Przegląd Antropologiczny • tom 59, s. 3–17, Poznań 1996

https://doi.org/10.18778/1898-6773.59.01

Analysis of transmissible and nontransmissible components of variation in human physique

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Abstract

The paper is aimed at decomposition the continuously varying phenotype into components due to transmissible and nontransmissible factors. The linear causal model (path analysis) was applied to incorporate the contribution of environmental sources of variation (described in terms of indices of socioeconomic status) to familial resemblance on physique (height and weight) in 342 nuclear families. Parameters of the causal model were estimated according to the TAU transmission model of Rice, Cloninger, Reich [1978] and linear constraints placed upon the parameters were tested. The proportion of total phenotypic variance accounted for by genetic and environmental transmissible factors was estimated to be 62% for height and 38% for weight.

Maria Kaczmarek, 1996; *Anthropological Review*, vol. 59, Poznań 1996, pp. 3–18, figs 5, tables 3. ISBN 83-86969-05-9, ISSN 0033-2003

Introduction

The study of inheritance of quantitative traits in human beings is a difficult undertaking because there is no possibility of experimental study in this field. The methodological implication of this fact is that quantitative analysis of the inheritance of body height or weight is restricted to the means, standard deviations and correlations available from samples of subjects sharing varying degrees of biological and environmental relationship [FALCONER 1981, CAVALLI-SFORZA, BODMER 1971]. Assuming that height and weight are multifactorial traits and normally distributed in a given breeding populations, the Pearson's correlation coefficient or Fisher's intraclass correlation coefficient are descriptive statistics for the relationship among relatives. These statistics constitute the maximum likelihood estimates of parameters of the multivariate distribution that describe the variation in physique among related persons.

With the advent of path analysis by Sewall Wright in 1934 and modern statistical methods for its implementation, new perspectives have to be opened for biometrical methods of indentifying different sources of variation in human quantitative traits [WRIGHT 1934 cited after LI 1975]. Path analysis and related structural equation models allow more complex hypotheses to be tested, where the contribution of environmental as well as genetic variances may be studied more comprehensively. These new approaches to analysis of quantitative traits are no longer limited to exclusive genetic hypotheses, but are extended to include cultural factors as

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well. The latter ones may be transmitted within and between families. The contrast between biological and cultural inheritance resolves itself into different nature of the transmission. In biological inheritance, information encoded in DNA is passed from parents to offspring whereas in the cultural inheritance, information may be passed in muldirectional ways not only on parent-offspring path but also among members of the group who may not be biologically related [CAVALLI-SFORZA, FELDMAN 1973].

There are numerous studies on familial correlations in body height and weight in the literature but these correlates do not enhance our understanding of the relative contribution of genes and environment in the phenotypic similarities within families (see for the review to [MUELLER 1976]). According to Fisher, the phenotypic similarity between relatives is due only to genetic factors (Fisher 1918, cited after FALCONER [1981]). However, in recent papers several authors have argued that the Fisherian treatment of a correlation between relatives tends to bias the analysis toward genetic causes because it omits potentially important cultural sources of correlation [RAO et al. 1976, GOLDBERGER 1978, Rice et al. 1978]. Cavalli-Sforza and Bodmer list the following important sources of familial covariance: 1. cultural factors; 2. within-individual genetic-environmental correlations that lead to correlated environmental variance components between parents and offsprings; 3. correlations between siblings sharing the same prenatal (maternal) and postnatal family environment where socioeconomic status and nutritional factors are the same [CAVALLI-SFORZA, BODMER 1971]. Indeed, as it has appeared from numerous data, environmental source of familial covariance has direct and indirect influences on variations in body size, particularly on a nutritional basis (among other authors: KAUR, SINGH [1981], SUSANNE [1987], BYARD et. al. [1983, 1993]).

Body size is the characteristic for which the level of family resemblance is high, but for which biological and environmental correlates are not yet well understood, in spite of the considerable attention that has been focused on the study of the genetics of these traits (namely body height). Therefore, the major purpose of this paper is to provide an additional information on the decomposition of the total phenotypic variance in body height and weight into components of transmissible and nontransmissible factors, with the resolution by path analysis.

Material and Methods

Data on body height and weight derives from 342 nuclear families, relatives of the children, participants in the Poznań Growth Study, carried out since 1985 [KACZMAREK 1995]. The longitudinal design of the study is aimed at monitoring the children's growth within stratified environments. Measurements of the child per se at the age of 14 years, of their parents and sibs, the interested correspondents supplied, were taken into analysis. Height of siblings were adjusted by regression procedure to 14 years of age. At this age the adult stature in boys is completed in 92,3% and in girls in 97,3% what means, especially in girls, that the stature at the age of 14 years is closed to its final value. It does not hold the same for weight. Maturation status of body weight at the age of 14 years is much lower than that of height and is completed

in 76,5% in boys and 88,5% in girls. In fact, the adult body size is gained at approximately 18 years of age althought in some individuals growth may be continued after the age of 18 [HULANICKA, KOTLARZ 1983].

The stratified environments are characterized by parental education, income, number of children in family and dwelling conditions. According to the rank of a single social variable, high (A), medium (B) and low (C) levels of socio-economic status (SES) were distinguished. High level of life conditions is said to be settled by families where both parents have academic education, one child, provide the highest financial and dwelling conditions; medium level of SES is settled by families, where parents have secondary education, two children and the medium values of the income and dwelling indices; low level of SES is settled by families where parents have primary or vocational education, three or more children, and do provide poor financial and dwelling conditions of life.

Analytical method of analysis.

Dimensions of adult body size are ultimate result of two biologically distinct processes: growth and development. Growth and development involve interactions among genes and between the timing and pattern of gene expression and the cellular and external environments in the course of ontogeny. Variation of these processes is attributable to genetic causes and environmental factors. There are four major categories of causal factors recognized as essential for control and regulation of the human development, growth and morphogenesis: intrinsic genetic, epigentic, maternal genetic and environmental factors. The causal factors of development mentioned above are related either by interaction or determination and constitute elements of the conceptual model of human growth and development presented graphically in Fig. 1.

