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The reflection of childhood growth episodes in adult morphology

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Introduction

Human growth is not a mere proportional enlargement of all parts of the body. It is a well-known fact that some structures complete growth early in life, whereas other parts of the body continue to grow well into adulthood. These patterns of structural maturation have long formed the basis for skeletal age determination. In this article, patterns of growth are applaied to a study of the life of past populations. The research was carried out as part of a study aiming at a general description and understanding of patterns of, and determinants for, growth in historical populations in Scandinavia.

The present study is focused on growth in the 3-6 age group. Growth at this stage of life is generally described as rather slow, linear and proportional. It is known (Clark et al. 1986) that the development of soft tissues is more sensitive to growth disruption than that of osseous tissues. Among dental and skeletal tissue, the neuro-osseous tissues are the most vulnerable ones; the vertebral neural canal is thus easily stunted (Brooke et al. 1984, Platt & Stewart 1962).

During this age interval several teeth complete calcification, and the neural arch of the thoracal vertebrae fuses (Roaf 1960, Hinck et al. 1966, Clark 1985). Afterwards the dimensions of the vertebral canal are regarded as more or less stable (Porter et al. 1980), and later vertebral growth primarily takes place in the vertebrate body (McKern 1970). Thus, the morphology of teeth and the neural arch might contain information on episodes of retarded growth among infants.

Material and methods

The empirical background is the skeletal sample from the Tirup cemetery (near Horsens, East Jutland, Denmark). The cemetery is believed to have been founded around A.D.1100, and burials ceased during the first quarter of the 14th century. Some 600 graves were found during the excavation in 1984 (Kieffer-Olsen et al. 1986). The site is located in the country-side, and the population that once lived there was undoubtedly rural.

Only adult individuals have been examined in the present analysis. Some 70 skeletons were sufficiently well preserved to be included. The criterion for inclusion was that the following three measurements could be taken of the skeleton:

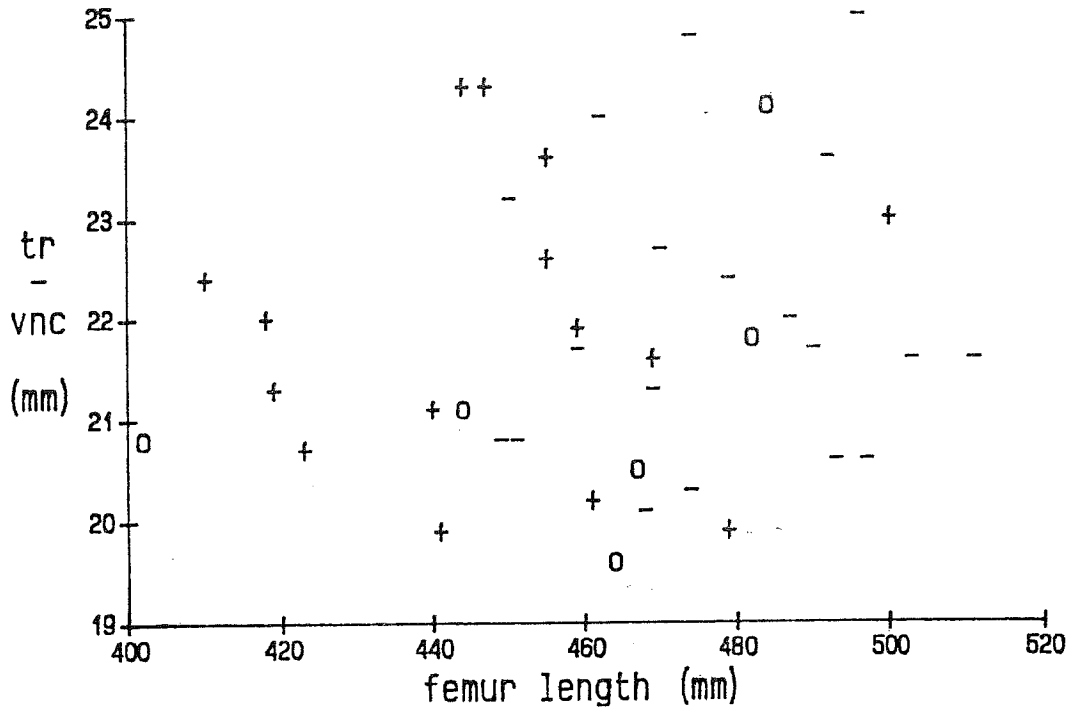

Fig. 1. Males from the Tirup cemetery, Jutland, Denmark. Simultaneous distribution of the transversal diameter of the vertebral neural canal (definition by Eisenstein 1983) of the first thoracal vertebra and femur length (M1, Martin & Saller 1957). Enamel hypoplasia noted (+), hypoplasia not present (-), hypoplasia not observable (0).

