td>3.1</td>
<td>51</td>
<td>92.5</td>
<td>1534</td>
<td>1.2</td>
<td>7</td>
<td>1.5</td>
<td>9</td>
<td>97.3</td>
<td>572</td>
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1Both $\chi^2$ values are highly significant ($P < 0.0001$) because M3 hypodontia is more common in whites than blacks.

**Fig. 1.** Percentage distribution of M3 hypodontia in the total sample.
is higher in whites. Perhaps because the overall incidence
though the extent of sexual dimorphism is appreciably
blacks, and more frequent in girls than boys in each race,
hypodontia is more common in American whites than
M3 (all expressivities combined) by race and sex. M3
dichotomy: the tooth either is present or absent. Features
occurrence (P = 0.53). Congenital absence of M3 is, then,
1,670 individuals where all 4 M3s could be scored. These
in both dental arches are uncommon, just 7 cases in the
86.1
80.8
92.5
Percent of Sample
White males
White females
Black males
Black females
Number of Missing M3s
0 1-4
0 20 40 60
0 80

Fig. 2. The frequencies of the congenital absence of
M3 (all expressivities combined) by race and sex. M3 hypodontia is more common in American whites than
absent rises to 70%. And, when neither upper M3 is absent, the frequency of both lower molars being
agenetic rises to a high of 94%.

Side effects
Sidedness is the interesting situation where there is
preferential laterality: Does absence of a tooth on one
side influence absence of the same tooth in the
opposing arch? The informative cases are those where
either the left or right molar is absent in the maxilla
and likewise (unilateral absence) in the mandible.
Unfortunately, cases of unilateral congenital absence
in both dental arches are uncommon, just 7 cases in the
1,670 individuals where all 4 M3s could be scored. These
7 cases were equally distributed (3:4) as to arrangements
where the ipsilateral tooth (same hemisphere) was
missing in the two arches versus where the contralateral
tooth (opposite hemisphere) was absent. At least with
these few informative cases, there is no suggestion of
sidedness.

Another way of viewing laterality is simply whether
M3 is more common on one side of the mouth than the
other. The maxillary left-right distribution of unilateral
presence is 33 (left only) compared to 32 (right only),
which is indistinguishable statistically from a random
spread of 50:50. In the mandible, the left-right distribution
of congenital absence is 27 (left only) compared to 34
(right only). This does not depart from a 50:50 chance
occurrence (P = 0.53). Congenital absence of M3 is, then,
equally distributed between sides.

DISCUSSION
Hypodontia in itself suggests a phenotypic
dichotomy: the tooth either is present or absent. Features
ABSENCE OF THIRD MOLARS IN ADOLESCENTS

There is an interesting but tangled history of this model. Many physical anthropologists, particularly those with interests in skeletal biology, attribute it to the work of Hans Grüneberg (1950, 1952) who marshaled the quasicontinuous model (QCM) as an explanation for the numerous minor skeletal variants he studied in mice, such as accessory foramina, ossicles, and other morphological features, that occur in some animals but not others. The utility of these “discrete” (i.e., present or absent) skeletal features for phenetic studies of human skeletal series was popularized by A. C. Berry and R. J. Berry (e.g., 1967, 1968, 1974).

Grüneberg’s work in turn rested on the seminal studies of Sewall Wright in the 1930s. Wright (1934a,b) explored the inheritance of the number of digits on the hind feet of guinea pigs, which normally have 3 digits but may have 4, and attributed the occurrence of 4 toes to the guinea pig’s genotype exceeding what he termed a “physiological threshold.” Indeed, his Figure 1 (1934b, p. 544) depicts the presumed underlying polygenic model as a normal curve overlying two successive thresholds, a lower one, where poorly-formed (“vestigial”) 4th toes occur, and a higher one, where the 4th toe is eumorphic. This development of a two-threshold scheme is precisely what was exploited later by Reich and others (Reich et al., 1972; Corbett et al., 2004) to provide practical statistical tests for distinguishing between single-gene and polygenic models of inheritance. While Wright did not formalize the QCM, he described its major features during his various breeding experiments. Denys Falconer (1965) elaborated the assumptions and statistical expectations of the QCM. Falconer described how heritability (\( V_{\text{additive}} / V_{\text{total}} \)) of a trait could be estimated from trait frequencies. However, this requires family data (information on relatives of known degrees of biological relatedness). Heritability cannot be calculated from samples of cases without known relationships, so this useful aspect of the QCM generally has been ignored in skeletal biological studies, but with some noteworthy exceptions; Saunders and Popovich (1978) recorded minor skeletal variants from radiographs of siblings enrolled in the Burlington Growth Study. Sjøvold (1984, 1996) analyzed skulls of Europeans where genealogical information had been preserved. Cheverud and colleagues (e.g., McGrath et al., 1984; Richtsmeier et al., 1984) used the unique setting of the island of Cayo Santiago (where genealogical affinities of most monkeys is known) to estimate heritability of several nonmetric bony features in macaques.

The work of Carter (notably 1969) warrants mention here because (A) he demonstrated the applicability of a threshold model for many common diseases (e.g., pyloric stenosis, diabetes mellitus, spina bifida cystica, and others), which did much to familiarize the health care community with this quasicontinuous model and

| TABLE 3. Correlation coefficients (kappa) between the four third molars taken pairwise
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<tr>
<td></td>
<td>Upper</td>
<td>Upper</td>
<td>Lower</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>Upper Right</td>
<td>0.66</td>
<td>(0.0400)</td>
<td>0.31</td>
</tr>
<tr>
<td>Lower Right</td>
<td>0.30</td>
<td>0.31</td>
<td>0.71</td>
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1Values in parentheses are the standards errors of the estimates; all 6 correlations are highly significantly different from zero (P < 0.0001).

Fig. 3. Dependence between arches in the occurrence of M3s. (Top) When both maxillary M3s are present, 94% of cases have both mandibular M3s present. (Middle) When just one of the two maxillary molars is present, the frequency of both lower M3s being present drops to 70%. (Bottom) When both maxillary M3s are congenitally absent, the frequency of both lower M3s being present is just 40%. These associations indicate nothing about cause and effect; the same dependencies are found if the lower molars are taken as the predictive variable.
(B) he listed several criteria that, when met, can be very suggestive of a polygenic threshold model.

