ANTHROPOLOGIE • XXXIII/1–2 • pp. 107–124 • 1995

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VARIATION IN PATTERNS OF HUMAN GROWTH AND DEVELOPMENT: ITS SOURCES AND CAUSES

ABSTRACT: The main purpose of this article is to provide a descriptive characterisation of human growth patterns in an holistic manner. Employing an evolutionary and ecological perspective, the uniqueness and similarities of growth and development between Homo sapiens and other primate species have been pointed out. These characteristics serve as a background for a detailed analysis and interpretation of all sources of variation in human growth patterns. The intrinsic and extrinsic factors that cause, control and regulate individual development have been integrated into a theoretical model and estimated numerically with appropriate mathematical approaches. Empirical data used for the statistical elaboration is derived from the Poznań Growth Study carried out longitudinally since 1985. The data consists a sample of children born in 1980 and examined anthropometrically in semi-annual intervals. The sample consists of 284 boys and 270 girls.

KEY WORDS: Primates – Causal factors controlling growth – Mathematical handling of growth pattern – Variation in human growth patterns

THE SHARED AND UNIQUE FEATURES OF HUMAN GROWTH IN COMPARISON WITH NON-HUMANS

A number of taxonomic schemes for classifying which human attributes are important for the uniqueness of Homo sapiens have already been proposed (Leakey, 1981; Lovejoy, 1981; Lewin, 1984; Smith, Spencer, 1984; Pilbeam, 1992). However, there are some characteristics of Homo sapiens which are more often indicated by paleoanthropologists as defining features: bipedality, large neocortex, reduced anterior dentition with molar dominance, complex technology and cultural behaviour, including language. Unique sexual and reproductive behaviour is also indicated as a defining feature. The pervasive nature of bipedal adaptation can be seen in a few basically anatomical features, such as limb proportions, joint structure, the anatomy of the hand, feet, pelvis and spine.

The development of each of these characteristics can be observed in the ontogenetic unfolding of the human pattern of growth and development. For instance, the differential growth of the legs and pelvis versus the arms and shoulder gridle makes walking on two limbs possible. Upright position and walking on two limbs are specific for humans.

The unique features of human development have been characterized mainly by the evolution of the childhood growth period, which results in a prolonged period of infant dependency and a rapid, large acceleration in growth velocity at adolescence leading to a physical and sexual maturation. Bogin (1992) lists the benefits of this unique pattern of human development: a prolonged childhood makes possible an extended period for brain development, time for the acquisition of technical skills such as tool making and food processing, and time for socialization, play and the development of complex social roles and cultural behaviour.

According to Pilbeam, the most important characteristic of human biology is an opportunity for the evolution of genetic structure, "... which permits an apparently endless variety of cultural behaviour..." (Pilbeam, 1992:62). Human cultural behaviour is controlled and patterned in a unique way, usually by its use of symbols (spoken and written languages). The symbolic abilities of man, which are many and pervasive, have been found to be peculiarly human attributes.

The described characteristics are some of the essentially unique patterns of human development. Although they set Homo sapiens apart from all other species, they

have their origin in evolutionary history (Gould, 1977). Therefore, it may be found that some basic growth patterns are identical for all organisms. The sigmoid shape of the mathematical curve of growth is one of the features of human growth shared with other species (Bertalanffy, 1960). The fact that the biological pattern of growth can be described so clearly in mathematical terms allows us to predict the course of development and its specific biological events and the nature of the growth processes, which are postulated as being continuous or pulsatile (Ashizawa, Kawabata, 1990; Lampl, Veldhuis, Johnson, 1992; Hermanussen Burmeister, 1993). Due to the mathematical properties of the growth curve, it is possible to make qualitative as well as quantitative comparisons between species.

MATHEMATICAL APPROACHES TO ANALYSIS AND INTERPRETATION OF THE HUMAN GROWTH PATTERN

Well-planned studies of normal children are the basis upon which a knowledge about growth pattern may be built. Knowledge of any changes in growth patterns is valuable evidence about the effectiveness (or otherwise) of measures to improve or maintain the health and nutritional status of the community. Most growth studies are concerned with observing in detail the growth of individuals, or with setting up standards giving normal variations in given parameters of a population of children at different ages. The former aim is usually achieved most effectively by a longitudinal study, the latter by a cross-sectional one. Longitudinal design of the growth study has advantages and disadvantages as well. The main advantages are that they accurately reveal the growth patterns of individuals and enable the study of changes in the speed of growth, the sequence of events in the process of eruption of teeth, the development of the secondary sex characteristics or the timing and sequence of the adolescent growth spurt. The main disadvantages are that they are time-consuming, and the sample is not sufficiently large to be truly representative of the population. For the purpose of growth norms, a cross-sectional study is more appropriate.

The nature of growth makes it suitable for mathematical approaches to its description and interpretation. The final product of growth processes, which are intrinsically linked to an increment in cell mass and an acceleration of cellular proliferation, is a matured biological form. This form usually tends more to resemble other members of the same species than members of another species. Owing to their predictability, growth and form are amenable to the precision of mathematical description. However, it is not an easy task to fit a proper mathematical equation to a series of individual measurements, since the growth process as defined above is biologically complicated, and as such even more complicated for mathematical treatment.

General growth curves are usually illustrated by the distance, velocity and acceleration curves (Medawar, 1945). The simplest form of growth curve may be introduced as a straight line described by the equation of linear regression:

$$y = a + bx$$

where:

- **a** is a constant representing the value of y at which the line crosses the y-axis (when $x = 0$)
- **b** is a constant indicating the slope of the line

If it were possible to describe growth in stature by a straight line, with stature on the y-axis and age on the x-axis, **b** would be the rate of growth and **a** would be the stature of a child at the starting point of examination (for example at birth) *(Figure 1)*.

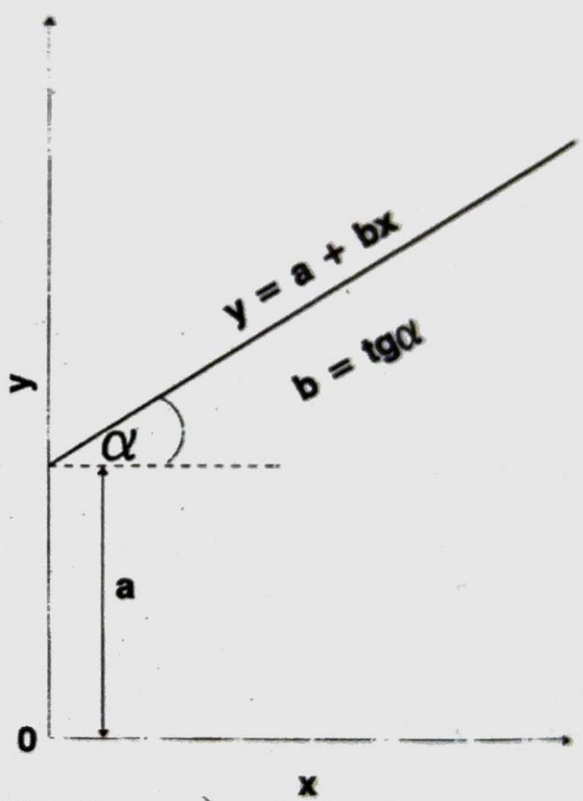


FIGURE 1. Diagrammatic explanation of the parameters of the linear regression equation $y = a + bx$.

However, most growth processes cannot be described by straight lines, as the shape of the growth curve is far more complex. In order to describe these curves mathematically, an equation with more constants should be used. The usual procedure is to plot a distance graph of the measurements for each child, and to then decide what shape of mathematically fitted curve fits them most accurately and describes them most effectively. The curve should be represented by an equation whose constants have some real biological meaning (as b would represent the growth rate if we were to fit individual measurements to the straight line). The fitting process involves calculation of the constants, which would have different numerical values for each child. In order to obtain a curve which would describe the group of children, we could take the mean value of the constants. Again, taking the mean value of **b** would be the average growth rate and the mean value

of a would be the mean value of the length at birth or at the beginning of the examination. A curve calculated in this way is called a mean constant curve. In most cases, this is quite different from a mean curve, which would be obtained by taking the mean value of the measurements at each age and then fitting the curve. In the case of the simplest form of growth curve – a straight line – the mean and mean constants are identical, but this is seldom true of more complex curves.

The distance curve (height versus time) is usually represented by a sigmoid curve characterized by an initial acceleration and then a period of deceleration in growth rate. This regularity of biological growth is more precisely described by the velocity curve, which is described mathematically by the first derivative of the general mathematical equation (rate of growth versus time). In the first approximation, there is a gradual decrease in the tempo of growth until the beginning of the pubertal spurt, when a maximum rate of growth occurs as a single peak (Peak Velocity) (Tanner, Whitehouse, Takaishi, 1966). The second derivative of the general mathematical equation of growth describes acceleration of growth rate versus time, where the point of zero acceleration corresponds to that inflection point in the growth curve at which the rate of growth stops increasing and begins to slow.

In the mathematical fitting procedure, longitudinal data present quite different problems than those of cross-sectional data. In the latter design of the study, each subject contributes a measurement at only one time point. Therefore, the measures and means of measures are unrelated to one another and are stochastically independent. As a result, conventional methods of linear and curvilinear regression may be used when fitting mathematical growth models.

In a longitudinal study a series of measurements are made on the same subject at different times, at appropriate intervals of time. The measurements are logically associated and stochastically correlated. In the fitting of individual growth models, much of the correlation between measurements may be suppressed and variation due to systematic error removed. The correlation that remains in the residual variation is due to short-term fluctuations in growth appearing as a response to environmental influences.

Generally, there are two approaches in the procedure of condensation and smoothing the series of individual measurements: non-structural and structural. In the non-structural approach the data are smoothed locally to supress measurement errors and short-term variation, such as, for example, polynomials or cubic spline functions used for smoothing the velocity curve and to estimate the pre-adolescent minimum and adolescent maximum (Largo et al., 1978; Hauspie, 1988). In the structural approach it is assumed that growth pattern may be described by a math-

TABLE 1. Mathematical models of growth patterns.