1.The maximum length of the femur (if both femora were preserved, the average of the two was used). Measured on an osteological board to the nearest mm. (M1, Martin & Saller 1957)

2.The transversal diameter of the vertebral neural canal (tr-vnc) of the first thoracal vertebra. Measured with a sliding caliper to the nearest tenth of a mm (Eisenstein 1983).

3.The posterior vertebral body height (p-vph) of the first thoracal vertebra. Measured with a sliding caliper to the nearest tenth of a mm (Ericksen 1976).

While investigating the skeletons, we noted dental enamel hypoplasia. If there were marked linear depressions, the individual was scored as having hypoplasia (+); otherwise no hypoplasia was noted (-).

The analyses were carried out as inspections of scatter plots, different markings being used for the different scores of hypoplasia. Most of the numerical analyses of these data were performed as univariate statistical analyses. However,

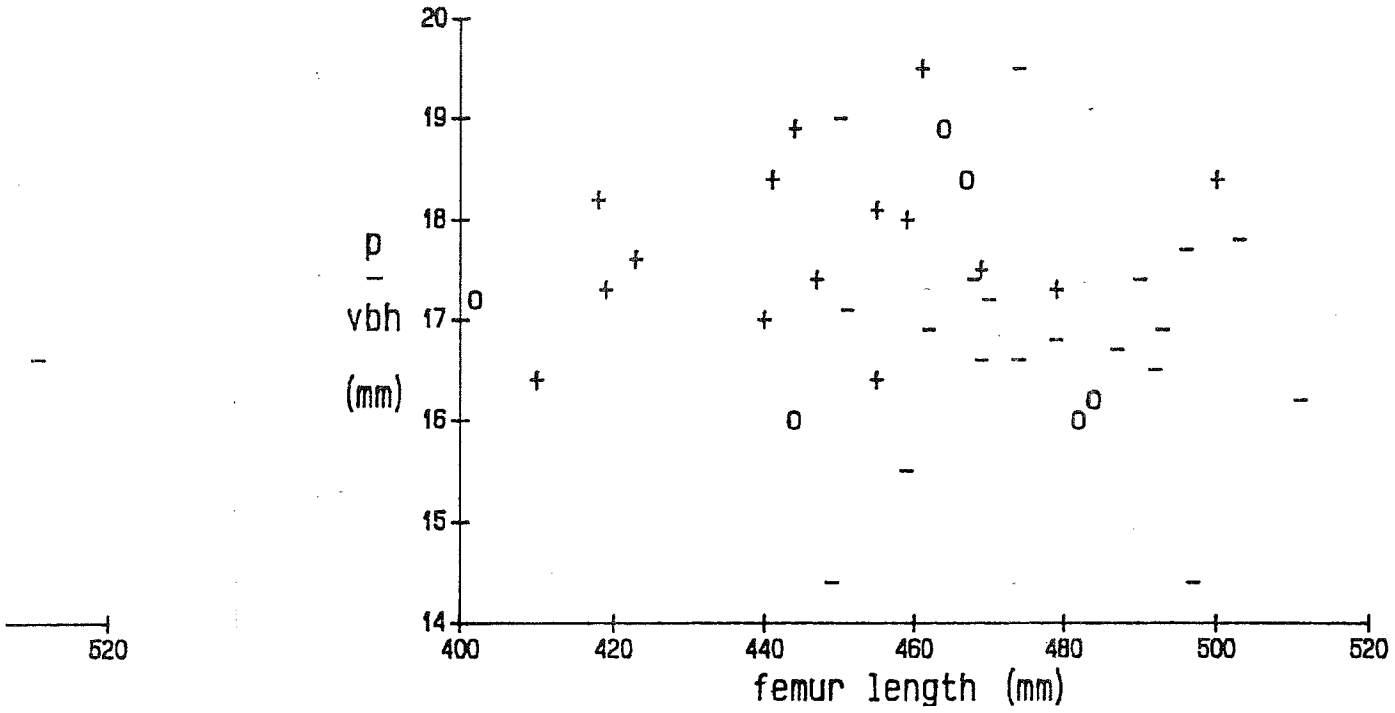

Fig. 2. Males from the Tirup cemetery, Jutland, Denmark. Simultaneous distribution of the posterior body height (definition by Ericksen 1976) of the first thoracal vertebra and femur length. Enamel hypoplasia noted (+), hypoplasia not present (-), hypoplasia not observable (0).

multiple regression analyses were carried out with the aid of the GLIM (3.77) statistical package.