While largely beyond the scope of this paper, it is informative to note that James (1971) pointed out that too many parameters need to be estimated than can be obtained from a QCM with one threshold. But, adequate parameters are available if two thresholds are supposed in the model, and James worked with Ted Reich (e.g., Reich et al. 1972; Corbett et al. 2004) to develop tests that can distinguish between inheritance due to a single-gene model versus a polygenic model. Suarez and Spence (1974) applied a basic form of this approach to the hypodontia family data collected by Grahnen (1956), concluding that a polygenic threshold model fit the data appreciably better than expectations of the effects of single gene.

**QCM and Hypodontia**

Davies (1968), Sofaer (1969), Bailit (1975), and Chosack et al. (1975), among others, alluded to the QCM fitting observations seen in population samples, but Brook (1984) was the first to seriously develop the QCM to hypodontia (and, at the other, complementary extreme, hyperdontia). Brook emphasized the developmental interrelationships between hypodontia and tooth size. There also is a well-documented relationship between hypodontia and crown sizes of the remaining teeth; people in the population who do not have hypodontia have statistically larger teeth than those with congenital absence (Garn and Lewis, 1962; Garn et al., 1962, 1963, 1970). Conversely, diminished crown sizes and microdontia are more common in those with hypodontia than in those with full complements of teeth. These clinical results are duplicated in laboratory animals (Grüneberg, 1950, 1952; Self and Leamy, 1978). The greater the extent of hypodontia, the greater the size reductions and the greater likelihood of microdontia (with associated missing cusps and simplified morphologies of the remaining teeth). Numerous studies of European groups have found higher frequencies of hypodontia in females than males (reviewed in Egermark-Eriksson and Lind, 1971). These several associations suggest that hypodontia has dentition-wide systemic effects, which is predictable since teeth form as repetitive elements (a meristic series; Bateson, 1894) using the same regulatory mechanisms controlled by the person’s genotype (Kettunen and Thesleff, 1998; Jernvall and Thesleff, 2000).

Grüneburg (1952) documented differences in the frequencies of third molar hypodontia among inbred strains of mice. Mice with larger teeth had lower frequencies of M3 hypodontia than strains with smaller teeth. The same relationship is evident in humans, where African Americans (with large teeth) exhibit M3 hypodontia infrequently compared to American whites with smaller crown sizes and higher frequencies (and greater severities) of M3 hypodontia (Harris and Clark, 2008). Hyperdontia (supernumerary teeth) is, in contrast, more common in males (e.g., Stafne, 1932; Khalaf et al., 2004).

This collage of interrelated features recently has been extended by Uslenghi et al. (2006) who showed that hypodontia is associated with slowed tooth development (also see Garn et al. 1961).

![Fig. 4. Schematic of the quasicontinuous model (modified from Brook, 1984). There is an underlying genotypic range in a population that influences a person’s risk for hypodontia (left extreme) and hyperdontia (right extreme). Sex-specific distributions are shown here to reflect the greater risk of congenital absence in women versus the greater risk of supernumerary teeth in men — at least in peoples of European extraction. Sexual dimorphism appears to be lower in peoples of sub-Saharan extraction. The vertical bars are depicted as broken lines since a person’s genotype can be modulated in either direction by the environment.](image-url)
OVERVIEW

The present study assessed the phenotypic patterns of third molars (M3) congenital absence in 1,700 teenagers composing a contemporary cohort of American blacks and whites from the Southeast United States.

- There is no difference by arcade, but agenic M3s are significantly more common in females than males and in American whites compared to American blacks.
- No evidence of sidedness (preferential absence on one side) could be discerned, and asymmetry (unilateral occurrence) is fairly uncommon versus symmetric presence or absence.
- Congenital absence of one M3 is highly predictive of other missing M3s, suggesting common developmental associations that probably are modulated by the person’s genetic background.
- While genes with rather severe effects on congenital absence have been documented, most cases of hypodontia are of unknown etiology, although population distributions are in concert with a quasicontinuous model of inheritance (also termed a polygenic threshold model).

LITERATURE CITED


Nature 192:989.
Wright S. 1934b. The results of cross between inbred strains of guinea pigs differing in number of digits. Genetics 19:537-551.
Supernumerary teeth are those that are additional to the normal complement. They may occur in any region of the dental arch and have been reported in both the primary and the permanent dentitions. The etiology of supernumerary teeth is still not clearly understood, but several theories have been suggested for their occurrence. The investigated material were the remains from the Bácsalmás-Óalmás burial site (from the 16th-17th centuries), where 472 skeletons were excavated from 1993 to 2003. For the purpose of this study, the dentitions of 164 adult individuals were examined. The examination was carried out using macromorphological methods, radiographic analysis and a dial caliper were applied. This paper describes a supernumerary tooth of an adult female skeleton. On the labial surface of the first mandibular premolar an extra tooth was observed. Radiographic examination of the fused teeth indicated that the crown of the premolar had fused incompletely with the crown of the extra tooth. The position of the extra tooth could have been the result of gemination of the tooth germ or the elaboration of the buccal cingulum. The cranium of the examined individual showed some mongoloid morphologic features, too. Our presumption about the formation of the supernumerary tooth may have contributed to the theories of the occurrence of supernumeraries. The sporadic occurrence of this anomaly was reported in recent and archaeological skeletal collections. This study showed that multiple permanent dental formation was present in past Hungarian populations, representing a contribution to the history of dental anomalies. Dental Anthropology 2009;22(1):19-22.

Heredity may be a relevant etiological factor in the occurrence of supernumerary teeth (Rajab and Hamdan, 2002). Supernumeraries are more common in the relatives of affected children than in the general population (Garvey et al., 1999). The reported prevalence of supernumeraries in the general Caucasian population for the permanent dentition ranges from 0.1 to 3.8% (Rajab and Hamdan, 2002). Supernumerary teeth seem to be more common in Asian populations, with a frequency higher than 3% being reported (Davis, 1987). Sexual dimorphism has been reported by most authors (Hurlen and Humerfelt, 1985; Mitchell, 1989) with males being affected more commonly.