Author	Model	Equation	Parameters	Age interval
		non-structural:		
Hauspie 1988	polynomial:	$y = b_0 + b_1 t + b_2 t^2 + \ldots + b_n t^n,$		0 adult
		structural:		
Bogin 1993	Count	$y = a + bt + c\log(t)$	abc	0–7 years
Hauspie 1988	Jenns-Bayley	$y = a + bt - e^{c+dt}$	abcd	0–8 years
Marubini et al. 1971	Logistic	$y = P + \frac{K}{1+e^{a-bt}}$	PKab	10–adult
Deming 1957	Gompertz	$y = P + Ke^{-e^{a-bt}}$	PKab	10–adult
Bock et al. 1973	Double logistic	$y = \frac{a_1}{1+e^{-b_1(t-c_1)}} + \frac{f-a_1}{1+e^{-b_2(t-c_2)}}$	$a_1b_1c_1b_2c_2f$	2–adult
Bock, Thissen 1980	Triple logistic	$y = a_1\left[\frac{1-p}{1+e^{-b_1(t-c_1)}}\right] + \left[\frac{p}{1+e^{-b_2(t-c_2)}}\right] + \frac{a_2}{1+e^{-b_3(t-c_3)}}$	$a_1b_1c_1a_2b_2c_2b_3c_3p$	1–adult
Preece, Baines 1978	Preece–Baines	$y = h_1 - \frac{2(h_1-h_\theta)}{e^{s_0(t-\theta)} + e^{s_1(t-\theta)}}$	$h_1h_\theta s_\theta s_1\theta$	2– adult
Jolicoeur et al. 1988	JPPS	$y = A\left\{1 - \frac{1}{1+(t/D_1)^{c_1} + (t/D_2)^{c_2} + (t/D_3)^{c3}}\right\}$	$AD_1C_1D_2C_2D_3C_3$	0– adult

ematical model, usually introduced as a family of functions that relate observable data records in terms of unobservable parameters. The parameters are then estimated by those values that best fit the function to the empirical data record. If the model is found to fit well, and the number of parameters is small relative to number of measurements, the statistical estimation of the parameters serves for smoothing and summarizing the record, indicating at the same time the main trends in the individual growth pattern.

There are two hundred mathematical equations that auxologists have at their disposal when fitting mathematical models to the individual data. Some of the most often used are presented in *Table 1* (Deming, 1957; Marubini et al., 1971; Bock et al., 1973; Preece, Baines, 1978; Helwin et al., 1979; Bock, Thissen, 1980; Szczotka, 1981; Hauspie, 1988; Jolicoeur et al., 1988; Jolicoeur et al., 1992).

In the present study, the Jolicoeur, Pontier, Abidi model (JPA2) has been chosen for fitting individual growth records (Jolicoeur et al., 1992). The JPA2 model is based on the assumption, previously pointed out by Robertson in 1908 (cited after Bock, du Toit, Thissen, 1994), that human growth occurs in a number of additive, more or less independent phases during a course of development which constitutes a continuous process of growth. The timing and intensity of each phase is assumed to be genetically programmed in the individual, but may vary in expression according to environmental conditions. In the JPA2 procedures three major phases of human growth have been distinguished: early and middle childhood and adolescence. The mathematical equation is a 7 parametric asymptotic formula *(Figure 2)*.

In order to provide numerical estimation of the growth pattern, two methodological approaches were used:

1. Structural analysis of population dispersion of the parameters of the model which allows description of the main tendencies in the growth in same-sex case.

Model JPA2 is a seven-parameter asymptotic growth curve:

$$y = a\left\{1 - \frac{1}{1+\left[b_1(x+e)\right]^{c_1}+\left[b_2(x+e)\right]^{c_2}+\left[b_3(x+e)\right]^{c_3}}\right\}$$

where:
y – size (cm), x – age (years)
a, b_1, b_2, b_3, c_1, c_2, c_3 – are 7 parameters:
a is adult size,
b_1, b_2, b_3 are three positive time-scale factors,
c_1, c_2, c_3 are positive, dimensionless exponents, reflecting the shape of the initial, central and final section of the growth curve.

(Jolicoeur, Pontier, Abidi 1992)

FIGURE 2. Mathematical formula for the JPA2 procedure for fitting individual growth curve.

2. Nonstructural analysis of residuals from the model in each individual case. Residuals were smoothed by Fourier procedure. The combined structural and non-structural approaches allow characterization of major trends in individual growth as well as point out the periodicity of growth. The procedures for fitting growth curves were implemented in the AUXAL programme (Bock, Thissen, du Toit, 1994).

The analysis showed that the fit of this model over the range from 5 to 14 years was good. The mean square error averaged 0.58 cm in boys and 0.52 cm in girls. Despite the good fit of the JPA2 model to measurements of height in the Poznań Growth Study, the average residuals in each yearly interval were not everywhere zero, showing that the model had some bias, and both the size of the root mean squares and the sign of the patterns in the residuals indicate the presence of some remaining systematic variation. There is thus some evidence for waves of slightly accelerated or decelerated growth, often lasting one or two years (minigrowth spurts), during middle childhood. These deviations from the main trajectory of growth – the periodicity of growth – reflect the individual strategy of development in various environments.

The tendency of growth was then assessed, according to the multi-level modelling of growth (Cieslik, 1979). The concept of multi-level growth is based on the equation of linear regression. The tendency can be assessed as positive (P) when the value of the statistic F is significantly different from zero and the parameter b has a positive sign. This describes a situation in which the individual undergoes transition to higher developmental levels in the subsequent stages of ontogeny. That is why one should consider the progression of development in relation to the previous stage as the most essential characteristic of this model. The tendency can be assessed as negative (N) when the value of the statistic F is significantly different from zero and the parameter b has a negative sign. This describes a situation that is opposite to the one described above. An individual forms its phenotype by going to lower and lower levels of development in the subsequent stages of ontogeny, within the range of his own reaction norm. When the value of the statistic F is not significantly different from zero, it means that developmental level tends to change in a multi-directional way (fluctuative). If intra-individual variation in the rate of growth in height is less than 0.22, the tendency is called stable (S). A stable tendency of growth characterizes the situation of a growing child, in which the genetic potential of an individual is used with the same intensity in each stage of his ontogeny and does not take advantage of the higher or lower developmental levels in relation to the previous stage. It may be said that the individual takes the same level of development within his own reaction norm. A multi-level (M) tendency of growth has been found when the value of statistic F is not significantly different from zero, and intra-individual variation in the rate of growth is higher than 0.22. This means that an individual forms his phenotype by changing developmental levels in a fluctuating way. It is

then possible to observe each of the previously introduced tendencies: stable, positive or negative.

CAUSAL FACTORS CONTROLLING HUMAN GROWTH: A THEORETICAL MODEL

A human being, like other animals, begins its life as a single cell – the fertilized ovum. Likewise, it passes through all the stages of its development in predetermined sequence in some environments or series of environments. It undergoes various processes of growth, differentiation and development and, as a result of them, changes into embryo, fetus, child and adult. The transformation of an organism from one developmental stage to another is the result of an individual strategy of development. A definite strategy of individual development is a unique interaction between its genes and the environmental milieu the developing organism meets with during its ontogeny. The most likely mechanism of the individual strategy of development seems to be multiple, and depends on the susceptibility of the genotype to various environmental factors (genetic flexibility) and the epigenetic regulation of growth. The result of genotype by environment interaction may be phenotypically recognized as an individual pattern of growth. Thus, it is evident that every individual has his/her own history of life and mode of growth and development.

Although growth and development may occur simultaneously, they are distinct biological processes. Growth is intrinsically linked with increment in cell mass and acceleration of cellular proliferation. Therefore, it may be defined as a quantitative increase in size or mass. Development is linked with differentiation (morphogenesis) and may be defined as progressive changes (either quantitative or qualitative) that lead from an undifferentiated state to a state highly organized and specialized for its function.

Causal factors that control and regulate the sequential ordering and timing of specific events that take place during the life cycle may be recognized as intrinsic and extrinsic factors. Four major categories have been pointed out: genetic, epigenetic, maternal genetic and environmental factors. Regarding the maternal genetic factor, it is emphasized that mammalian (and also human) development is under the control of two separate genomes: that of progeny and the maternal one. All categories of causal factors of development mentioned above are related either by interaction or by determination. They constitute elements of the model of human growth and development *(Figure3)*.

The proposed model is a compilation of two traditional approaches: that of quantitative genetics (genes and environment) and that of developmental biology (epigenetic factor). It seems that this model is sufficient for a better understanding of the relative roles of the intrinsic genetic and epigenetic control and regulation of growth and development.

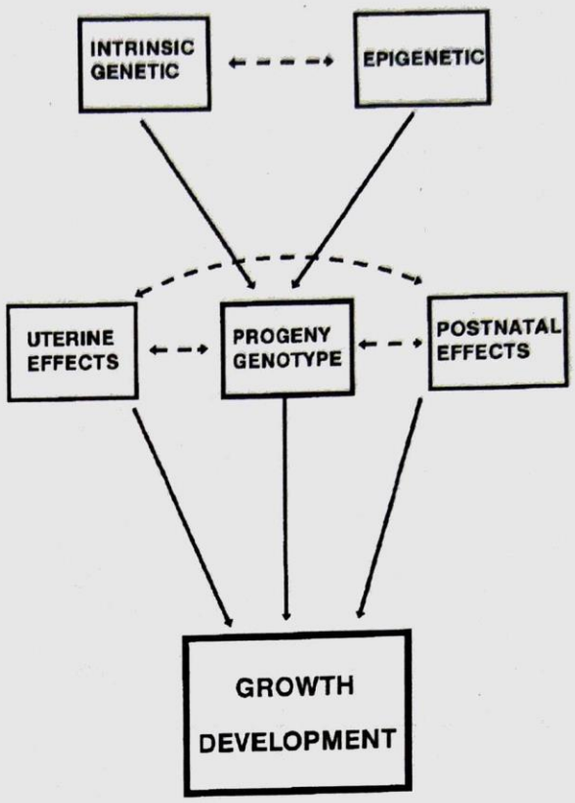


FIGURE 3. A model of the factors determining the human growth and development.

Genetic (endogenous) control of development is based on the fact that the progeny genome provides the blueprint for development. This does not mean that genes directly influence the shape of a particular morphological structure. They may, rather, act locally (intrinsically) by coding for structural or regulatory elements or regulating their expression and activities. They may also act epigenetically to influence the activities of cells at a distance (hormone regulation).

There are non-heritable conditions which may have a significant impact on development. Environmental (exogenous) factors may blur the contribution of genetic and heritable epigenetic factors to development within the reaction norm of genotype.