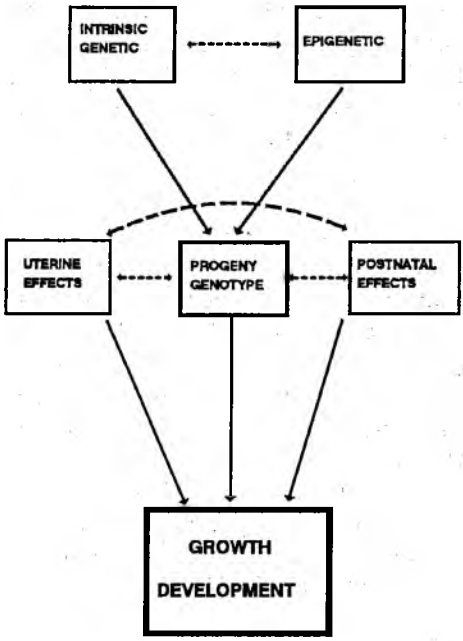

Fig. 1. A model of the causal factors controlling human growth, development and morphogenesis.

It is considered that the model makes a compilation of two conventional approaches to analysis of quantitative traits: that of quantitative genetics (intrinsic genetic and environmental factors) and that of developmental biology (epigenetic factor). Genetic control of development results from the fact, that progeny genome (its genes are transmissed from parents) provides the "blue-print" for development, and heritable epigenetic factors control the timing and pattern of gene expression. The environmental control of development means that non-heritable factors may blur

the contribution of intrinsic genetic and heritable epigenetic factors to the development within the reaction norm of genotype.

Analytic interpretation of this theoretical model tends to establish the part played by heredity and environment in determining quantitatively varying characters. Because of its additive property, total phenotypic variance of the quantitative trait can be formulated as:

$$V_P = V_G + V_E + 2\,\mathrm{cov}_{GE} + V_{GE} + e$$

where: V_P – total phenotypic variance; V_G – genetic variance; V_E – environmental variance; $2\mathrm{cov}_{GE}$ – genotype and environment covariance, if any; V_{GE} – variance caused by the effect of genotype-environment interaction, if any; e – error.

If genetic variance is subdivided into different component parts and environmental variance is subdivided in the within and between family components, the above equation may also be written as: [SUSANNE 1994:32]

$$V_P = V_A + V_D + V_I + V_{EC} + V_{EW}$$

where: V_P – total phenotypic variance; V_A – the additive factors; V_D – the dominant factors; V_I – the epistasis factors; V_{EC} – the environmental variance contributing to the between family component; V_{EW} – the environmental variance contributing to the within family component.

As it has already been mentioned, there are different sources of environmental variance. Having regard to this, the environmental variance in human beings may be subdivided: [CAVALLI-SFORZA and BODMER 1971: 588-589]

$$V_E = V_{ind} + V_{fam} + V_{soc} + V_{rac} + V_{GE}$$

where: V_E – environmental variance; V_{ind} – the variance among individuals within families. It is included in all families, but may vary from family to family; V_{fam} – the variance among families within socioeconomic strata. It inflates the covariance between parent and offspring; V_{soc} – the variance among socioeconomic strata. Cultural differences among families or social groups may be maintained by sociocultural inheritance that leads to correlations between relatives that are very difficult to distinguish from those that are due to genetic determination. This part of environmental variation may be better understood when the concept of "culture gene" and "extended phenotype" are taken into consideration [CORLUY 1983]; V_{rac} – the variance in environmental conditions accompanying racial differences, included in which are the sociocultural differences above; V_{GE} – the variance due to the genotype-environment interaction that occurs when given genotype shows different phenotypes in different environments within the frame of its reaction norm [VOLTERECK 1909, STEARNS 1992].

However, it should be stressed once more, that it is possible in experimental animals, but not for human beings, to control the environment precisely. Indeed, separating V_{GE} from V_G and V_E in human beings is a problem of extreme complexity. Therefore, for further considerations we neglect sources of variation that result from genotype-environment covariance and genotype-environment interaction, by undertaking the assumption that both, *GE* covariance and *GE* interaction are not significant. It is also assumed that there is no evidence of X-linked influence and no or little linkage between genes of the postulated polygene for a trait. It is further postulated, that the only source of random errors in the model is the error of

measurement. In our sample, technical error of measurement (TEM) for intra-observer error in height is 0.42 cm and inter-observer error in weight is 1.03 g. The value of reliability coefficients ranges from 0.94 for weight to 0.98 for height. As a result of the above: $cov_{GE} = V_{GE} = e = 0$ thus, the total phenotypic variance is partitioned into components representing uncorrelated genotypic and environmental determination. The simplified form of the initial equation is as follows:

$$V_P = V_G + V_E.$$

where: V_P – total phenotypic variance; V_G – genetic variance; V_E – environmental variance.

Statistical method of analysis: path model.

Numerical estimates for deriving expected familial correlations are based on path analysis. The path analysis is a multifactorial procedure of computing correlations between variables connected by the specified causal linear relationship: putative causal variables with the response variables. This approach enables the investigator to grasp a complex system of relationships and to examine its implications with the use of parameters termed "path coefficients". Path coefficients between causal (independent) variables and response (dependent) variables are the standardized regression coefficients of the dependent variables and are obtained through a sequence of multiple regression analyses. Relationships between variables are presented in schematic form as the path diagram. In the present study, the procedure developed by RICE, CLONINGER and REICH [1978] known as the TAU transmission model for sources of resemblance between parent and offspring and between two siblings, was used. A graphical representation of the TAU transmission model used in this study is presented in Fig. 2.