The occurrence of this anomaly is also reported in archaeological skeletal collections. Hillebrand (1908) found 14 supernumerary teeth during the paleostomatological investigation of 4,100 skulls. Schwerz (1916) described this anomaly in two out of 510 cases. The sporadic occurrence of supernumeraries in past populations was reported in several other studies.
This report describes a rare developmental anomaly of a mandibular tooth of a young adult (25-30 yrs) female.

**MATERIALS AND METHODS**

The material for this report was the skeletal population of Bácsalmás-Óalmás burial site found in a sand pit, where 472 skeletons were excavated from 1993 to 2003. On the basis of the archeological and historical data, this group immigrated from the Balkan Peninsula to the southern part of Hungary in the sixteenth century. For the purpose of this study, the dentitions of 164 well-preserved adult individuals (76 males, 75 females, 13 indeterminate) were examined. Altogether 2,693 teeth (with the exception of the third molars) were used for the investigation. The examination was carried out using macromorphological methods and radiographic analysis. A dial caliper was used for the metric analysis.

**RESULTS**

During the paleostomatological investigation, altered number of teeth was one of the examined anomalies. The present report is a case of hyperdontia. The lower left first premolar of the young adult female revealed double tooth formation. Only this one case of hyperdontia was found from among the 164 skulls in the skeletal series of Bácsalmás-Óalmás. Due to postmortem loss, the young adult female had no upper left incisors and the upper right central incisor was missing. Pitted hypoplasia on the incisors was seen. Mild periodontal atrophy was collecte...
Supernumerary teeth may occur singly or in multiples in any region of the jaws in the same person. This study describes a unilateral supernumerary mandibular tooth of an adult female skeleton. Radiographic examination of the fused teeth indicated that the crown of the normal premolar had fused incompletely with the crown of the extra tooth. The fused teeth have two root canals and two partly separate roots. Communication between the pulp chambers of the teeth could be detected radiographically. The position of the extra tooth can be the result of gemination of the first premolar, which means that two morphological units were created by division of the tooth germ. The result is the incomplete formation of two teeth. According to Pindborg (1970), a true concretion develops during the formation of teeth and it is caused by the lack of space. But from another perspective the extra tooth can be the elaboration of the buccal cingulum of the premolar. In support of this concept, the crown is not fused completely and the roots are separated.

Regarding the etiology of this dental anomaly, Rajab and Hamdan (2002) considered heredity as an important etiological factor in the occurrence of supernumerary teeth. Heredity is not conclusive as no other supernumerary was found in this skeletal collection (164 skulls).

The fact that supernumerary teeth are more common in Mongoloid racial groups seems to be conclusive in this case because the investigated skull also shows Mongoloid characteristics.

Clinical complications related to double teeth include caries along the grooves dividing each other and periodontal atrophy, esthetics, and malocclusion (Silva and Silva, 2007). In extant groups, the majority of such teeth are asymptomatic, so endodontic treatment is unnecessary in most cases (Cetinbas et al., 2007).

The sporadic occurrence of supernumerary teeth is reported in recent (Hassan et al., 2006) and archeological (Sutton, 1985; Smith, 2004) skeletal collections. Kocsis (1993) investigated the permanent frontal teeth of 1,997 individuals originating from different archeological periods of Hungary. He found 23 supernumeraries with a highest frequency in the 10th century AD.

This case report shows that permanent dental formations in the premolar region were also present in the past populations of Hungary, representing a contribution to the history of dental anomalies.

LITERATURE CITED


Hillebrand J. 1908. Újabb adatok az ember fogainak alaktanához. Budapest: Stephaneum.


The case report by Gyongyi Szabó and colleagues (Dental Anthropology 2009;22(1):18-21) raises several interesting issues. A challenging aspect of examining teeth—which are the end-products of foregone cascades of developmental events—is that interpretations of the formative processes that produced the final form are conjectural, and there is no way to test assumptions. Experience and encountering repeated occurrences of a dental condition are helpful, but they are hardly infallible.

**Terminology**

A fundamental consideration raised by this case report is terminology. Specifically, what constitutes a supernumerary tooth? Or, for that matter, what is a tooth? I looked through a number of recent papers on hypo- and hyperdontia, and there is a striking absence of an operational definition of what a “tooth” is. Recognition of a tooth evidently is considered so obvious (or so difficult) that it doesn’t warrant a definition. It seems that mineralized tissues (dentin, enamel) are an important criterion, but this is simply because most studies nowadays are radiographic surveys, so premineralized tissues are undetectable. However, dental histologists are quite comfortable that the premineralized structures seen in the bud, cap, and bell stages constitute a “tooth,” so mineralization cannot be an essential feature.

Popular textbooks on dental anatomy (e.g., Zeisz and Nuckolls, 1949; Kraus et al., 1969; Ash, 1993) launch right into descriptions of the morphology of each tooth type, apparently supposing that a definition would be superfluous. The normally-occurring teeth (20 primary, 32 permanent) are all characterized by a crown (enamel, dentin, pulp) and one or more roots (cementum, dentin, pulp), but it is not clear whether a dental element must have all of these features to achieve “toothness.” Also, sizes of the crown and root do not seem to be important criteria. One might claim that teeth obviously are found in the two dental arches, but locality is not definitive given the extraordinary
places a “tooth” can occur. Fig. 4 is an example where a supernumerary incisor (probably a mesiodens) is hidden in the subject’s incisive foramen. The literature describes ectopic teeth located in various midface regions, such as the nasolacrimal duct (Alexandrakis et al., 2000), the bony orbit (Savundranayagam, 1972), and the eyelid (Subramaniam et al., 1966). Many of us were taught in an embryology class or elsewhere about dermoid cysts (e.g., Shafer et al., 1983), which contain well-differentiated skin and other identifiable tissues (e.g., hair, sweat glands, bone, cartilage, etc.), including teeth. These “teeth” commonly are of identifiable types, often incisors and premolars, which shows that the same complex of biochemical signals that produce a tooth in a dental arcade can perform just as well elsewhere in the body (e.g., Jernvall and Thesleff, 2000). This is not surprising given the landmark embryological studies.

Fig. 5. A mesiodens—a supernumerary tooth located between the maxillary central incisors—is common. Note how this erupted mesiodens displaces the incisors. There is chipping of the occlusal border of the mandibular right central incisor because of the edge-to-edge malocclusion. Supernumerary incisors typically are single-cusped and conical with a single root.