The concept of the reaction norm is a basic one for better understanding of the genetics of growth and the description of non-heritable variation of morphological traits (Stearns, 1989). Therefore, the notion of the reaction norm of genotype may be defined, according to the classical definition introduced by Voltereck in 1909, as a set of phenotypes produced by a genotype across environment (Voltereck, 1909). The meaning of the reaction norm is often presented by a graph (where it is shown as a line or curve) that plots phenotypes against environmental factors (Suzuki et al., 1986) *(Figure 4)*.

As is shown in *Figure 4*, a hypothetical genotype denoted as G1, which interplays (interacts) with series of environments, may produce a definite profile of phenotypes. In this way, mapping of the genotype on to the phenotype as a function of the environment is possible, and

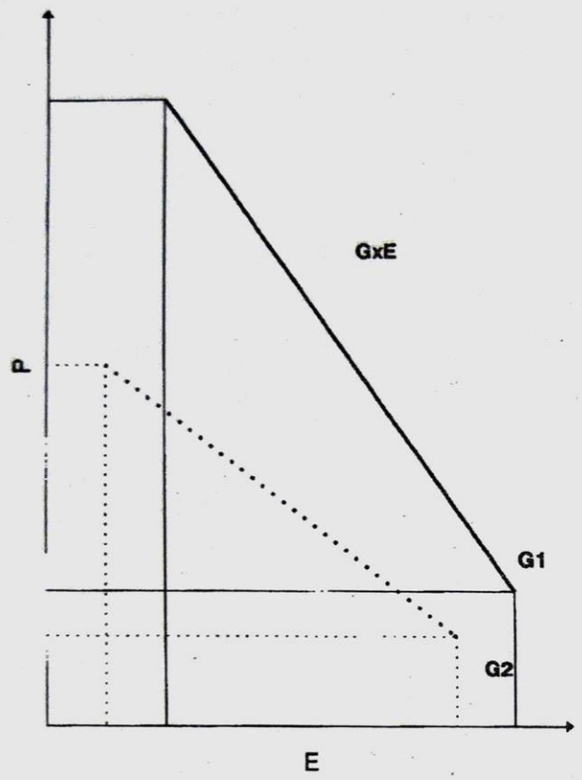


FIGURE 4. A concept of the reaction norm.

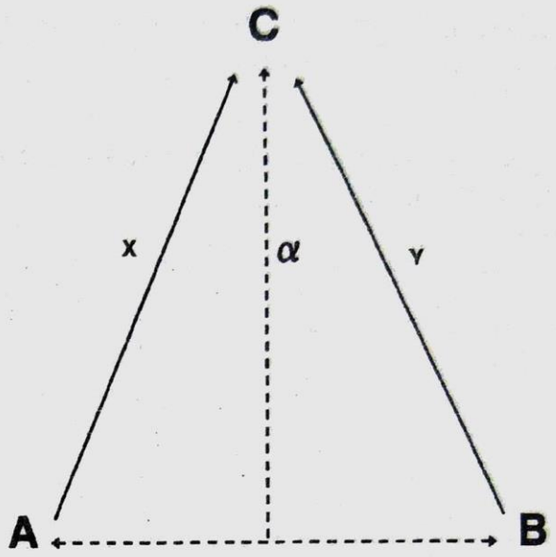


FIGURE 5. A meaning of the interaction.

this is another meaning of the reaction norm (Stearns, 1992). The additive effects of many genes and the reaction norm are two major sources of continuous variation in the metric traits observed in a population.

Genetic and environmental factors are related by the interaction. In the modes introduced, interaction is understood in the classical sense introduced by Fisher (Fisher, 1918 after Falconer 1974). *(Figure 5.)*

As it is described graphically, factor C (a definite metric trait) depends on factor A (genes) and B (environmental conditions) and on the interaction between A and B, which is denoted on this graph as alpha. Genotype by environment interactions alter during ontogeny. The nature of changes as well as their intensity result in a definite strategy of the development of an organism, which may be phenotypically recognized as its pattern of growth.

It is well know that all normal, healthy and well-nourished children follow the same basic pattern of growth from birth to maturity – a standardized way of growth-defined by the potential of growth given by an individual's genetic make-up. This will only be possible if the environment is compiled; if not, the pace of growth is far from standard. A non-standardized process of growth may elucidate the adaptible responses of organism to environmental stresses and, at the same time, the individual strategy of development.

Since numerical estimation of growth is most often based on quantitatively varying morphological traits, body height dimension is taken as representative for metric traits in the proposed model. Body height – stature, an easily measured trait, is in fact a complex morphological structure composed of a number of separate component parts such as the head, neck, trunk and lower extremities. Each part develops at its own rate and under the control of different causal factors. However, the integration of of ontogenies of these separate parts constitute the final form – the body height (Tanner 1963, Bielicki, Welon 1978).

INTRINSIC (GENETIC) AND EXTRINSIC (ENVIRONMENTAL) COMPONENTS OF VARIATION IN HUMAN GROWTH PATTERNS

Analytic interpretation of the theoretical model tends to establish the part played by heredity and environment in determining quantitatively varying characters. Generally, methods that allow partitioning of phenotypic variability into that arising from intrinsic genetic and extrinsic factors have a multifactorial approach. They are based on the resemblance between relatives and twins (Susanne, 1975, 1977, 1994). These procedures definitively preclude any identification or analysis of the numerous pathways and genes involved.

Studies of familial resemblance for stature are compatible with the assumption that the genetic source of normal variability within population depends on allelic diversity of a number of loci, where each segregating allele may have a small overall effect. Further, there are indications that different genetic factors influence growth in height at different ages (Verschuer, 1934). According to the Iwamoto–Knussman's model, an additional complex of genes called "adolescence complex" has been proposed as contributing to the course of development (Knussmann, 1968, 1969, 1978). There are also indications that effects of the actions of individual genes are obscured by the actions of non-heritable environmental factors (see theoretical model of factors determining human growth and development in *Figure 3*). Numerical estimates that allow partitioning of phenotypic variability into that arising from

intrinsic genetic and extrinsic (environmental) factors and of their relative importance to the growth pattern in stature are based on the path analysis. The procedure developed by Rice et al. (1978), called the tau transmission model for sources of resemblance between parent and offspring and between two siblings, was used. A detailed explanation of all variables taken into account in this multifactorial procedure is given in *Figure 6.*

In the case of a quantitative character, the study of its variation is obviously our first concern (Falconer 1989). The phenotypic variance V_P can be divided into:

$$V_P = V_G + V_E + 2cov_{GE}$$

where: V_P – total phenotypic variance,
V_G – genotypic variance,
V_E – environmental variance,
$2cov_{GE}$ – variance caused by the interaction between genotype and environment; this component is often presumed to be equal to zero, thus we obtain the simplified relation: $V_P = V_G + V_E$.

Considering the tau transmission model, a standardized phenotype (P) can be expresses as an additive function of the transmissible component (T) and the nontransmissible component (R) in the form:

$$P = tT + \sqrt{1-t^2}\,R$$

where: t^2 is the proportion of the total phenotypic variance that is transmissible.

Empirical materials used in this study consist of one cohort of children born in 1980, participants in the Poznań Growth Study which has been carried out since 1985 till now. The sample consists of 554 subjects: 284 boys and 270 girls aged 5 to 14 years old. Somatic measurements (i.e. standing body height) have been taken in half-year intervals using a GPM anthropometer with acceptable accuracy. In our sample, intra-TEM for body height was 0.42 cm and inter-TEM was 0.62 cm. The resulting intra- and inter-TEM do not differ from those figures cited in the literature (Malina, Hamill, Lameshow 1973, Cameron 1984). Interested correspondents supplied measurements of their own relatives: parents and sibs. The data were used for study of family resemblance.

Environmental factors were recognized as the socioeconomic level of family and the number of children in family. It is well-known from numerous works that this complex environmental factor (SES) plays an important role in the regulation of growth. The SES of the family was assessed according to following social variants: parents' education, index of family income per person and index of density, measured as number of persons per one room. According to the distribution of the social and economic conditions of the family, three levels of life conditions were distinguished: high (A), medium (B) and low (C).

In the first step of the empirical analysis, genetic sources and causes of the phenotypic variation will be discussed, based on the resemblance between relatives. Familial correlations and sample size are presented in *Table 2.*

As can be seen from the data presented in the *Table 2,* similarities between all examined relatives are statistically

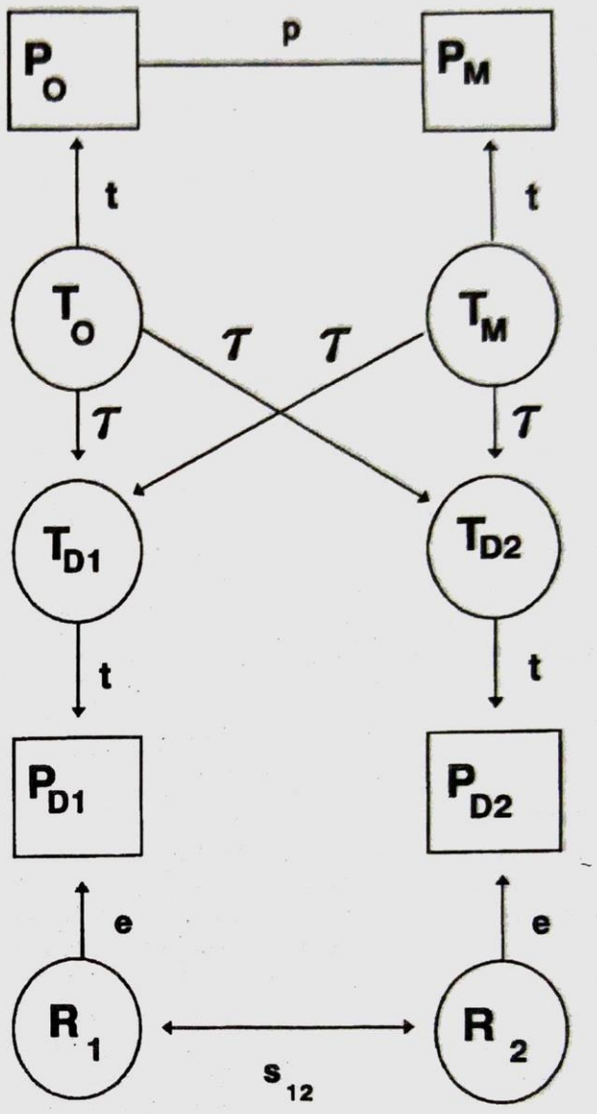


FIGURE 6. The tau transmission model for sources of resemblances between parent and offspring and between two siblings. Observed variables are shown in squares, where P denotes phenotype: O – father, M – mother, D_1, D_2 – offspring. Latent variables are shown in circles: T denotes genetic and cultural factors that are transmissible from parent to offspring. The parameters of the model are:

p – phenotypic assortative mating between spouses,
t – the transmissibility of the phenotype,
tau – the transmission of the phenotype (tau= 0.5 is polygenic transmission),
s – the correlation of nontransmissible components among full siblings.