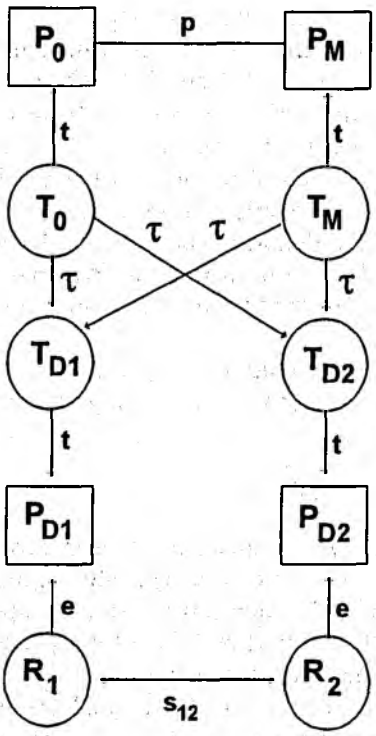

Fig. 2. The TAU transmission model for sources of resemblances between parents and offspring and between two siblings. Observed variables are shown in squares, where P denotes phenotype: O – father, M – mother, D_1, D_2 – offspring. Latent variables are shown in circles: T denotes genetic and cultural factors that are transmissible from parent to offspring; R denotes nontransmissible factors. The parameters of the model are: p – phenotypic assortative mating between spouses; t – the transmissibility of the phenotype; τ – the transmission of the phenotype (tau = 0.5 is polygenic transmission); s – the correlation of nontransmissible components among full siblings; e – error.

The path diagram depicts a nuclear family with two children where the subscripts O, M, D1 and D1 refer to the father, mother and children, respectively. The observed variables are presented in boxes, where P denotes phenotype. Latent or unobservable variables are in circles, where T denotes genetic and cultural factors that are transmissible from parents to offsprings and R denotes nontransmissible factors.

The diagram represents a linear structural model connecting variables in the form:

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

where: P – standardized phenotype; T – transmissible component either genetic or cultural; R – nontransmissible component; t^2 – the proportion of the total phenotypic variance that is transmissible.

The first step of our analysis was to predict the pattern of correlations before incorporating them in path analysis. The equality of parent-offspring and sibling correlations were tested following test of homogeneity [SNEDECOR, COHRAN 1967]. The full TAU transmission model, used in the present study, has five parameters: p, t, τ_F, τ_M, $s_{12} = s_{21}$, where: p is the correlation between the spouses' phenotypes; t^2 is the transmissiblity e.g. proportion of the total phenotypic variance that is transmissible; τ_F, τ_M are the respective effects of the father's and mother's transmissible components on the transmissible components of their offspring. Assuming that the value of 0.5 is the expectation under simple polygenic inheritance, τ_F and τ_M were fixed at 0.5 and all analyses were undertaken under this constraint; s is the correlation between the nontransmissible components of full siblings due to their shared environments.

From our data, father-mother and sex-specific parent-offspring and sib pairs yielded a total of eight observed correlations. Disregarding sex-specific correlations we used four of the mentioned above correlations e.g. for parameters estimation: father-mother, father-offspring, mother-offspring, sib-sib.

The plausibility of the full and constraint models were tested by a goodness of fit statistic comparing the observed correlations with those deduced from the model using the likelihood ratio test [KENDALL, STEWARD 1973, RICE et al. 1980].

A series of hypotheses were tested, that: 1. there is no marital resemblance ($p = 0$); 2. there is no sibling correlation in nontransmissible environmental factors ($s = 0$); 3. there is no intergenerational transmission ($t^2 = 0$); and 4. no familial resemblance ($t^2 = p = s = 0$).

All statistical procedures were implemented in the programme of the Css: STATISTICA/1993.

Results

Variability in growth pattern at specified ages and adolescent growth spurt in height

Our observed data on growth in height of 284 boys and 270 girls aged 5 to 15 years from Poznań Growth Study, were fitted to the JPA2 model [JOLICOEUR, PONTIER, ABIDI 1992]. By means of the fitting process, the mean heights and height velocities were calculated for ages 6, 9, 12 and 14 and for characteristics of the adolescent growth spurt in height. The

means and standard deviations are shown in Table 1.

Table 1. Means and standard deviations of height and height velocity at specified ages and adolescent spurt in height in male and female Poznań participants.

Characteristic	Boys *N*=284		Girls *N*=270	
	Mean	*SD*	Mean	*SD*
height at 6 (cm)	117.32	4.05	116.65	5.38
height at 9 (cm)	134.95	5.38	133.78	6.58
height at 12 (cm)	151.96	6.91	154.39	7.52
height at 14 (cm)	166.49	7.31	162.85	6.09
velocity at 6 (cm/y)	6.36	1.03	6.20	1.29
velocity at 9 cm/y)	5.61	1.29	4.93	1.03
velocity at 12 cm/y)	7.52	1.45	5.87	1.51
velocity at 14 cm/y)	3.00	2.21	0.98	1.29
age TO (yrs)	10.44	1.26	8.56	1.44
age PHV (yrs)	13.68	1.21	11.49	1.29
height TO (cm)	142.67	7.93	132.78	7.79
height PHV (cm)	163.86	7.21	150.33	6.75
velocity TO cm/y)	4.82	0.97	5.23	0.78
velocity PHV cm/y)	9.44	1.80	7.71	1.69

The height data show considerable variability. However, the pattern of variability is shifted when measurements are made at specified ages (chronological age) and at adolescent growth spurt (biological age). When taking the first approach, the smallest range of variability in the growth status and velocity is found at the age of 6 irresepective sex. The largest range of variability both in the growth status and velocity is found at the age of 14 in boys and 12 in girls. When the adolescent growth spurt is under consideration, the greatest variability in both age and height is seeen at spurt take-off. Whereas, the variability of velocity at peak is twice larger than that at take-off (*SD* is 0.97 cm at TO and 1.80 cm at PHV in boys; in girls 0.78 cm and 1.69 cm respectively). The different pattern of variability in growth status and velocity between boys and girls supports our present knowledge about the process of growth in metric traits.