Results

The first of the scatter plots (Fig. 1) illustrates the simultaneous distribution of femoral length and the transversal diameter of the vertebral neural canal of the first thoracal vertebra (tr-vnc). Evidently, there is a weak correlation between these two measurements (r=0.13, n=40, t=0.81, P>0.4). This finding is in agreement with earlier observations by Clark et al. (1986). The same holds true if the correlation for the given hypoplasia score is estimated (r=0.044, n=34, t=0.24, P>0.8). The most interesting feature to emerge from Figure 1 is the fact that men with enamel hypoplasia have considerably shorter femora than men without enamel disturbances (averages 448.0 versus 477.6 mm t=3.99 P<0.001). There is no evidence, however, of a "hypoplasia effect" on the dimension

of the vertebral canal (tr-vnc) (averages 21.9 versus 22.0, t=0.16, P>0.8).

Figure 2 illustrates the simultaneous distribution of femoral length and the posterior vertebral body height (p-vbh) of the first thoracal vertebra. In this case, there is no indication of a biologically important correlation between the two measurements (r=-0.13, n=40, t=0.83, P>0.4). The significant hypoplasia effect on femoral length is visible in this plot, too. Further, the second coordinate (p-vbh) is significantly differentiated by the hypoplasia score (averages 17.76 versus 16.87 mm, t=2.41, P<0.05).

The metrical, and thus numerical, difference between the size of the neural canal and the body height of the vertebrae (tr-vnc minus p-vbh) can be seen as a negative measure of growth after this infant period. The simultaneous distribution of femoral length and this difference is shown in Figure 3. It appears that individuals with hypoplasia (+) have smaller values for this difference than individuals without hypoplasia (-)(averages 4.03 versus 5.23 mm, t=2.20, P<0.05). The correlation between the coordinates in Figure 3

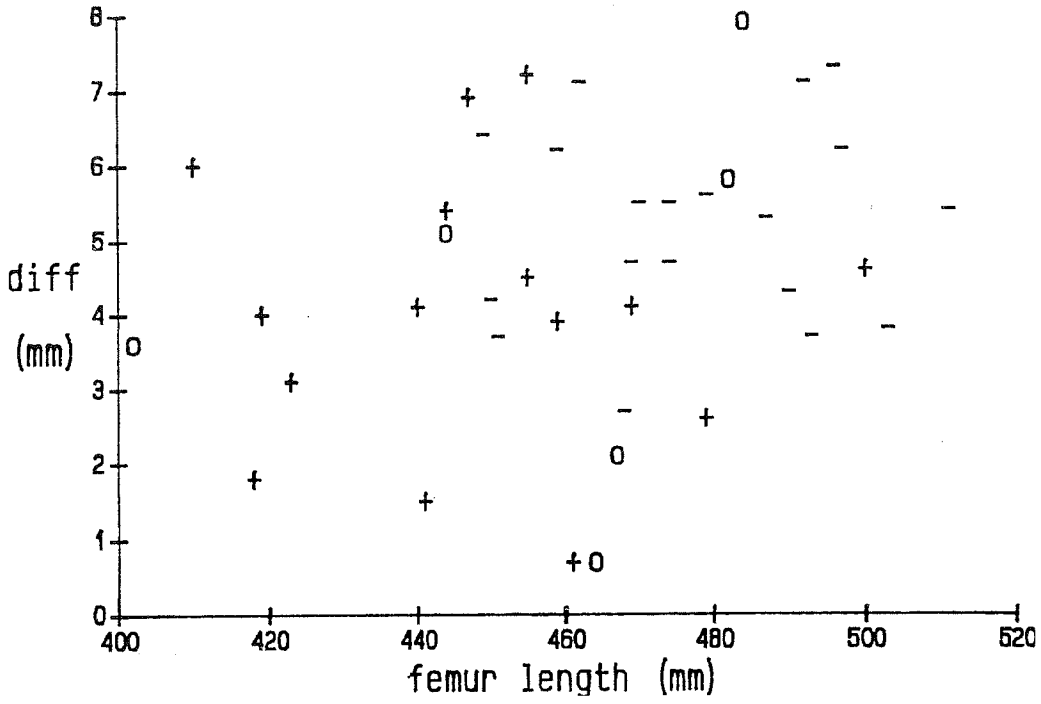

Fig. 3. Males from the Tirup cemetery, Jutland, Denmark. Difference between the transversal diameter of the vertebral neural canal (tr-vnc) and the posterior vertebral body height (p-vbh) related to femur length. Enamel hypoplasia noted (+), hypoplasia not present (-), hypoplasia not observable (0).

is not statistically significant (r=0.25, t=1.58, P>0.1).