TABLE 2. Observed familial correlations and sample size for stature from Poznań population.

Relatives	number of pairs	r	Se
Spouses	342	0.249*	0.16
Father – son	204	0.348*	0.19
Father – daughter	184	0.370*	0.18
Mother – son	201	0.314*	0.15
Mother – daughter	179	0.422*	0.11
Brother – brother	149	0.448*	0.09
Brother – sister	185	0.429*	0.07
Sister – sister	135	0.498*	0.09

significant. Correlation coefficients between the same-sex pairs are slightly higher e.g. (r)father-son>(r)mother-son or (r)mother-daughter>(r)mother-son. However, the suggestion of sex specific similarities between relatives does not hold true. Differences between the same-sex pairs and different-sex pairs are not statistically significant.

The tau transmission model enables us to test further hypotheses about familial similarities.

In *Table 3* are shown likelihood ratio chi-square values for the general model and specific hypotheses involving linear model constraints. In general model (first column) four parameters were tested: t^2, s_{11}, s_{12}, s_{22}, under the assumption that tau = 0.5 (polygenic transmission) and similarities between spouses p = +0.249. The maximum likelihood ratio chi-square value for the stature is 1.48 (df = 3), which means that the general model provides a good fit to the observed familial correlations for stature. The hypothesis of no significant parent assortative mating (the second column, p = 0) can be rejected, since the chi-square value 8.24 is significantly different from zero at $\alpha = 0.05$. This indicates quite clearly that spouses were attracted by their stature. In the third column of *Table 3*, the likelihood ratio chi-square value for the hypothesis of no vertical transmission is presented ($t^2 = 0$). This hypothesis is also rejected (chi-square value is 48.20). This result gives evidence for direct intergenerational transmission of stature. The next three columns of the *Table 3* show likelihood ratio chi-square values for the hypotheses of no residual sibling resemblance. They cannot be rejected, which means that there is no resemblance between siblings due to common environmental conditions of their life. The last hypothesis tested is of no familial resemblance (the last column of the *Table 3*). It can be seen that this hypothesis should also be rejected. There is resemblance in stature between relatives and, as was noted before, intergenerational transmission of stature. The estimated transmissibility value t for stature in our material is t = 0.789, thus $t^2 = 0.624$ with SE = 0.059. This figure is in accordance with those known from the literature (Neale, Cardon, 1992).

Growth is a more or less continuous phenomenon from conception to maturity, and the various factors acting on growth can change in the different stages of development. The relative importance of two major determinants of growth: genotype and environment can vary during the development of an individual. Considering this fact, growth curves in height of examined children (distance and velocity curves) against five categories of mid-parent value of stature were plotted. The mid-parent value of stature was distributed into five groups, with a class interval of a half SD each. The result is shown in *Figure 7.*

The growth status of children whose parents are taller is higher within the whole period examined (5–15 yrs). It can be seen from the plot that a marked gradient of the course of the developmental paths exists, which is especially clear as far as the distance curve is concerned. This fact suggests strong genetic control of the pattern of growth in body height, as well as high heritability of height.

It is also perfectly clear that body height is particularly prone to nutritional and environmental influences. Environmental control of the growth pattern tends to be verifed within the range of socioeconomic conditions. Distance and velocity curves of growth plotted against three levels of family SES are presented in *Figure 8.*

When analysing the graph, there is clear evidence for the influence of socioeconomic factors on the pattern of growth in height, in such a way that there are social gradients of the growing as well as the attained (adult) height predicted from the mathematical formula. The conclusion is: the better the living conditions, the taller the children. The social gradients in body height are more regular and marked in boys than in girls. This remark support previous statements about the higher ecosensitivity of boys.

When the adolescent growth spurt is taken into account, similar social gradients can be observed *(Table 4).* However, the observed social differences, again more regular and marked in boys than in girls, are not statistically significant.

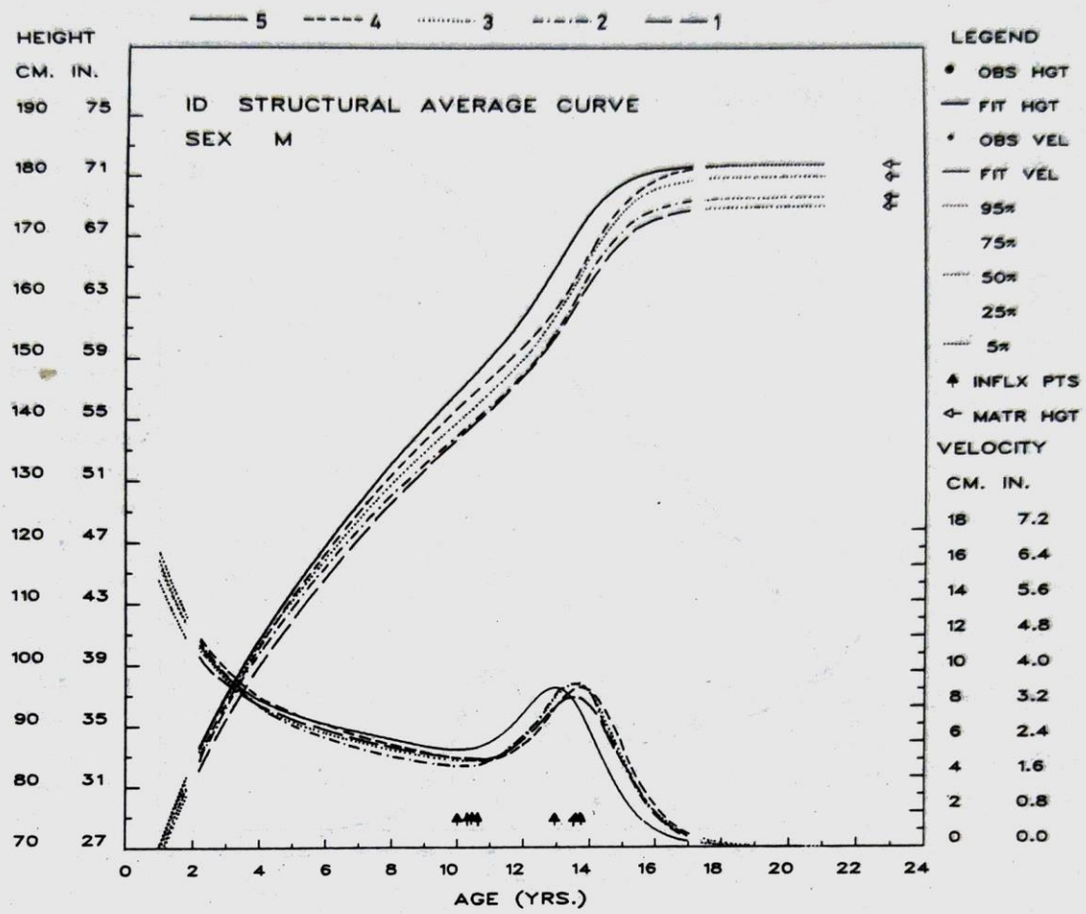
TABLE 3. Parameter estimates and likelihood ratio chi-square values for the stature under the TAU transmission model.

	General model df = 3	p = 0 df = 1	$t^2 = 0$ df = 1	$s_{11} = 0$ df = 1	$s_{12} = 0$ df = 1	$s_{22} = 0$ df = 1	t=p=s=0 df = 3
Stature	1.48	8.24*	48.20*	2.60	1.90	1.49	58.40*

* Correlations significantly different from zero at $\alpha = 0.05$

TABLE 4. Descriptive statistics of adolescent growth spurt in height.

Girls	SES	$\bar{x}$	S	S_e
PHV (years)	A	11.60	0.98	0.12
	B	11.32	0.98	0.12
	C	11.56	0.85	0.09
PHV vel. (cm/yr)	A	7.61	1.44	0.18
	B	7.85	1.97	0.22
	C	7.66	1.07	0.12
TO age (years)	A	8.66	1.04	0.13
	B	8.44	1.08	0.12
	C	8.60	0.90	0.10
TO vel. (cm/yr)	A	5.22	0.80	0.10
	B	5.30	0.82	0.09
	C	5.19	0.70	0.08
Predicted adult stature (cm)	A	167.19		
	B	166.60		
	C	167.02		
Boys	**SES**	$\bar{x}$	**S**	S_e
PHV (years)	A	13.61	1.23	0.14
	B	13.80	1.29	0.15
	C	13.87	1.03	0.13
PHV vel. (cm/yr)	A	9.59	2.40	0.28
	B	9.45	2.53	0.29
	C	9.36	1.83	0.23
TO age (years)	A	10.39	1.19	0.14
	B	10.55	1.13	0.13
	C	10.37	0.93	0.12
TO vel. (cm/yr)	A	4.86	0.66	0.08
	B	4.77	0.66	0.08
	C	4.83	0.55	0.07
Predicted adult stature (cm)	A	180.59		
	B	179.60		
	C	178.28		