Fig. 7. An exceptionally large tuberculum dentale is located on the subject’s right maxillary central incisor (arrow). Large examples such as this with a free cusp often are labeled talon cusps because the appearance of an incisor with a labial and lingual cusp is reminiscent of a raptor’s claw (e.g., Harris and Owsley, 1991). Based on size, this tubercle (with a free apex, a pulp horn, and an independent root) would qualify as a “tooth,” but it is not counted as such because (A) it developed from the cingulum of the parent tooth and (B) it is a fused feature of the incisor, sharing dentin and pulp.

Fig. 6. A cropped view of a panoramic radiograph of a subject with dentin dysplasia I, where root formation is severely restricted, so several of the teeth appear to be ‘root-less’ though they generally erupt normally. Note the apical radiolucencies around several of the teeth, which is characteristic of this condition. Also, the pulp chambers are obliterated and filled with dysplastic dentin.

Fig. 8. Radiograph of a compound odontoma in the maxillary midline. There are four ‘toothlets’ visible here, but it is unclear whether they should be labeled as four supernumerary teeth because of their petite size and absence of any crown-root morphology. Even on X-ray, it is evident that these dental elements consist of enamel, dentine, and a pulp chamber. Note how this tumor is preventing the subject’s right maxillary central incisor from erupting and how it maintains a several-millimeter gap between the left central and right lateral incisor. Radiograph courtesy of James E. Turner.

4Figures 1-3 are those in published in the prior article by Szabó et al. (2009).
of growing implanted tooth buds in the globes of eyes of laboratory animals (e.g., Yoshikawa and Kollar, 1981). Dermoid cysts occasionally occur in ovaries (e.g., McGinnis and Parham, 1978; Dick and Honoré, 1985; Liberis et al. 2008), which means they should be recoverable archeologically, though I’m unaware of any reference to them.

A tooth does not have to be normal size or shape to be counted. Diminutive elements, such as pegged and microdont teeth, are routinely counted. Many authors include mineralized elements of any morphology, including “dental masses” of amorphic mineralized objects as found in odontogenic tumors, notably compound odontomas (e.g., Shafer et al., 1983; Owens et al., 1997). A supernumerary tooth in the maxillary incisor region is probably the most common sort of supernumerary tooth, and these are characteristically petite single-cusped, often conical teeth with a single root (Fig. 5).

Once mineralized, a “tooth” normally has a crown and root, but there are exceptions: A primary tooth in which the root has been completely lysed as part of the exfoliation process is still considered a tooth. So-called root-less teeth (as in dentin dysplasia I; OMIM #125400) also are considered teeth, though roots can be quite abbreviated if present (Fig. 6). Size alone does not define a tooth. The lingual tubercle (talon cusp) in Figure 7 is virtually as large as the incisor crown proper, but it would not be counted as a tooth because (1) it is developmentally a component of that incisor and (2) it has always been united with the incisor. At the other extreme, Figure 8 shows a compound odontoma (de Oliveira et al., 2001), where four distinct tooth-like ‘denticles’ are evident (with the normal but impacted central incisor apical to them). Do these ‘toothlets’ qualify as teeth? They have fully-differentiated enamel and dentin, but no crown-and-root morphology.

Does a “tooth” need to be physically separate from others to be counted? This seems to be an important distinction implied in most studies (Patterson, 1956; Hershkovitz, 1967). For example, cusp-like cingular elements are not counted as teeth. Tubercles, accessory cusps, and styles are considered parts of the main tooth. Cingular elements can be fairly large, but they are almost invariably coalesced with the permanent tooth so there should be no misidentification. These include talon cusps on the incisors, tuberculum dentale on canines, Carabelli’s cusps on the lingual of upper molars, and paramolar tubercles on the buccal aspect of upper and lower molars (Scott and Turner, 1997). All of these cingular elements normally are single-cusped, and they all have at most a single root (e.g., Bolk, 1916). Ambiguity arises when, apparently in rare instances, a dental feature becomes physically separated from the main tooth (Dubuk et al., 1996). Paramolar tubercles do occasionally achieve physical independence when large, and these meet criteria for a “tooth,” namely possession a crown (enamel), root (dentin), and a tooth-like morphology (though simplified).

Overlooking the details of what constitutes a tooth, there are countless anthropological and clinical dental studies of abnormal tooth numbers—either the congenital absence of one or more teeth (hypodontia) or hyperdontia, an excess number of teeth (Table 1). Studies rarely press the definition of a tooth too closely; instead, wording is used such as: hypodontia is a deficit in the normal dental formula or hyperdontia is teeth in excess of the normal dental complement.

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**Fig. 9.** A rare instance of bilateral fusion of the primary maxillary incisors (arrows). (Most cases are unilateral.) Fusion is confirmed by (A) the appearance of confluent tooth forms in each quadrant and (B) the ‘absence’ of independent lateral incisor teeth. Both compound teeth are carious, but their shared enamel, pulp chambers, and root dentin are evident. Radiograph courtesy of Ann S. Smith.

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**TABLE 1. Operational definitions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Definition</th>
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<tr>
<td>Hypodontia</td>
<td>Congenital absence of one to five permanent teeth, generally excluding third molars.</td>
</tr>
<tr>
<td>Oligodontia</td>
<td>Absence of more than five teeth. The study may or may not exclude third molars.</td>
</tr>
<tr>
<td>Anodontia</td>
<td>The complete absence of all primary and/or permanent teeth. The phrase “partial anodontia” (actually denoting hypodontia or oligodontia) is an oxymoron.</td>
</tr>
<tr>
<td>Hyperdontia</td>
<td>Presence of one or more teeth in excess of the species’ normal dental formula.</td>
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Ontogeny

The structure imaged in Szabó’s figures clearly emanates from the premolar’s buccal cingulum, and it shows developmental features in common with the tooth proper. As Szabó et al. point out, there is a common pulp chamber, and the dentin is confluent between the tooth crown proper and the tubercle even though the tubercle has a well-developed root and pulp chamber (Ohishi et al., 1999). It is most probable that this cingular feature was initiated by an enamel knot that, in the presumptive tooth, was located at the cusp apex, which has now (Fig. 2) been abraded or is hidden by subsequent enamel deposition. A primary enamel knot is essential for a tooth’s formation, and later-forming secondary knots define each of a tooth’s cusps (e.g., Jernvall et al., 1994; Thesleff and Jernvall, 1997;

Fig. 10. Radiograph showing fusion between a lower right central and lateral incisor (labial view). The lateral incisor is to the left of the figure. Note the confluence of enamel and dentin between the crowns, though the pulp chambers and roots are separate.