The simultaneous distribution of the two vertebral dimensions is presented in Figure 4. The overall correlation between these two measurements is insignificant (r=0.12, t=0.76, P>0.4). However, among the 19 individuals without hypoplasia, the correlation is significant and positive (r=0.53, t=2.60, P<0.02).

Discussion

The present article is focused on evidence indicating episodes of growth retardation visible in adult male skeletal morphology. Males were chosen as the initial focus in a more general study of factors affecting growth, as males are generally more susceptible to environmental factors than females (Wolański 1980). The presence of dental enamel hypoplasia formed during the studied age interval is regardedas evidence of episodes of environmental stress (disease) which might have led to growth retardation.

The significant shortness of the femora of individuals with hypoplasia supplies

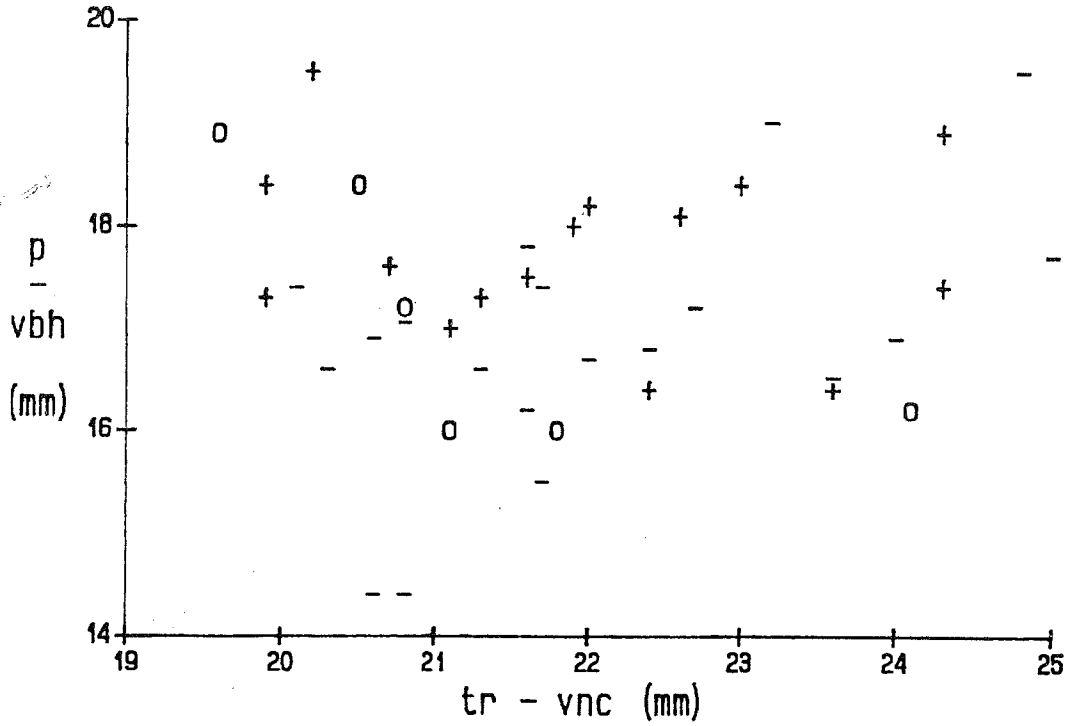

Fig. 4. Males from the Tirup cemetery, Jutland, Denmark. Simultaneous distribution of the posterior vertebral body height (p-vbh) and the transversal diameter of the vertebral neural canal (tr-vnc). Enamel hypoplasia noted (+), hypoplasia not present (-), hypoplasia not observable (0).