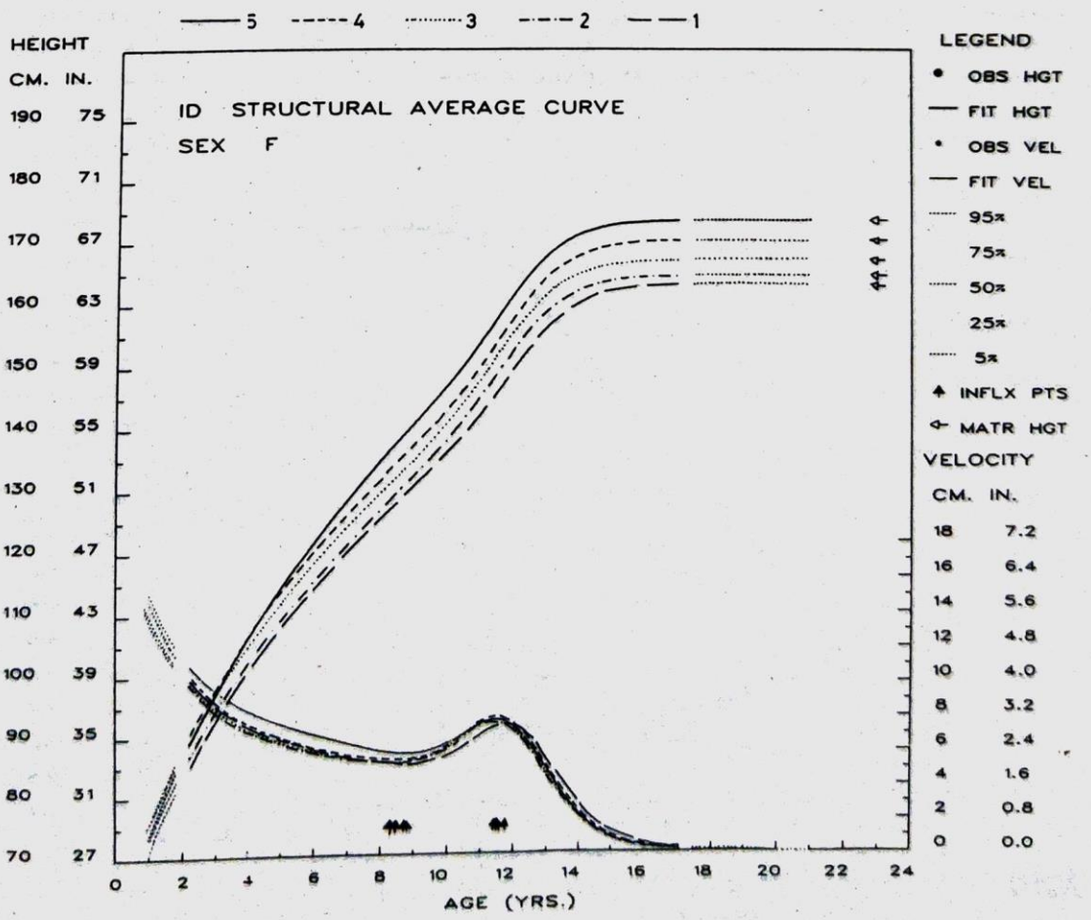
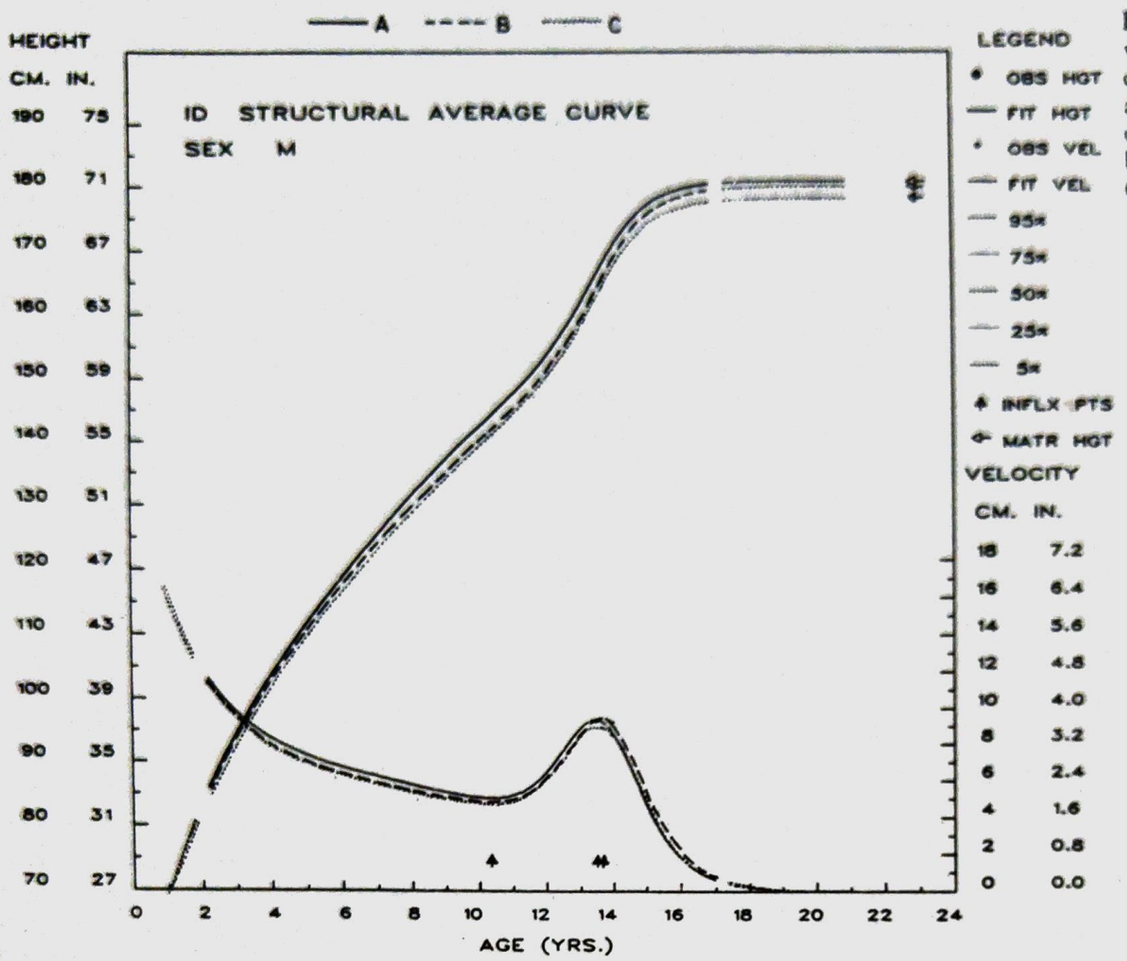


FIGURE 7. Distance and velocity curves of the children's body height according to the category of their mid-parent value: M – boys, F – girls (see text for explanation).



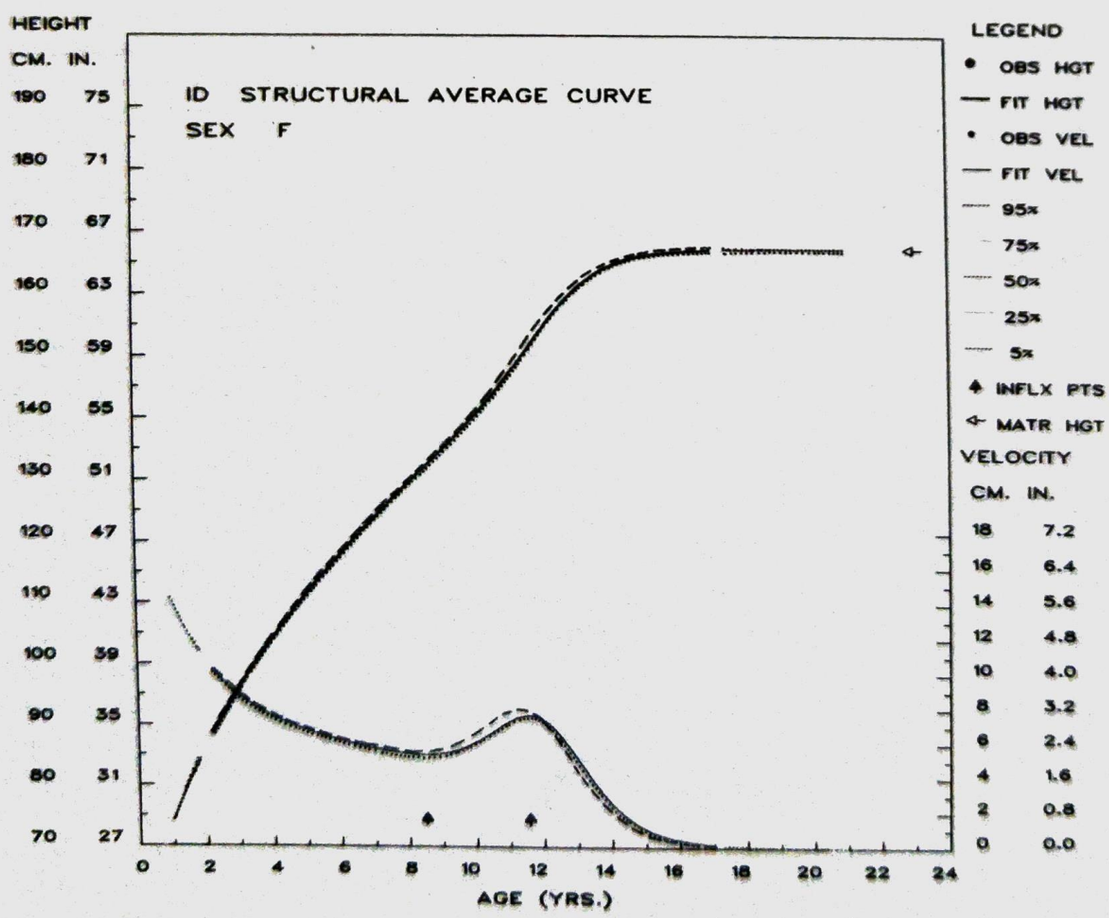
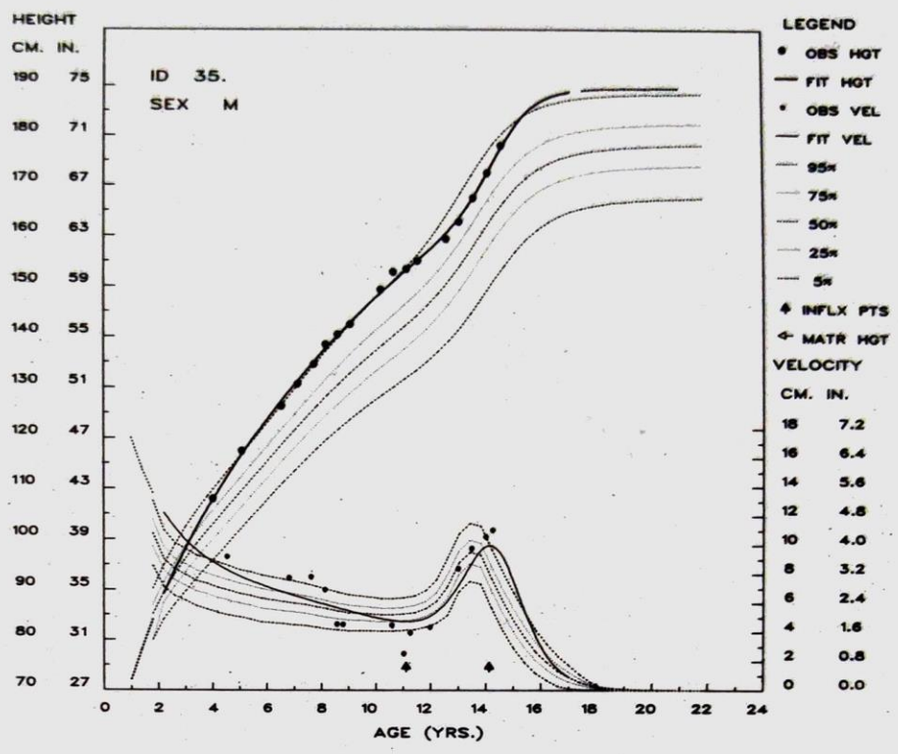


FIGURE 8. Distance and velocity curves of the children's body height according to the SES category of family: M – boys, F – girls (see text for explanation).