Fig. 11. Example of acquired concrescence between a second and third molar. Roots of the two teeth are only united by cementum; there is no confluence of the underlying dentin.

Fig. 12. A paramolar tubercle on a maxillary left second molar. This tubercle (arrow) clearly is associated with the metacone rather than the molar’s paracone. Bolk (1916) was very keen that paramolar tubercles were only derived from the paracone, though Kustaloglu (1962) showed that this is untrue.

Fig. 13. A rare instance of two paramolar tubercles on a maxillary left second molar (arrow). (No cingular feature could be seen on the contralateral molar.) It appears that both tubercles are attached to the paracone (mesiobuccal cusp), though part of the distal tubercle crosses onto the metacone. Note too a large, single paramolar tubercle on the paracone of the third molar. (Paramolar tubercles rarely occur on permanent first molars.)
The second process involves fusion, where two tooth buds begin to form independently, but, again, for reasons unknown, the formative teeth grow together. Fusion typically starts at the cap or bell stage, so that the united teeth are combined along the lengths of their crowns and roots (Fig. 10). Fusion must involve the dentin, so the twinning is initiated during formation of the outer enamel epithelium (Avery, 1994). The key feature for identification is that, counting the fused pair of elements as one, there needs to be a ‘missing’ tooth elsewhere in that morphogenetic field. This method of defining fusion is not thorough-going, because it supposes that development was disruptive enough to meld two tooth buds, but the same disruption did not cause agenesis of the “missing” tooth. Reliance on the fused tooth morphology can be a help here, but convincing discrimination between fission and fusion may be impossible from inspection of the end product alone.

A rare but classic case of tooth fusion is in people (and laboratory animals) with developmental midline problems, notably holoprosencephaly (HPE). HPE is the embryological failure of divisions of the head to form along the left-right, transverse, and/or craniocaudal axes (Cohen, 2001). A remarkable dental consequence of this heterogeneous group of anomalies can be a solitary median maxillary central incisor (SMMCI). Nanni et al. (2001) provide a current review of this condition. Experimental work shows that sonic hedgehog (shh), a signaling protein, is critical for the initiation of a tooth germ, probably by directing epithelial cell proliferation. In mice, the absence of shh can either prevent maxillary incisor formation (congenital absence) or cause these incisors to fuse. The maxillary central incisors begin formation close together and these tooth germs coalesce into a single symmetric central incisor (Hardcastle et al., 1998). Of note, the molar teeth are unaffected. Alterations in the structure-function of shh provide the common etiology between the head (central nervous system) and tooth anomalies (Cohen, 2004).

Aside from fusion and fission (gemination), yet a third situation occasionally occurs, namely concrescence. Pindborg (1970) valuably distinguishes between true concrescence and acquired concrescence. Acquired concrescence occurs when two fully formed teeth are only united by the fluorescence of cementum (Fig. 11). Colby et al. (1961:42) note that two factors are required here, (1) the teeth, specifically the roots, of adjacent teeth

activated regulators are intensified in interplacodal regions, which inhibit tooth formation and, thus, account for the orderly spacing of teeth. Gemination might, then, be viewed as an exception where two sets of signals are preserved (or initiated) within the same dental sac that, then, gives rise to ‘twinned’ but fused teeth. Geminated teeth (more common in the primary dentition) usually have a shared root and shared pulp cavity.

These morphological components that are developmentally important to tooth formation are not considered, though, as being independent of each other. That is, the fused teeth develop from the same set of signals, and, as such, their number cannot be increased through fusion. Twinning needs to occur during the cap or bell stage prior to crown mineralization, but the actual process is not understood. A critical feature defining geminated teeth is the presence of all of the other teeth in the morphogenetic field, so the twinned teeth clearly are not fused teeth (Fig. 9). Twinning requires duplication of the biochemical signals for tooth development within the dental sac. This occurs at the cap or bell stage, not the crown stage.
need to be in close proximity and (2) hypercementosis—excessive cementum deposition—unites the proximate roots. Acquired concrescence is only distantly related to the fission and fusion of teeth because it occurs after tooth formation in contrast to being the consequence of some developmental aberration. In contrast, true concrescence involves confluence of the roots (dentin) of adjacent teeth, so it is a sort of fusion.

**Paramolar tubercles**

Numerous researchers have described “paramolar tubercles,” a term coined by Bolk (1916:110). Bolk surveyed some 30,000 skulls, so he is still a contender for the record number of identified tubercles. Bolk argued that these tubercles on maxillary molars always develop from the mesial cusp. In fact, they can arise from the cingulum of either buccal cusp, and Kustaloglu (1962) notes that they therefore should be labeled parastyles (mesiobuccal) or metastyles (distobuccal) depending on the cusp of origin. Figure 12 shows a characteristic expression, where the tubercle developed buccal to the metacone, well distal of the lingual developmental groove that demarcates the union of the paracone and metacone. Figure 13 shows a second molar with two equal-size paramolar tubercles, and it appears that both developed from the tooth’s paracone.

Such buccal tubercles are less common in the mandible, where, occasionally, they develop from the mesiobuccal cusp, thus making them protostylids (e.g., Dahlberg, 1950). Protostylids occur frequently enough that there is an ASU dental plaque to score their size (Turner et al., 1991; also see Hlusko, 2007; Skinner et al., 2008). Paramolar features also can occur on the premolars. Figure 14 is reproduced from Kustaloglu’s article, showing a large tubercle on the facial aspect of the paracone (buccal cusp) of a maxillary premolar; this example is not dissimilar from the example described by Szabó and coworkers (Fig. 1).