Genetic control of growth pattern

Growth is more or less continuous phenomenon from conception to maturity and factors influencing growth processes change according to 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 the stature was distributed into five groups with a class interval of the half *SD* each, as follows:
1. <$-\infty$, 162.8 cm>; 2. <162.8, 167.5 cm>; 3. <167.5, 172.2 cm>; 4. <172.2, 176.8 cm>; 5. <176.8, $+\infty$ cm>. The results are presented in Fig. 3.

There is clear evidence for the mid-parent gradients in the children's distance and velocity curves, more clear in girls that in boys. The taller parents the taller children.

When the midparent-offsprig correlation is taken at specified age e.g. at the age of 14, it is interesting to note, as it is evident from the regression of offspring height on midparent height (Fig. 4), that the correlation is more emphasized in girls (respective coefficient of correlation *r* in

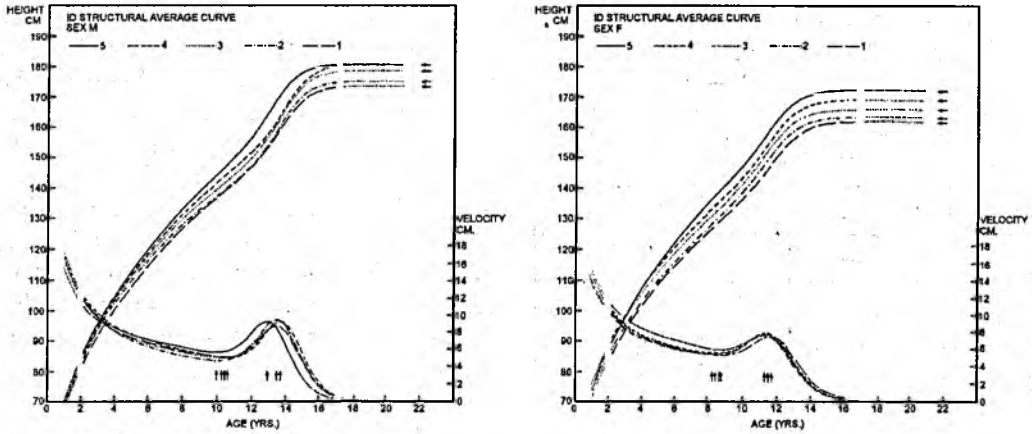

Fig. 3. Distance and velocity curves of the children's body height according to the category of their mid-parent value: M – boys, F – girls

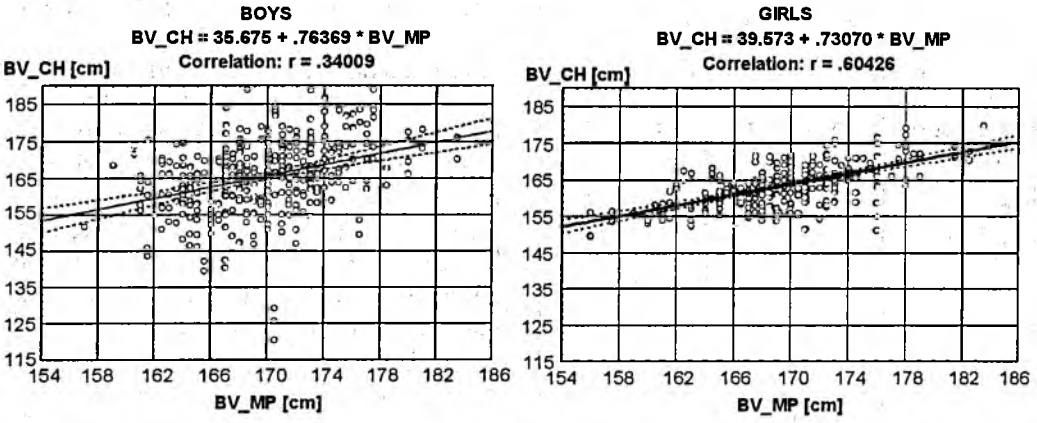

Fig. 4. Regression (95% confid.) of offspring height at the age of 14 on midparent height

14 years old girls is 0.60 and in boys is 0.34) who are closer at this time than boys to their adult value in stature.

Genetic control of growth and hereditary component in phenotypic variation as derived from the midparent gradient in children's height and the regression of offspring height on midparent height, may be translated by an increase of the autocorrelation, where measurement of the child during growth is correlated with his/her final height in adulthood.

Environmental control of growth pattern

It is also perfectly clear that body height is particularly prone to nutritional and environmental influences. Environmental control of the growth pattern is tend to be tested within the range of socioeconomic conditions. Distance and velocity curves of growth plotted against three levels of SES of family are presented in Fig. 5.

From pattern in distance and velocity curves, resulting socioeconomic gradients

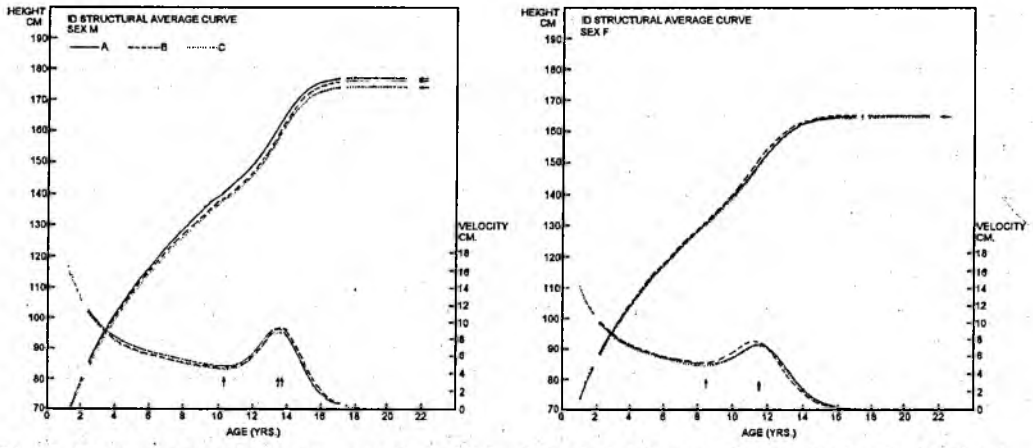

Fig. 5. Distance and velocity curves of the children's body height according to the level of SES: A – high (continuous line), B – medium (broken line), C – low (dotted line); M – boys, F – girls.

in heigh are shown. The higher SES, the better conditions of life, the taller children. It seems that this gradient is more emphasized in boys that in girls.