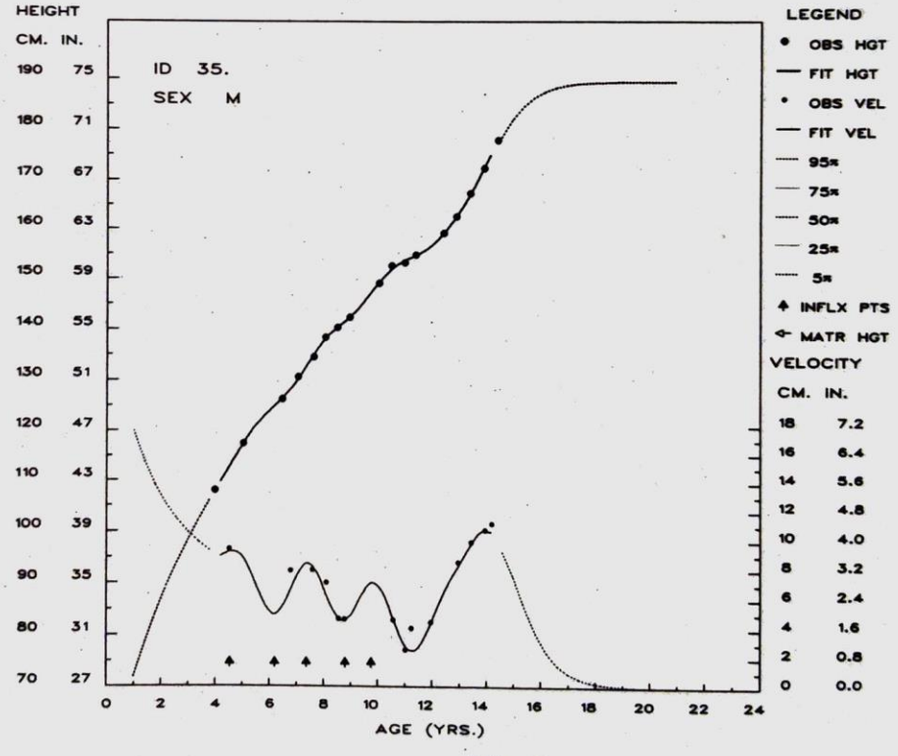
FIGURE 9. Boy 35: a – Structural distance and velocity curves, b – Non-structural distance and velocity curves: Fourier estimation of residuals, c – Tendency of growth in body height: model of linear regression.

TO	AGE (YRS)	11.10
TO	VEL (CM/YR)	4.78
PHV	AGE (YRS)	14.12
PHV	VEL (CM/YR)	10.02

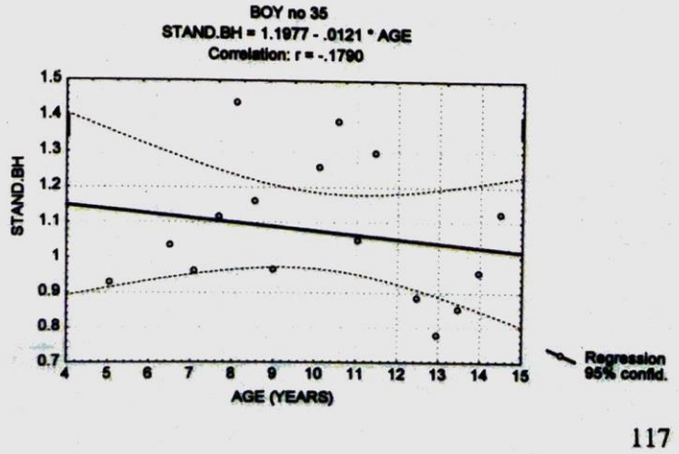
a)

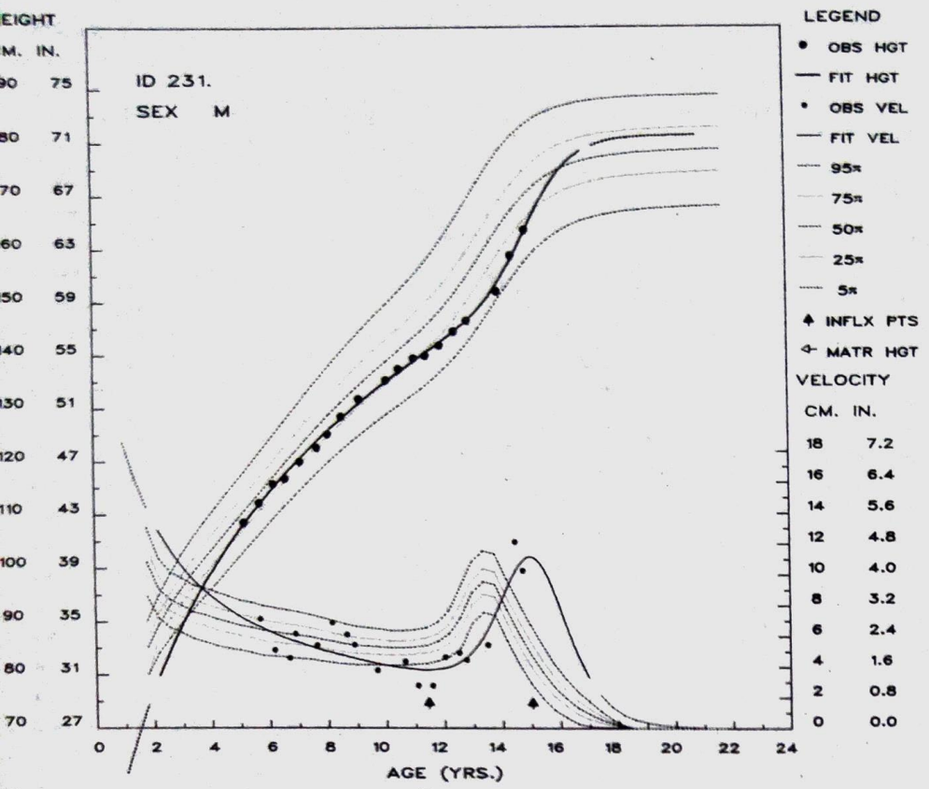


b)

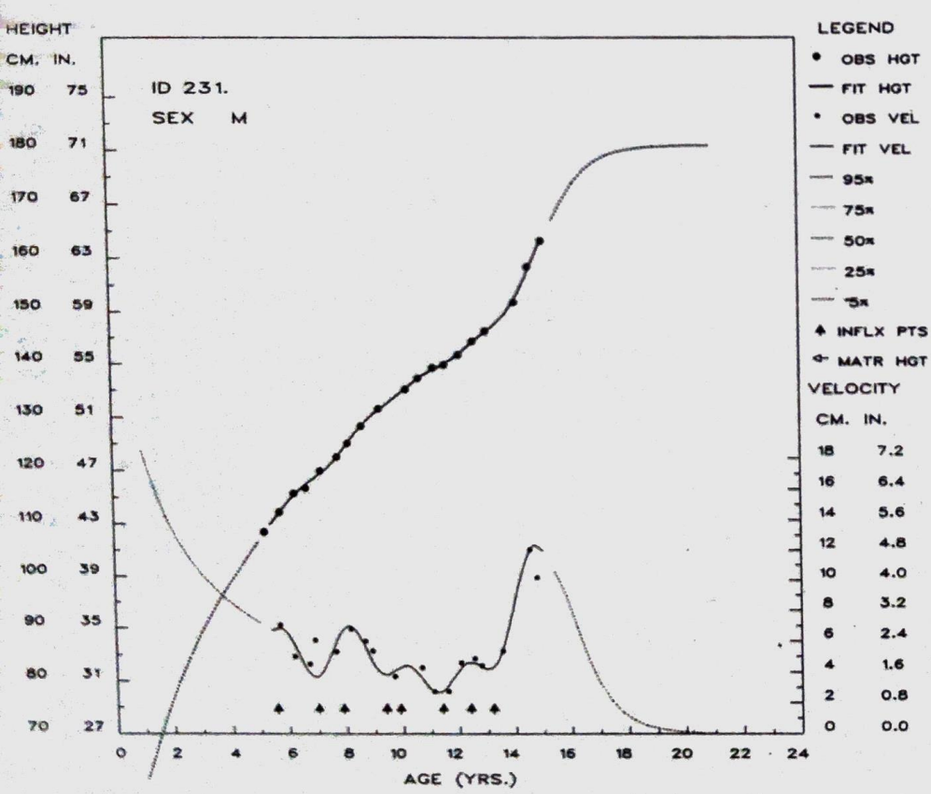


c)





a)



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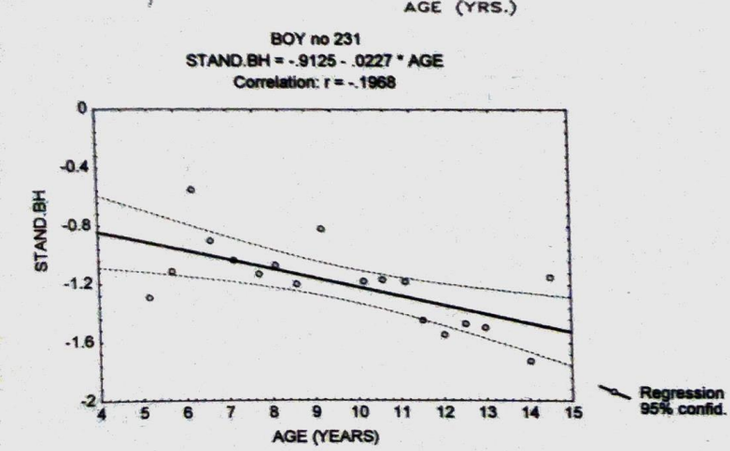