**Hyperdontia**

Various ideas have been put forth over the years to explain why a supernumerary tooth might occur. Some of these are noted in the reviews by Rajab and Hamdan (2002), Botra et al. (2005), and elsewhere. These conjectures are of historical interest, but they comport poorly with current knowledge of the molecular control of tooth formation (e.g., Mitsiadis and Smith, 2006). A popular idea was atavism, which is the idea that some phylogenetic ancestral condition (where more teeth were the norm) is being re-expressed. Recall, for instance, that the baseline mammalian condition was at least 44 teeth (Gregory, 1922; Ji et al., 2002), and the human dental formula involves reductions of all tooth types except the canines (see review by Peterkova et al., 2006). Another conjecture was that one or more of the normally-occurring teeth splits (the dichotomy theory) to produce additional teeth (Foley and Del Río, 1970; Taylor, 1972). Another idea with some persistent credibility involves an extension of dental lamina at the end of the tooth row that is induced to form an additional tooth (Saarenmaa, 1951), but this idea must include the reciprocal epithelial-mesenchymal inductions that promote tooth formation, “extra” dental lamina in itself does not cause teeth to form. Such historical conjectures suppose that extra teeth are due to additional developmental activity, with the term “hyperactivity” often used in some vague sense to explain the over-production of teeth.

Recent evidence suggests that the opposite is true—that biochemical signaling is responsible for stopping the enumeration of teeth and is necessary for holding a species’ dental formula in check. A prime example is now known in some detail for humans: Runx2 is a transcription factor that is key for osteogenic cell differentiation (Ziros et al., 2008). Mutations of Runx2, which also is known as Cbfa1, can cause cleidocranial dysostosis (CCD; OMIM #119600), the condition that is, perhaps, archetypical of hyperdontia in man (Jensen and Kreiborg, 1990; Whittington and Durward, 1996).

People with CCD are likely to exhibit hyperdontia, especially in the premolar region (along with systemic problems of non-eruption due to a failure of bone resorption ahead of the erupting tooth). CCD shows the important role that Runx2 normally plays in preventing excess budding of the dental lamina. However, hyperdontia in people with this autosomal dominant allele show variable expressivity, ranging from no extra teeth to cases with numerous extra teeth. The percentage of cases of CCD with hyperodontia is around 1/5, showing that even in this archetypical condition, the formation of extra teeth is uncertain—presumably due to differences in allelic conditions and differences in genetic backgrounds.

Comparably, Kantaputra and coworkers (2008) describe a single subject with unerupted teeth in the premolar-molar region evidently due to an inherited defect in Trps1 causing gain of function. These authors suggest that this mutation mimics the dental phenotype of persons with Runx2.

Murashima-Suginami et al. (2007, 2008) show that up-regulated bone morphogenetic protein (BMP) signaling causes supernumerary tooth formation in mice, notably in the incisor region (also see Kassai et al. 2005). They interpret their experimental results as showing that odontogenic mesenchymal cells normally are killed off at the end of tooth rows because antagonists to BMP play a crucial role in controlling the enumeration of tooth buds. When an antagonist (termed ectodin or, synonymously, “uterine sensitization associated gene-1” or USAG-1) is absent, BMP function is left uncontrolled, and the result is supernumerary tooth formation. That is, ectodin normally binds to BMP
and inhibits its function; without inhibition, BMP can promote additional tooth sites.

These studies show that extra teeth result from inadequate suppression of tooth-forming capacity, not the over-activity of tooth-promoting events.

At this point in our understanding of tooth-promoting conditions, it is unknown (A) how many genes (alleles, proteins) are responsible along the involved pathway of tooth development either to form an extra tooth or curtail formation of a normal tooth, (B) whether extra teeth at the end of the dental lamina (e.g., mesiodens, fourth molars) are due to the same causes as those within the tooth rows (such as the common extra premolars). Other issues of interest involve (A) how and why human population differences in hypodontia and hyperdontia have developed, (B) what causes the persistent sex differences (hypodontia is more common in females; hyperdontia is more common in males) seen among humans, and (C) why the locations of missing and extra teeth differ among human groups. It also is effectively unknown how the environment affects any or all of these differences. It seems unlikely that there are simple or single, all-inclusive reasons for any of these issues. It is noteworthy that most supernumerary teeth are few in number within and among subjects, undersize and morphologically simplified. Overall, it seems to be a major genotypic effort to increase tooth number, perhaps because of the large number of necessary steps needed to form a tooth.

Studies of supernumerary teeth in laboratory animals have certainly been informative (D’Souza and Klein, 2007). Several studies show that perturbations of signalling molecules—either genetic knockouts or the overproduction of certain molecules—can cause the formation of extra teeth. For example, mice that over-express ectodysplasin (Pispa et al., 2004) or under-express antagonists to FGF (fibroblast growth factor; Klein et al., 2006) can produce supernumerary teeth. But, it is important to question the relevance of these findings to humans. Mice—the favored animal for studying tooth development—have a diastema in each quadrant where formation of lateral incisors, canines, and premolars is suppressed. However, several of these teeth initiate formation but are arrested and resorbed in the bud stage (Peterkova et al. 2002, 2006) so the “rescue” of these tooth buds to permit them to develop into “supernumerary diastema teeth” is of considerable interest, but it is fundamentally different from the human condition where no primordia normally form.

**Laterality**

Another question raised by Szabó’s case report is why their tubercle occurs unilaterally. Conventional wisdom is that the genotypic information is the same in the left and right hemispheres of the body (Polak 2003), so disparate phenotypes between quadrants are supposed to be the exception rather than the rule. Researchers familiar with dental morphology recognize that, while left-right symmetry may be the norm, even striking exceptions are not hard to find. Alvesalo and coworkers (1975) suggested that, for Carabelli’s trait, expression on one tooth is always associated with some expression on the contralateral tooth, but this has not been my own experience. Kustaloglu (1962) examined the osteological collections at the Chicago National History Museum (roughly 500 individuals) and found that paramolar tubercles tend to occur unilaterally more often than bilaterally, with a ratio of 18:4 among the permanent molars, though bilateral occurrences predominated in the primary dentition.