Intergenerational components of the phenotypic variation in physique

Table 2 shows parent-offspring and sibling correlations in height and weight. The data indicate that similarities between relatives are statistically significant.

As resulted from the Snedecor, Cohran test of homogeneity, the parent-offspring and sibling correlations are not sex specific, neither for heigth nor for weight. For the hypothesis about the equality among sexes, denoted as: $F - s = F - d = M - d = M - s = B - b = B - s = S - s$, the value of chi-square is 3.64 for height and 3.98 for weight with $df = 6$.

From path analysis, resulting likelihood ratio chi-square values for the stature and body weight under the full TAU transmission model and linear constraints that were placed upon parameters are presented in Table 3.

The degrees of freedom of chi-square were given by the number of constraints.

Table 2. Observed familial correlations and sample size for body height and weight from Poznań population.

Relatives	Stature			Weight		
	number of pairs	r	S_e	number of pairs	r	S_e
Spouses	342	0.249*	0.16	342	0.174*	0.18
Father – son	204	0.348*	0.19	204	0.132*	0.17
Father –daughter	184	0.370*	0.18	184	0.139*	0.16
Mother – son	201	0.314*	0.15	201	0.235*	0.14
Mother – daughter	179	0.422*	0.11	179	0.253*	0.12
Brother – brother	149	0.448*	0.09	149	0.256*	0.11
Brother – sister	185	0.429*	0.07	185	0.210*	0.10
Sister – sister	135	0.498*	0.09	135	0.229*	0.11

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

Table 3. Goodness-of-fit chi-square test for general path model and likelihood ratio chi-square tests for simplified path models.

	General model $df = 1$	$p = 0$ $df = 1$	$t^2 = 0$ $df = 1$	$s = 0$ $df = 1$	$t^2=p=s=0$ $df = 3$
Stature	1.48	8.24*	48.20*	1.74	58.40*
Weight	0.70	4.80*	19.10*	0.49	30.24*

df = degrees of freedom; * significant at $\alpha = 0.05$; ** significant at $\alpha = 0.01$

The general model was the no-constraint model (first column), three parameters were estimated: t^2, p, s, under the assumption that $\tau_F = \tau_M = 0.5$ (poligenic transmission) and similarities between spouses for height is $p = 0.249$ and for weight is $p=0.174$. The result of the goodness of fit chi-square test for the full model $\chi^2 = 1.48$ for height and for weight $\chi^2 = 0.70$ with $df = 1$. The one degree of freedom was given by four observed correlations less three estimated parameters (t, p, s). The results obtained indicate that the general model provides a good fit to the observed familial correlations for height and weight as well.

In the next columns of Table 3 are presented the likelihood ratio chi-square values for hypotheses involving linear model constraints in addition to the assumption that $\tau_F = \tau_M = 0.5$. Considering hypothesis of no parent assortative mating denoted as $p = 0$ with 1 *df*, the result of chi-square test is shown in the second column of the table 3. This hypothesis can be rejected because the chi-square value 8.24 for stature and 4.80 for weight are significantly different from zero at $\alpha = 0.05$. This indicates that there is assortative mating between spouses and it is known that it is positive [KACZMAREK 1995].

The third column of Table 3 shows likelihood ratio chi-square values for the hypothesis concerning parent-offspring (vertical) transmission, denoted as $t^2 = 0$ with 1 *df*. For stature $\chi^2 = 48.20$ and for weight $\chi^2 = 19.10$ what result in rejection of this hypothesis for both traits – height and weight. The results of test gives evidence for direct intergenerational transmission of genetic and cultural causal factors controlling and regulating children's growth in body height and weight.

Further test, i.e., that there is no sibling correlation in nontransmissible environmental factors e.g. that $s = 0$ with $df = 1$, resulted in $\chi^2 = 1.74$ for stature and $\chi^2 = 0.49$ for weight what means that the null hypothesis cannot be rejected. In other words, similarity between siblings in body height and weight are only due to transmissible genetic and cultural factors, not due to shared environmental conditions of their life influenced by nontransmissible factors.

The last hypothesis tested is of no familial resemblance denoted as: $t^2 = p = s = 0$. The respective values of likelihood ratio chi-square for height and weight are: $\chi^2 = 58.40$ and $\chi^2 = 30.24$ resulted in rejection of the hypothesis for both traits. There is significant similarity in physique between biologically related persons. Familial resemblance is the result of the intergenerational (vertical) transmission of genetic and cultural factors. In our sample, the estimated transmissibility value t for the stature is $t = 0.789$, thus $t^2 = 0.624$ with $SE = 0.059$ and for the weight $t = 0.620$, thus $t^2 = 0.384$ with $SE = 0.132$.

Discussion

The results of the present study are shown to be in accord with other studies of anthropomentric data where family resemblance in physique were analyzed by path analysis [BOUCHARD et al. 1980, DEVOR et al. 1986]. The estimates of transmissibility for linear dimensions (body height) indicate higher level of intergenerational transmissiblity ($t^2 = 0.624$) than for body weight ($t^2 = 0.384$).