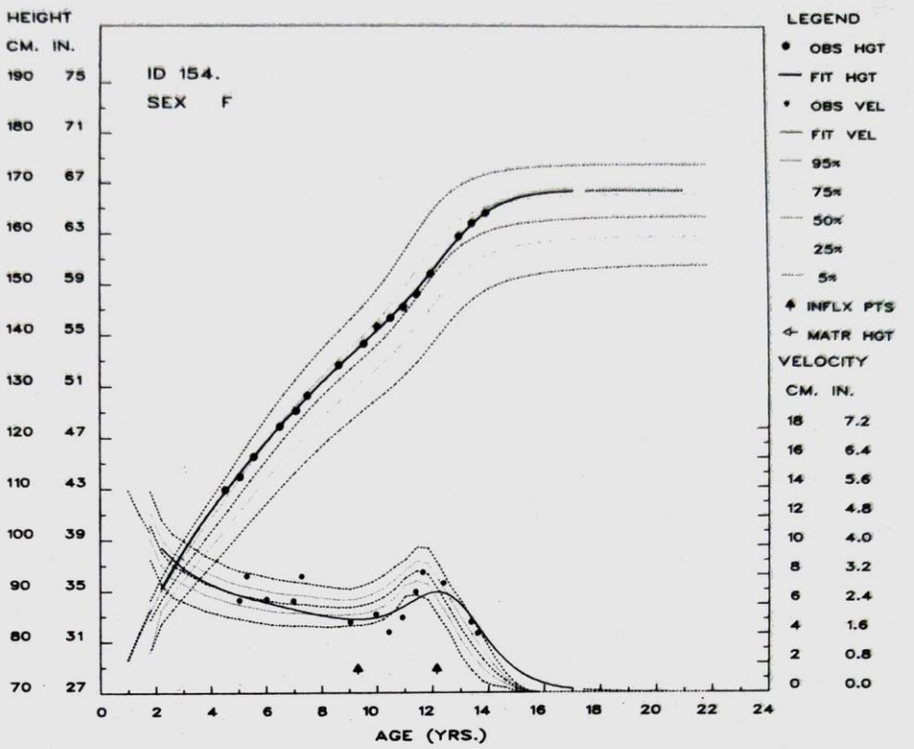
c)

FIGURE 10. Boy 231: a – Structural distance and velocity curves, b – Non-structural distance and velocity curves:Fourier estimation of residuals, c – Tendency of growth in body height: model of linear regression.

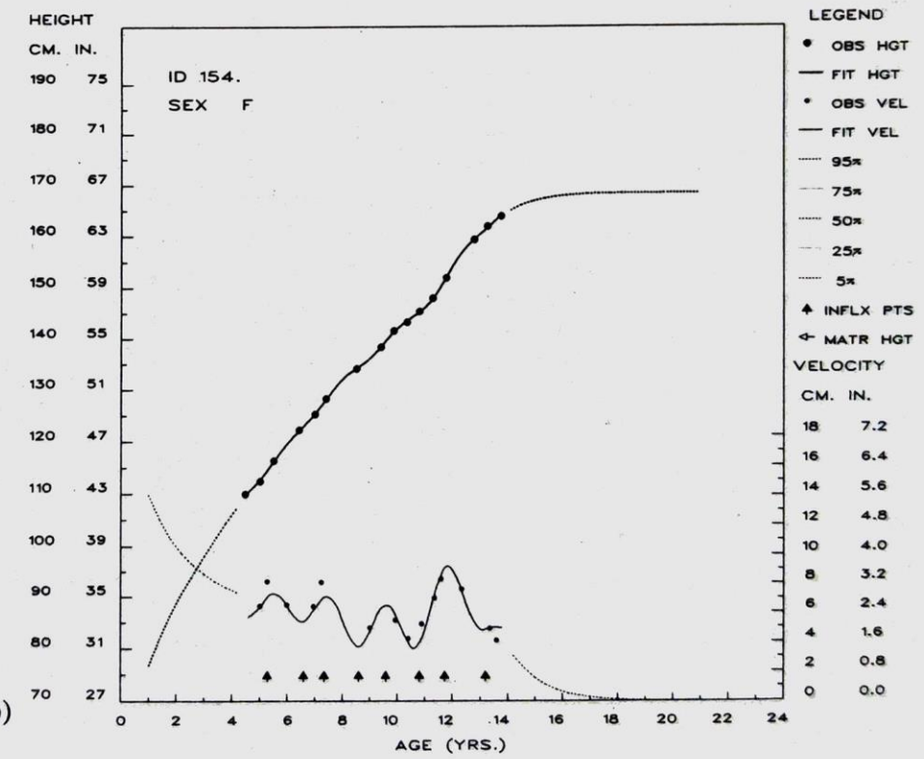
FIGURE 11. Girl 154: a – Structural distance and velocity curves, b – Non-structural distance and velocity curves:Fourier estimation of residuals, c – Tendency of growth in body height: model of linear regression.

TO	AGE (YRS)	9.29
TO	VEL (CM/YR)	5.04
PHV	AGE (YRS)	12.14
PHV	VEL (CM/YR)	6.90

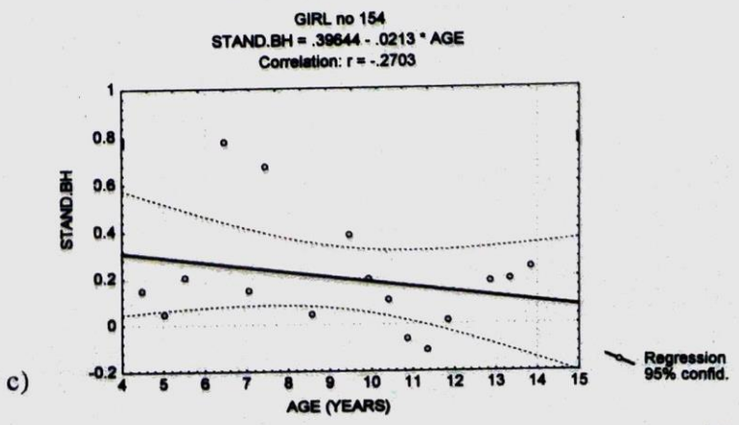
a)

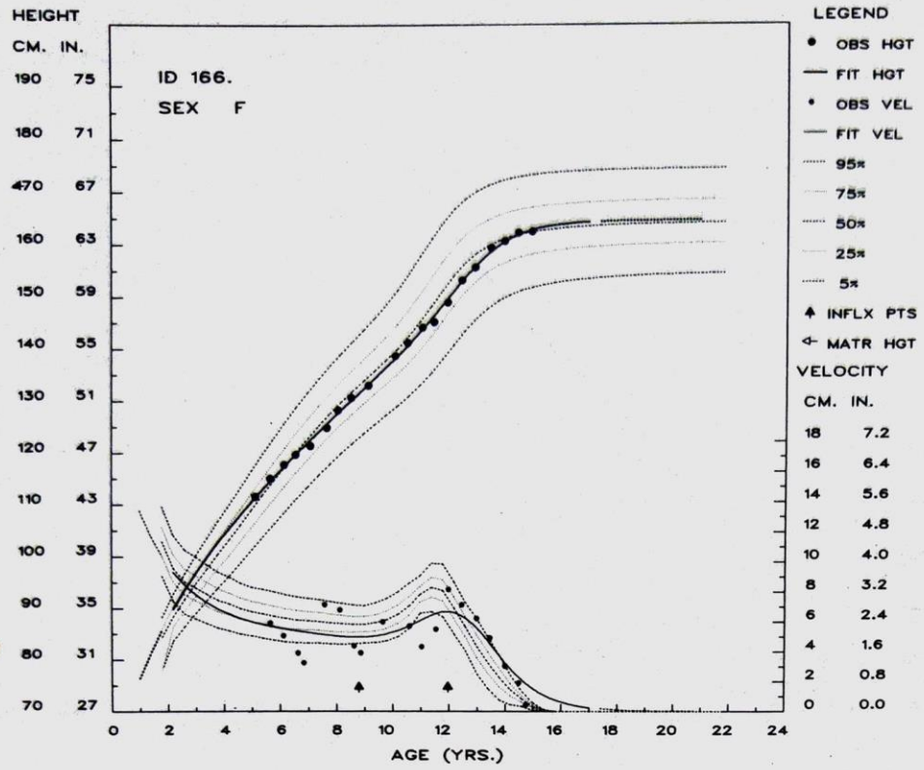


b)

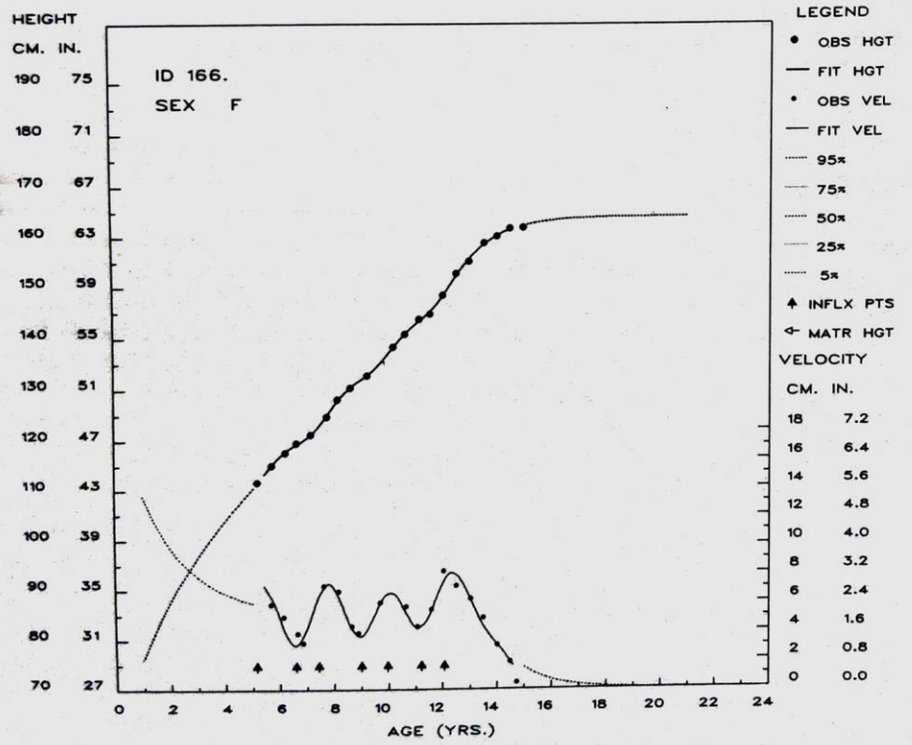


c)