Dental anthropologists have embraced the idea that morphologic dental traits have a quasicontinuous mode of inheritance. Supporting evidence stems primarily from animal studies (e.g., Grüneberg, 1950, 1952) because few anthropological studies have subjects of known biological relationship (cf. Saunders and Mayhall, 1982; Sjøvold, 1996). The quasicontinuous (QC) model of inheritance suggests that morphological dental traits are under polygenic control, but with a threshold below which the feature is not expressed (Wright, 1934a,b; Falconer, 1965). The question arises whether unilateral expression (Fig. 1) is indicative of the subject’s genotype being close to the threshold. That is, subjects with a genotype for trait expression might be prone to expressing the trait unilaterally due to local environmental vagaries between the jaw’s quadrants. Supposition is that genotypes farther above the threshold would be more likely to exhibit bilateral symmetry. This aspect of a QC model does not seem to have been tested for dental traits.

**OVERVIEW**

In sum, my contention is that the case described by Szabó and coworkers is a paramolar tubercle on the lower left first premolar, and, thus, should be labeled a protostylist. It is possible that this cingular feature developed from local trauma or infection, which would account for its unilateral expression, though that is sheer speculation. This tubercle is unquestionably a developmental feature of the premolar itself, as its union (shared enamel, dentin, and pulp cavity) precludes it being a supernumerary tooth.

These comments are set forth in hopes of stimulating discussion among readers regarding this interesting case.

**LITERATURE CITED**


369:1012-1016.
Wright S. 1934b. The results of cross between inbred strains of guinea pigs differing in number of digits. Genetics 19:537-551.

This is the third book in Springer’s series on Vertebrate Paleobiology and Paleoanthropology; it consists of the proceedings of the first symposium on Human Evolution held at the Max Planck Institute for Evolutionary Anthropology (Leipzig, Germany). The volume illustrates the diverse and innovative ways that teeth inform our understanding of human evolution. Recent advances in the analysis of dental morphology, microstructure, development, and wear are showcased with respect to how they have increased knowledge of hominin phylogeny, ontogeny, and adaptation to changing dietary environments. An introduction to the volume by Simon Hillson provides a synopsis of key themes and unique perspectives presented in each chapter. The four main sections of the volume begin with an introductory chapter by scholars that have made a significant impact on the field. These introductions provide useful analytical summaries of each contribution and place them in the broader context of research in dental anthropology and paleoanthropology. Some, such as Fred Grine’s introduction to Part IV: ‘Dentition and Diet’, which focuses on dental macro- and micro-wear, is comprehensive, historical, and well referenced - with 135 citations. Others, including Wood’s introduction to Part III: ‘Dental Development’ are brief, yet highlight key features of each chapter in the section. A bit perplexing is Macchiarelli and Bailey’s introduction to Part II: ‘Dental microstructure and life history’, where on several occasions the reader is uncertain which author’s observations and opinions are being presented (‘in my view’, ‘I would also like to note’, ‘in my personal view’). A brief synopsis of each of four section of the volume follows.

Part I: ‘Dental evolution and dental morphology’, contains seven chapters, and begins with Pilbrow’s analysis of occlusal odontometric variation in great ape molar teeth. Results indicate that great ape molar metrics exhibit patterns of inter-species and sub-species taxonomic diversity. Despite small sample sizes, lack of understanding of inter-trait associations, and use of a classification system designed for scoring modern human tooth crown morphology, Bailey and Wood explore the evolutionary divergence of the Homo and Paranthropus lineages using post-canine morphometric variation. They find that increased dental crown complexity in Paranthropus is not a primitive retention and that dental trends said to be characteristic of Homo actually appear relatively late in human evolution. Maxillary molar cusp morphology of South African australopithecines is analyzed by Moggi-Cecchi and Boccone who find similarities (in crown base areas) and significant differences (in relative area of anterior cusps and molar size sequences) between A. africanus and A. robustus. Crown morphology of fossil samples from Gran Dolina (TD-6) and Sima de los Huesos are used by Martinón-Torres and colleagues, to assess phylogenetic issues related to the early colonization of Europe. They conclude that a coordinated assessment using biological and cultural evidence holds promise.

An innovative technique—neural network analysis using Self Organizing Maps—for describing dental morphology is used by Manni and colleagues to evaluate the relationship between archaic and modern Homo sapiens. Though it has some advantages, this new technique may have limitations that preclude its adoption by other investigators. The final two chapters in Part I focus on exciting new, non-destructive advances in imaging dental structures and tissues. Olejniczak and associates discuss methodological aspects of 3D data acquisition by micro-computed tomography of primate molar teeth. Precise and reliable portrayal of the enamel-dentine junction and measures of enamel cap thickness are tightly linked to methodological parameters such as slice thickness and pixel resolution. The advantages of high resolution X-ray computer tomography (HRXCT) for obtaining digital 3D data and volumetric properties of dense tissues, is reviewed by Gantt and colleagues.

Five chapters comprise Part II: ‘Dental microstructure and life history.’ This section begins with an analysis of dental microstructure, growth and life history of Megaladapis, providing estimates of gestation length, molar crown initiation, formation and completion times and minimum emergence ages for M1 and M2. Schwartz and colleagues find that molar development is rapid and poorly explained as a function of adult body mass. Microstructural indicators of dental development in a single female specimen of Pan paniscus are described by Ramirez-Rozzi and LaCruz. Preliminary results from the analysis of perikymata and striae counts reveal high appositional rates and short crown formation time for I1 while molar crown formation time is similar to that of the common chimpanzee.

New data on chimpanzee and human molar crown development are presented by Tanya Smith and associates, who document variation in incremental features within and between genera. Within cusp types humans show greater average cusp formation times than chimpanzees due either to thicker cuspal enamel and/or higher mean periodicity values. High variability in cusp formation times and overlapping ranges raise concerns for interpreting small samples. Enamel microstructure of Australopithecus africanus
is documented by Bromage and colleagues, who employed a portable confocal scanning optical microscope to circumvent analytic issues such as limited magnification and specimen preparation. Cross-striation periodicity and data on striae-EDJ angles are presented and crown formation time for a single molar (STW 284, M2) is estimated at between 3.0 and 3.2 years. In the final paper in this section, Guatelli-Steinberg and associates compare imbricational enamel growth in the anterior teeth of Neandertals and three modern human groups from diverse eco-geographic settings. While no significant difference was found in imbricational enamel formation times for anterior teeth, differences were evident in the shape of growth curves (from cusp tip to cervix) and in mean perikymata numbers across anterior tooth types.