Although, from our investigation it could not be estimated how much of family resemblance is due to genes and how much to the environment, the results are certainly compatible with the results of several studies on familial data or on twin data [SUSANNE 1975, SUSANNE et al. 1978, KAUR et al. 1981, DEVI et. al. 1983, BYARD et al. 1993, HAUSPIE et al. 1994]. A comparison of the results obtained with those from studies in which twin data were used, showed that the latter produced somewhat higher estimates for most traits [WILSON 1976, BERGMAN, GORĄCY 1984]. Following CAVALLI-SFORZA & BODMER [1971], heritability estimates for stature ranges from 50% to 95% of the total phenotypic variance.

The obtained midparent gradients in growth curves in height confirm that growth status and growth rate are under genetic control. The published results indicate that the influence of the hereditary factors on body size is altered during individual life. The influence of fetal hereditary factors on body size is small in comparison with the fetal effects of common environment. The latter was estimated at 49 to 62% [PENROSE 1961, RAO et al. 1974]. In the postnatal period of growth and development, the hereditary component of variation in body size increases as a result of an increase of the autocorrelation. The pattern of autocorrelation shifts from the first year of life, then autocorrelation increases very rapidly to puberty when it slightly decreases [TANNER 1960, ASHIZAWA et al. 1977]. As it can be seen from studies on twins, ecosensitivity of the growth processes increases in the time of puberty, it is also more strongly expressed in boys than in girls [BERGMAN 1988, HAUSPIE et al. 1994].

Genetic control of patterns of growth confirm the strong genetical influence. Heritability is around 0.8 with a possible rank order of: {age at PHV >adult size > velocity at PHV} [BERGMAN et al. 1981, HAUSPIE et al. 1994]. When the proportion of the total phenotypic variance was taken into accout the pubertal parameters were shown to be attributed to the differences between family with a range of 15 to 33%, or even 41 to 71%, confirming the genetical influence on the adolescent growth spurt both in time and its intensity [HAUSPIE et al. 1982, BYARD et al. 1993]. For events of pubertal maturation (maturation of secodary sex traits, age at menarche, growth spurt) correlations observed in familial studies are relatively low [SUSANNE 1980, SHARMA 1983].

The socioeconomic gradients in body height found in our data, support the fact of ecosensitivity of the body height to environmental stresses. According to the Jedlińska's findings, three variables: size of housing estate, education of both parents and number of children in family, taken together, explain as much as 9% of the total phenotypic variance in prepubertal and postpubertal phases of growth in stature in both sexes. In the course of puberty the amount of the variance explained by the above variables increases

to 11% in girls and to 20% in boys [JEDLIŃSKA 1985]. These findings may be misleading as environmental component (described in terms of the social variables) of the total phenotypic variance constitutes only small part of it. The majority of the total phenotypic variance is explained by genotypic component. Indeed, the empirical data based on family resemblance in physique indicate a clear evidence for the significant amount of the genetic component in the total phenotypic variance in stature (results of this work, BYARD et al. [1991], HAUSPIE et al. [1994]).

Ecological consequences of the socioeconomic status are always explained as secondary influences, interacting with nutrition, altitute, climate, migration, effects of behaviour in family which are connected with organization of life in family, nutrition, minimal stresses, higiene and health state (among many other: GOLDSTEIN [1981], BIELICKI [1986], HAUSPIE et al. [1996]). TANNER [1992:390] emphasizes the role of family, its organization, life style and well being in normal growth of a child. Following studies on Mexican and Guatemalan children [JOHNSTON 1980, 1994] a canonical correlation analysis was used to calculate the rank order of importance of each of the independent variables: parental occupation, rural-urban residence, ethnicity and nationality for growth. (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. Direct influence on growth and development has the pattern of nutrition, an adequate supply of energy, amino acids, water, lipids, vitamins and minerals [SUZANNE et al. 1987, DAVIES, PREECE 1988]. On the other hand it is known that a pattern of nutrition is closely connected with the level of education of parents, namely of mothers who are carried after their children. In this way education is again pointed out as the decisive factor for postnatal growth.

Tests of $s = 0$ indicate that nontransmissible environmental factors are nonsignificantly relevant to the resemblance in physique between siblings. Neither common prenatal environment nor postnatal ones are relevant for the similarities. The similarities between sibs depend on transmisibillity component of the variance. It is true that during development correlations between sibs of the same age increase gradually [GARN et al. 1966, FURUSHO 1974]. The same is true for MZ and DZ twin pairs. The differences between MZ twins who share the same genotype disappeared gradually when the concordant gene activity starts. DZ concordance observed at birth through maternal factors decreases regularly after birth [VAN VERSCHUER 1934, WILSON 1979]. Different influences of common familial environment has been shown by Mueller [1978]. The greater the age or parity difference between sibs, the lower the sib correlation at least for measurements sensible to the environment.

In analysing nuclear families, observations on phenotypes alone do not allow for the explicit resolution of genetic and environmental sources of familial resemblance. Rice et al. [1978] have emphasized that the transmission of cultural factors can stimulate the polygenic inheritance in the pseudogenetic model so that t^2 is not identical with heritability h^2. In other words when τ is given as 0.5 under polygenic inheritance, cultural transmis-

sion may be confounded with genetic transmission. For the traits such as body height and weight, an example of non sex-specific cultural transmission may be that of nutritional behaviour. If the purpose is to resolve the genetic and cultural sources of family resemblance it may be advised to gather data on more remote relatives together with that on nuclear families in order to make use of the comprehensive path models.

Conclusions

Through an analytic path model it was possible to search for transmissible and nontransmissible components of familial resemblance in physique. The results of this investigation may be concluded as follows:

1. Factors transmissible from parent to their offspring, being sources for familial resemblance in physique are of biological and cultural properties. The cultural factors described in terms of social and economic variables may influence patterns of behaviour within family which may be either permissive or not to the child's growth.

2. The proportion of variance accounted for by genetic and cultural transmissible factors is estimated to be 62% for height and 38% for body weight.

3. Nontransmissible environmental effects common to siblings are not relevant for their resemblance in body height and weight.