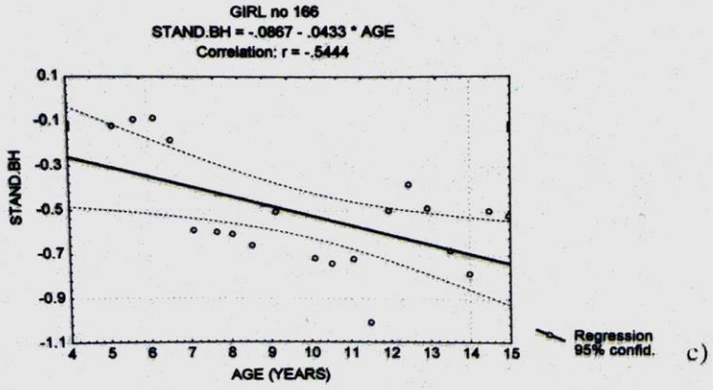




a)



b)



c)

FIGURE 12. Girl 166: a – Structural distance and velocity curves, b – Non-structural distance and velocity curves: Fourier estimation of residuals, c – Tendency of growth in body height: model of linear regression.

The theoretical concept of an individual strategy of development will be shown by empirical examples taken at random from the whole sample. Distance and velocity curves have been plotted against the age with the use of two methodological approaches: structural and non-structural. The structural procedure (figure a) enables estimation of the status and rate of growth against the course of growth norms. This procedure, used in combination with Fourier analysis of residuals, enables us to emphasize the individual pattern of periodicity of growth (figure b). Finally, the tendency of growth assessed after the equation of linear regression (figure c) makes it possible to characterize the periodicity of growth as being rather monotonically directional or fluctuating.

Figure 9a shows observed and fitted height and empirical velocities of a tall boy (**boy 35** from high SES family). The large dots are the measured heights during the time period from 5 to 14 years old and the small dots are empirical annual velocities. The plot includes the computerized version of the course-of-growth norms. Small arrows at the bottom of the plot mark the turning points of the velocity curve that describe two major events of growth in puberty: take off (TO) of pubertal spurt and peak height velocity (PHV).

It is clear that this tall boy follows a perfectly normal pace of growth throughout middle childhood (50 percentile). He departed from the velocity norms only when his adolescent growth spurt was delayed for about 8 months (25 percentile) (mean age of TO in the sample is 10.44 yrs). The boy is 153.45 cm tall and his maturation has been completed by 81%. Velocity of take off is 4.78 cm/y. Peak height velocity of this boy took place at the age of 13.68 yrs, and is late by half a year in comparison with the average age of the population. The pubertal growth spurt in body height has been sufficiently prolonged. At this time the described boy moved his percentile position up enough to reach 180 cm of his adult stature, which situated him on the position of the 95th percentile of the Polish adults. There is a clear indication of periodicity in growth *(Figure 9b)*. However, there are only two minimal and two maximal peaks of velocity. Timing and intensity of the local minigrowth spurts reflect the differential influence of environment on growth. The tendency of growth described in terms of the altering developmental levels of the boy is assessed as nondirectional – stable (S) tendency *(Figure 9c)*. This means that the growth pattern goes on at the same developmental level.

Boy 231 *(Figure 10a)* lives in a family with low SES. He is short, and the tempo in the prepubertal period is systematically restrained. The pubertal growth spurt begins later than average, at the age of 11.45 yrs. Velocity at the take off (minimum prepubertal peak MPP) is 3.80 cm/y what means that is 1 cm, also lower than average. PHV is also delayed, in comparison with the average age in the group, by two years (15.03 yrs). However, the intensity is high (11.17 cm/y). Pubertal spurt is prolonged for about half a year. The total adolescent growth is figured as 41.33 cm, and the prediction for the attained height is 180.86 cm. The boy will be at the position of the 95th percentile of Polish adults. When the non-structural growth curve is analysed, a strong periodicity of growth can be found *(Figure 10b)*. There are 8 turning points presented in the velocity curve. Strong periodicity of growth can be emphasized also as the multi-level (M) tendency of growth *(Figure 10c)*. The growing boy changes his developmental levels in such a way that his intra-individual variation exceeds a value of 0.22.

When we take girls into account, there is the example *(Figure 11a)* of the quite normal developmental pace of **girl 154,** living in a family with low SES. She is slightly taller than her peers at the same age. The velocity curve indicates a tendency for restrained tempo around the pubertal spurt, therefore this girl has altered her percentile position from 75 to 50 percentile. The pubertal growth spurt in body height begins one year later than in the whole sample (age at TO of the girl is 9.29 yrs and mean age of TO is 8.56 yrs, respectively). Velocity at TO is 5.04 cm/yr. The delayed PHV time does not influence its duration, which is 2.85 years. The total adolescent increment is 31.11 cm, and the attained height is predicted as 168.63 cm. This girl will be assessed at the 50 percentile, as an average height in the population. Non-structural analysis indicates as many as 4 minima and maxima, and these are intensive to various extents, from 3.61 cm/yr to 8.98 cm/yr *(Figure 11b)*. Such a strong periodicity of growth is reflected in multi-level (M) tendency of growth *(Figure 11c)* which means that the growing girl undergoes changes in her developmental levels.

Girl 166 lives in a family with medium SES *(Figure 12a)*. The distance curve is at the 50 percentile, which means that the status of growth is perfectly average. Velocity curve indicates that the tempo is rather slow, since it is at the position of 25 percentile. The pubertal growth spurt in height begins at age 8.80 yrs (only two months later than average) and the velocity (MPP) is 5.01 cm/yr. PHV begins at 11.96 yrs, and its intensity is lower than average – 6.70 cm/yr. The total adolescent increment is 33.11 cm and attained height is 163.90 cm, slightly smaller than at average. The non-structural velocity curve indicates as many as 7 short-term growth spurts at various ages and intensity *(Figure 12b)*. There is a tendency for negative (N) pace of growth in body height in this girl *(Figure 12c)*. The negative tendency indicates that growth in body height takes place at monotonically lower developmental levels in the subsequent stages of ontogeny.

DISCUSSION

Considering complex characters or patterns such as height or growth, it is clear that they imply interaction of many factors. Therefore, as was previously pointed out, normal variability within a population for those characters is typically assumed to result from additive polygenic inheritance, but environmental factors make some contribution to the variation. The results obtained indicate a high

heritability for stature. The value of t^2, which indicates that 62% of the total variance is transmissible, is very similar to the data provided by Bouchard et al. (1980), Devor et al. (1986). The child on midparent regression also provides a support for the genetic control of growth. This does not mean that the same genes would operate during the whole growth period. As is postulated in Iwamoto-Knussman's model infancy, prepubertal spurt and pubertal spurt can be influenced by different genes.

Following numerous auxological studies of family resemblance for stature, it has been recognized that factors associated with the environment in which the subject lives modify the genetically programmed course of development. The environment, as mediated by five comprehensive, well-documented major factors – nutrition (food availability), high altitude environments, climate (heat, cold and relative humidity tolerance), urbanization and migration and socioeconomic status – is associated with variation in the size, proportions and composition of the human body. Environmental factors, both natural as well as cultural, influencing course of development are of special importance for growing children. Status and rate of growth are found to be the most sensitive indicators of environmental impacts of growth during childhood (Tanner, 1963; Goldstein, 1971; Chrząstek–Spruch, 1979; Lasker, Mascie–Taylor, 1984; Bielicki, 1986; Szopa, 1992). It was found that the adequacy of the total quantity of food consumed are essentials for human growth (Kimura, 1984; Susanne et al., 1987).

Following studies on Mexican and Guatemalan children (Johnston 1980), a canonical correlation analysis was used to calculate the rank order of importance of each of the independent variables for growth: parental occupation, rural-urban residence, ethnicity and nationality. (The absolute value of the canonical score is a measure of the relative influence of each of the independent variables on growth status). The canonical scores ranked in the following order: rural-urban residency 1.68, parental occupation (SES) 1.43, nationality 0.98, ethnicity 0.90.

Socioeconomic status has an equally important impact on growth and development but always as a secondary influence interacting with nutrition, health care and well-being. It is accepted that the education of the parents is connected with eating habits and numerous other factors which affect the growth and development of the child. Some authors have even shown that the status and rate of development depends more on psycho-social factors than on purely economic conditions (Tanner, 1992).

The results of this analysis suggest that SES influences the pattern of growth in height in such a way that the better the living conditions, the taller the children. This tendency is more marked in boys than in girls. They also emphasise that even within a seemingly homogeneous population subtle differences in SES have a significant influence on growth.

CONCLUSIONS

The results obtained indicate clear evidence for an individual strategy of development as the impact of genetic and environmental causes. Although there is clear evidence for high heritability of body height, environment plays an important role in modifying the genetically programmed course of growth. As development proceeds in different environments interacting with genotype, it seems that the proposed conceptual model of factors determining processes of growth is sufficient for better understanding the mechanism responsible for biological differences between social classes. The mechanism is assumed to be multiple, and is the result of the specific relationship between genetic susceptibility to environmental factors, and the regulation of growth by epigenetic factors which enables the undertaking of an individual strategy of development phenotypically recognized as a non-standardized pattern of growth (as was shown by individual examples). It seems that studies postulating a pulsatile nature of the growth in height can approximate this problem even better (Ashizawa, Kawabata, 1990; Lampl et. al., 1992; Hermanussen, Burmeister, 1993).

ACKNOWLEDGMENTS

This work was supported by KBN grant 6P 20401605. My special thanks are due to Dr. M. Sitek for her support with statistics and to Miss D. Juszczak for her kind assistance in collecting data.

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