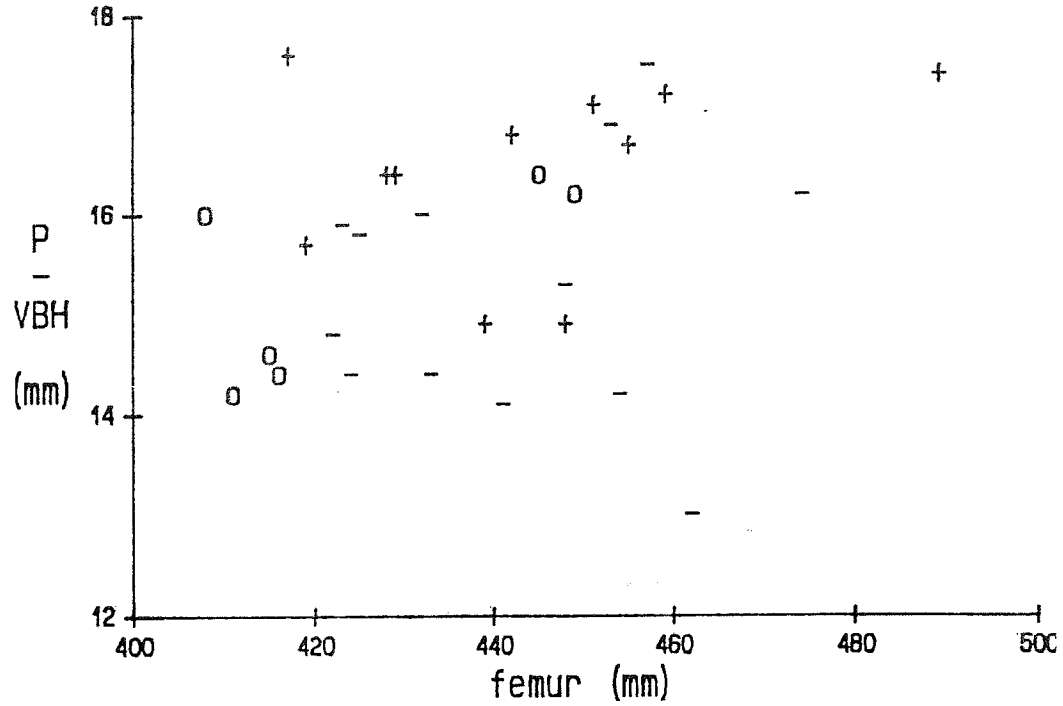

Fig. 5. Females from the Tirup cemetery, Jutland, Denmark. Simultaneous distribution of the posterior body height of the first thoracal vertebra and femur length. Enamel hypoplasia noted (+), hypoplasia not present (-), hypoplasia not observable (0).

evidence of a growth-retarding effect produced by environmental stress experienced between the ages of 3 to 6 years. It appears that the enamel hypoplasia score accounts for as much as 30 per cent of the total variance for femoral length. If this observation is verified by more comprehensive studies, it must lead to two conclusions of supreme importance to the understanding of human morphogenesis: first, it appears that femoral length is more susceptible to environmental influence than adult body height; secondly, early childhood is the period of life in which the environmental component of adult height is formed.

The plots indicate that the ordinary normal distribution model for the analysis of biometric variation is not suited to the extraction of biologically important information on patterns of morphological covariance from data like the ones presented here. At least two of the three analysed measurements are heavily affected by a more or less discrete environmental factor active in the studied age interval. Femoral length is often used as an indicator of total skeletal size, and this

it is the most frequent measurement for height prediction. The factor which produces enamel hypoplasia reduces the length of the femur. However, the same factor increases the height of the vertebral body.

The pattern of conflicting effects on different osseous structures suggests an interpretation of the observed variation. It appears that inhibited femoral growth during early childhood cannot be regained at a later age. However, catch-up growth following a growth-retarding incident in pre-school-age children has been widely described (Eveleth & Tanner 1990). This means that other structures than the long bones must grow excessively in order to compensate for the height reduction caused by the shorter legs.

It is probable that the posterior vertebral body height (p-vbh) is closely correlated to the total length of the spinal column. If this is indeed the case, the increased vertebral body height of individuals with hypoplasia is an indication of catch-up growth.

The data on the females from the same cemetery show no evidence favouring an effect of hypoplasia on femoral length. However, the posterior vertebral body height (p-vbh) is significantly increased in women with dental hypoplasia (averages 16.46 versus 15.27 mm, $t=2.59$, $P<0.02$)(Fig. 5). This indicates that female femoral length is less affected by environmental stress than male femoral length. The significant hypoplasia effect on female vertebrae dimensions (p-vbh) indicates important catch-up growth in both sexes. In a way, our findings contradict other observations on growth versus environmental conditions during childhood. Lasker and Mascie-Taylor (1989) found that the height of children at ages 7, 11 and 16 years in England today varied along with social class. They also reported that growth for height after the age of 7 was virtually unaffected by social-class differences. Their results would predict a strong positive correlation between femoral length and the dimensions of the vertebral neural canal. This was not found by us (Fig. 1).

The conclusions indicated in this article must be seen in the light of the limited amount of data behind them. However, we do feel that our observations facilitate the entrance of new arguments into the debate on nature versus nurture. These conclusions are by no means final solutions to the problems they have a bearing upon; but they can facilitate the formulation of new research topics of direct relevance to the factors governing human growth.

Acknowledgements

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