References

ASHIZAWA K., C. TAKAHASHI, S. YANAGISAWA, 1977, *Stature and body weight growth patterns from longitudinal data of Japanese children born during World War II*, J. Hum. Ergol., 6, 29–38

BERGMAN P., 1988, *Zagadnienie genetycznej determinacji rozwoju w okresie pokwitania*, Mat. i Prace Antropol. 108, 165–216

BERGMAN P., F. GRZESIOWSKI, A. SZMYD, 1981, *Verwendung der logistischen Funktion zur quantitativen Beschreibung des Korperlangenwachstum bei Zwillingen*, Homo, 32, 81–89

BERGMAN P., M. GORĄCY, 1984, *The timing of adolescent growth spurt of ten body dimensions in boys and girls of the Wroclaw Longitudinal Twin Study*, J. Hum. Evol. 13, 339–347

BIELICKI T., 1986, *Physical Growth as a Measure of the Economic Well-being of Populations: The Twentieth Century*, [in:] *Human Growth*. Falkner F., J.M Tanner., (eds..), vol. 3, 2nd edition, Plenum Press, New York, 283–305

BIELICKI T., Z. WELON, 1971, *Further investigations of parent-child similarity in stature as assessed from longitudinal data*, Hum. Biol., 43, 4, 517–525

BOUCHARD C., A. DEMIRJIAN , R.M. MALINA, 1980, *Path analysis of family resemblance in physique*, Stud. Phys. Anthrop., 6, 61–70

BYARD P.J., R.M. SIERVOGEL, A.F. ROCHE, 1983, *Familial correlations for serial measurements of recumbent length and stature*, Ann. Hum. Biol., 10, 3, 281–293

BYARD P.J., S. GUO, A.F. ROCHE, 1991, *Family resemblance for patterns of growth in early childhood*, Am. J. Hum. Biol., 3, 331–337

BYARD P.J., S. GUO, A.F. ROCHE, 1993, *Family resemblance for Preece-Baines growth curve parameters in the Fels Longitudinal Growth Study*, Am. J. Hum. Biol., 5, 151–157

CAVALLI-SFORZA L.L., W.F. BODMER, 1971, *The Genetics of Human Populations*, W.H. Freeman and Company, San Francisco

CAVALLI-SFORZA L.L., M.W. FELDMAN, 1973, *Cultural versus biological inheritance: phenotypic transmission from parents to children*, Am. J. Hum. Genet., 25, 618–637

DAVIES P.S.W., M.A. PREECE, 1988, *Body composition in children: methods of assessment*, [in:] *The physiology of human growth*, Tanner J.M., M.A. Preece (eds.), Cambridge University Press, 95–107

DEVI M.R., G.G. REDDI, 1983, *Heritability of body measurements among the Jalari population in Visakhapatnam*, Ann. Hum. Biol. 10, 483–485

DEVOR E.J., M., MCGUE, M., CRAWFORD, P.M. LIN, 1986, *Transmissible and nontransmissi-*

ble components of anthropometric variation in the Alexanderwohl Mennonites: II Resolution by path analysi, Am. J. Phys. Anthrop., 69, 83–92

FALCONER D.S. 1981, *Introduction to Quantitative Genetics,* 2nd Ed. London, Longman Group Limited

FURUSHO T., 1974, *Genetic study of stature,* Jpn. J. Hum. Genet., 19, 1–14

GARN S.M., C.G. ROHMANN, 1966, *Interaction of nutrition and genetics in the timing of growth and development,* Pediatr. Clin. North. Am., 13, 353–362

GOLDBERGER A.S., 1978, *The nonresolution of IQ inheritance by path analysis,* Am. J. Hum. Genet., 30, 442–445

GOLDSTEIN H., 1971, *Factors influencing the height of seven-year-old children – results from the National Child Development Study in Human Biology,* Ann. Hum. Biol., 43, 397–411

HAUSPIE R.C., S.R. DAS, M.A. PREECE, J.M. TANNER, 1982, *Degree of resemblance of the pattern of growth among sibs in families in West Bengal (India),* Ann. Hum. Biol., 9, 171–174

HAUSPIE R.C., P. BERGMAN, T. BIELICKI, C. SUZANNE, 1994, *Genetic variance in the pattern of the growth curve for height: a longitudinal analysis of male twins,* Ann. Hum. Biol., 21, 4, 347–362

HAUSPIE R.C., H. CHRZĄSTEK-SPRUCH, G. VERLEYE, M.A. KOZŁOWSKA, C. SUSANNE, 1996, *Determinants of growth in body length from birth to 6 years of age; a longitudinal study of Lublin children,* Am. J. Hum. Biol., 8, 21–29

HULANICKA B., K. KOTLARZ, 1983, *Ostateczna wysokość ciała,* Przegl. Antrop., 49, 1–2, 15–25

JEDLIŃSKA W., 1985, *Wpływ niektórych czynników środowiska społecznego na wysokość ciała dzieci szkolnych w Polsce,* Przegl. Antrop., 51, 1–2, 15–37

JOHNSTON F.E., 1980, *Nutrition and growth,* [in:] *Human Physical Growth and Maturation,* F.E. Johnston, A. Roche & Ch. Susanne (eds.), New York, Plenum, 291–300

JOHNSTON F.E., 1994, *Growth, growing up, and the social and economic environments of children,* [in:] *Auxology '94,* O. Eiben (ed.) Humanbiol. Budapest., 25, 135–143

JOLICOEUR P., J. PONTIER, H. ABIDI, 1992, *Asymptotic models for the longitudinal growth of human stature,* Am. J. Hum. Biol., 4, 461–468

KACZMAREK M., 1995, *Wpływ warunków życia na wzrastanie i rozwój człowieka,* Wydawnictwo Naukowe UAM, Seria: Antropologia 20, 1–111

KAUR D.P., R. SINGH, 1981, *Parent-adult offspring correlations and heritability of body measurements in a rural Indian population,* Ann. Hum. Biol., 8, 333–339