Part III is devoted to ‘Dental development’ and consists of four chapters spanning dental genetics and tooth size, dental development sequences, inter-group variation in calcification stages and new methods for reconstructing dental ontogeny. Tooth size variation in outbred pedigreed populations of baboons and mice were used by Hlusko and Mahaney to test expectations derived from dental field theory. In mice, incisor size appears to be genetically independent of molar size, and circumstantial evidence from fossils suggests that some level of independence exists in the expression of anterior and post-canine tooth size in primates. Braga and Heuze introduce the concept of modularity to assess interactions between inter-dependent elements in growing dentitions. They observe considerably greater plasticity and variability in development timing of incisors than of other teeth and advise caution in using incisor teeth as a reliable substitute for other permanent teeth in the interpretation of fossils. Preliminary results from an on-going analysis of permanent molar calcification stages (M1 and M2) in African-American and European-American children are presented by Monge and associates, who find evidence of earlier maturation among children born in the 1990s. A re-evaluation of what constitutes ‘normal’ dental development and greater appreciation for the range of plasticity in dental calcification is encouraged.

Serial micro-CT scans are used by Smith and colleagues to reconstruct the topography of the dento-enamel junction and quantify cusp volume and relationships during successive stages of development. This research suggests that spatial relationships consist of shape differences that are established early in morphogenesis by differential development within the tooth germ, and that differences in cusp size and proportions are modified at the crown surface by enamel apposition.

Dental wear and elemental ratios in fossil hominin and modern human teeth are addressed by five diverse contributions to Part IV, entitled ‘Dentition and diet”. An innovative method known as laser ablation inductively coupled plasma mass spectrometry (LA-ICP-MS) was used by Humphrey and colleagues to determine changes in Sr/Ca ratios across the neonatal line in deciduous teeth of formula-fed and breast-fed children. Marked reduction in Sr/Ca ratios were detected across the neonatal line in breast-fed children but not in formula-fed children, a result that holds promise for interpreting the chronology of dietary transitions in infancy and early childhood. Tooth crown topography, a landmark-free, 3D method of describing crown morphology, is employed by Ungar to show that differences in diet can be inferred from worn teeth in extant apes, that species-specific wear patterns allow inferences of function from form in worn teeth, and that differences in molar crown topography in Paranthropus and Australopithecus suggest differences in diet and fallback foods.

A retrospective review of past accomplishments and vision of future developments in the field of dental microwear is provided by Teaford, who regards ‘low magnification’ methods and scale-sensitive fractal analysis as ‘next steps’ in this rapidly developing field. Ulhaas and colleagues employ 3-D analysis of occlusal surface wear to comparatively assess variation in three hominin taxa: *A. afarensis, A. africanus, and Paranthropus robustus*. Using a portable optical triangulation scanner, inter-specific differences in the mode of reduction in occlusal relief was responsible for enhancing variation in wear facet orientation, an observation that implies low levels of interspecies competition for food. In the final chapter of the book, Estebananz and associates use micrographs (SEM) and 3-D topographic images of molar buccal surfaces to characterize striation density and enamel surface roughness in three extant and three fossil hominin taxa. Postmortem surface damage and automated data acquisition were considered in this study which found a clear and significant association between some measures of enamel roughness and microwear pattern, a finding of value in inferring diet.

Overall, I found the volume a valuable review of emerging methods and new approaches to the use of dental morphology, microstructure, development, and wear in unraveling critical issues in human evolution. The hominin focus of the volume, made some chapters (Part II, chapter 2: lemur dental development; Part III, chapter 2: quantitative genetics of mice and monkeys) seem either out-of-place, or a refreshing departure from the main theme. The book is top-heavy with introductions (to the volume and then again to each individual section), yet lacking in summary, synthetic or integrative perspectives either by section, or for volume as a whole. This is an unfortunate omission. Though diverse in their objectives and methods, the contributions to this volume exhibit significant overlap in the questions posed and the results derived. A
comparative assessment of contributions, followed by a summary of the issues and themes that were consistently affirmed, as well as those on which divergent interpretations exist, would have been a valuable service to the reader.

As with many edited volumes, contributions are variable though in different ways; some chapters fail to yield definitive conclusions due to limitations of either sample size or methodology or both; while others present innovative and potentially useful analytical methods that suffer from operational complexity limiting their adoption by other investigators. Finally, it’s sad that a volume devoted to cutting-edge technology contains so many annoying errors. For example, some text citations are missing from the References in the introduction to Part I (page 5, Martin-Torres et al., 2007; and Kono, 2004). Elsewhere (Part I, chapter 5), text references to illustrations are incorrect: a) on page 70, in discussing lower second premolar morphology, the reader is referred to Figure 3, which illustrates lower second molar occlusal surfaces. Again on page 73, in discussing molar cusp number, the reader is referred to Figure 5 which illustrates lower first premolar teeth. Sloppy editing, or inept use of the spell-checker, results in some awkward sentences; for example, on page 188, we read “... when discussing variation in enamel developmental, ...” and “In light of this, ...”, and page 189, “... If is unclear why this population ....”

Researchers, teachers and graduate students in human and dental evolution, and possibly in allied clinical fields, will find the volume an indispensable and essential aid in keeping abreast of current developments in dental anthropology. However, given the rapid rate of change in method and theory in dental paleoanthropology, I’m concerned about the shelf-life of books devoted to cutting-edge issues and technology that require a significant financial investment.

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DAA Subscription

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Original Articles

Denice Higgins, Toby E. Hughes, Helen James, Grant C. Townsend
Strong genetic influence on hypocone expression of permanent maxillary molars in South Australian twins .......................... 1

Edward F. Harris
Patterns of hypodontia among third molars in contemporary American adolescents ........................................... 8

Gy. Szabó*, G. Kocsis S., E. Molnár
Joined supernumerary mandibular teeth in the premolar region: Report of a Hungarian archeological case ......................... 18

Commentary, Edward Harris .......................................................... 21

Book Review

John R. Lukacs
Dental Perspectives on Human Evolution: State-of-the-Art Research in Dental Paleoanthropology. ................................. 30