KENDALL M.G., A. STUART, 1973, *The Advanced Theory of Statistics, vol. 2. Inference and Relationship,* New York, Hafner

LI C.C., 1975, *Path Analysis – A Primer,* Boxwood Press, Pacific Grove, California

MUELLER W.H., 1976, *Parent-child correlations for stature and weight among school-aged children: A review of 24 studies,* Hum. Biol., 48, 379–397

MUELLER W.H., 1978, *Transient environmental changes and age limited genes as causes of variation in sib-sib and parent-offspring correlations,* Ann. Hum. Biol. 5, 395–398

NEALE M.C., L.R. CARDON, 1992, *Methodology for genetics studies of twins and families,* NATO ASI Series, Series D: Behavioural and Social Sciences, vol. 67, Kluver Academic Press

PENROSE L.S., 1961, *Recent Advances in Human Genetics,* Churchill, London

RAO, D.C., N.E. MORTON, S. YEE, 1974, *Analysis of family resemblance. II. A linear model for familial correlation,* Am. J. Hum. Genet., 26, 331–359

RAO, D.C., C.J. MAC LEAN, N.E. MORTON, S. YEE, 1975, *Analysis of family resemblance. V. Height and weight in northeastern Brazil,* Am. J. Hum. Genet., 27, 507–520

RAO, D.C., N.E. MORTON, S. YEE, 1976, *Resolution of cultural and biological inheritance by path analysis,* Am. J. Hum. Genet., 28, 228–242

RAO, D.C., P.M. LASKARZEWSKI, J.A. MORRISON, P. KHOURY, K. KELLY, R. WETTE, J. RUSSEL, C.J. GLUECK, 1982, *The Cincinnati Lipid Research Clinic Family Study: Cultural and biological determinants of lipids and lipoprotein concentrations,* Am. J. Hum. Genet., 34, 888–903

RICE J., C.R. CLONINGER, T. REICH, 1978, *Multifactorial inheritance with cultural transmission and assortative mating. I. Description and basic properties of the unitary models,* Am. J. Hum. Genet., 30, 618–643

RICE J., C.R. CLONINGER, T. REICH, 1980, *General causal models for sex differences in the familial transmission of multifactorial traits: An application to human spatial visualizing ability*, Soc. Biol., 27, 36–47

SHARMA J.C., 1983, *The genetic contribution to pubertal growth and development studied by longitudinal growth data on twins*, Ann. Hum. Biol., 10, 163–171

SNEDECOR G., W. COHRAN, 1967, *Statistical Methods*, Ames, Iowa State University Press, 186–218

STEARNS S.C., 1992, *The evolution of life histories*, Oxford University Press

SUSANNE C., 1975, *Genetic and environmental influences on morphological characteristics*, Ann. Hum. Biol., 2, 279–287

SUSANNE C., 1980, *Socioeconomic differences in human growth*, [in:] *Human Physical Growth and Maturation*, Johnson F.E., A.F. Roche, C. Susanne (eds.), Plenum Press, New York, 329–338

SUSANNE C., 1994, *Genetics of growth*, [in:] *Auxology '94*, O. Eiben (ed.), Humanbiol. Budapest., 25, 31–39

SUSANNE C., D. DASH SHARMA, 1978, *Multivariate analysis of head measurements in Punjabi families*, Ann. Hum. Biol., 5, 179–183

SUSANNE C., R. HAUSPIE, Y. LEPAGE, M. VERCAUTEREN, 1987, *Nutrition and growth*, World Review of Nutrition and Diet, 53, 69–170

TANNER J.M., 1960, *Genetics of human growth* [in:] *Human Growth. Symposia of the Society for the Study of Human Biology*, Pergamon Press, Elmsdorf, New York

TANNER J.M., 1992, *Human growth and constitution*, [in:] *Human Biology: an introduction to human evolution, variation, growth and ecology – 3rd ed.*, Harrison G.A., J.M. Tanner, D.R. Pilbeam, P.T. Baker (eds.), Oxford University Press, London, New York, Sydney, 339–435

VAN VERSCHUER O., 1934, *Die Erbbedingtheit des Korperwachstums*, Zeitschrift. Morph. Antrhop., 34, 398–412

VOLTERECK O., 1909. *Weitere experimentelle Untersuchungen uber Artveranderung, speziell uber das Wesen quantitativer Artunterschiede bei Daphninde*, Verh. Deutsch. Zool. Gesell., 12, 110 –172

WILSON R.S., 1976, *Concordance in physical growth for monozygotic and dizygotic twins*, Ann. Hum. Biol., 3, 1–10

WILSON R. S., 1979, *Twin growth: initial deficit, recovery, and trends in concordance from birth to nine years*, Ann. Hum. Biol., 6, 205–220

Streszczenie

ANALIZA PRZEKAZYWALNYCH I NIEPRZEKAZYWALNYCH KOMPONENTÓW WARIANCJI FENOTYPOWEJ BUDOWY CIAŁA CZŁOWIEKA. W pracy przedstawiono metodologiczne i metodyczne aspekty badań nad genetycznymi i środowiskowymi determinantami zmienności budowy ciała człowieka. Materiał empiryczny obejmował pomiary wysokości i masy ciała rodziców i rodzeństwa dzieci uczestniczących w Poznańskich Badaniach Długofalowych, łącznie 342 rodziny podstawowe. Środowisko życia rodzin wyznaczono w zależności od poziomu statusu społeczno-ekonomicznego. Do modelu teoretycznego dopasowano statystyczny model przyczynowo-skutkowy (model analizy ścieżkowej) na podstawie którego obliczono udział czynników przekazywanych (genetycznych i kulturowych) przez rodziców w całkowitej wariancji fenotypowej cech wielkości ciała potomstwa. Udział ten wynosi odpowiednio: 62% dla wysokości ciała i 38% dla masy ciała.