Relative Effect of Genetic and Environmental Factors on Body Height: Differences Across Birth Cohorts Among Finnish Men and Women

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ABSTRACT

Objectives. This study examined the change in heritability of adult body height across birth cohorts in Finland.

Methods. In 1981, cross-sectional questionnaires were completed by 10 968 twin pairs born before 1958. The effect of genetic factors was estimated via genetic modeling.

Results. Heritability increased from the cohort born before 1929 (0.76, 95% confidence interval [CI] = 0.65, 0.88 in men; 0.66, 95% CI = 0.55, 0.77 in women) to that born in 1947 through 1957 (0.81, 95% CI = 0.73, 0.87 in men; 0.82, 95% CI = 0.75, 0.89 in women).

Conclusions. Heritability of height increased across Finnish birth cohorts born in the first half of this century and leveled off after World War II. Environmental factors, compared with genetic factors, appear to be more important among women than men. (*Am J Public Health*. 2000;90:627–630)

Body height is a multifactorial characteristic that responds to both genetic and environmental influences. The genetic background of body height is not yet well known, but the latest studies suggest that the *PHOG* gene in the pseudoautosomal region of sex chromosome X is likely to have a significant effect on body height.[1] Environmental factors during pregnancy, childhood, and adolescence, such as maternal smoking,[2] undernutrition during pregnancy[3] and early life,[4] diseases,[5] increased energy consumption,[6] and even psychosocial factors,[7] can contribute to a slower growth velocity and to the final achieved body height.

A few estimates of the heritability of body height (i.e., the proportion of variance due to genetic factors) have been made. In Anglo-American studies,[8–10] heritability has been found to be about 0.80. Lower estimates have been found in West Africa (0.56)[11] and Finland (0.58),[12] possibly because of a greater effect of environmental factors or genetically homogeneous populations. The effect of environmental factors on body height has been examined in genetically homogeneous populations. Sociodemographic differences in body height are associated with differences in the standard of living.[13] In developed countries, the greatest differences have been found between birth cohorts.[14]

In societies with a low standard of living, heritability is likely to be lower than in affluent societies because of the stronger effect of environmental factors. Mueller[15] compared parent–child correlations for body height in 24 previous studies. He found that correlations were higher in European than in non-European countries, probably because of differences in environmental factors.

Finland offers good opportunities to study environmental factors as determinants of body height, because the standard of living has increased rapidly and steadily during this century.[16] Comparatively low parent–child correlations have been found for birth cohorts from 1953 through 1968,[17] suggesting that environmental factors may have contributed to body height even after World War II.

The purpose of this study was to examine the level of and change in the heritability of body height across the birth cohorts born in Finland during the first half of this century. It was expected that heritability would increase over the birth cohorts as the standard of living improved. It was also expected that the changes would be smaller among women than men because women have better resistance to adverse environmental influences.[18]

Methods

The data were derived from the Finnish Twin Cohort Study.[19] This data set consists of all Finnish twin pairs of the same sex born before 1958 with both co-twins alive in 1974. The first questionnaire was mailed to these twins in 1975, and a second questionnaire was mailed in 1981 (N = 33 534, response rate 85%).[20]

Body height was elicited with the question "How tall are you in centimeters?" The reliability of self-reported body height was studied in a subsample (n = 100 men and 127 women) by measuring body height after the subjects responded to a similar questionnaire in 1990.[21] The correlation between self-reported and measured body height was 0.98 among men and 0.96 among women.

Twin zygosity was determined by a very accurate validated questionnaire method.[22] Among pairs who replied to the 1981 questionnaire, zygosity could not be determined for 899 pairs, who were excluded; 52 pairs with no information on body height also were excluded. The final study group consisted of 3466 monozygotic and 7450 dizygotic twin pairs. The high dizygotic-to-monozygotic ratio reflects the high rate of dizygotic twins born in the first half of the 20th century in Finland.

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This brief was accepted September 23, 1999.

In a quantitative genetic study, the phenotype is assumed to result from 4 factors: an additive genetic component (A), the genetic effects caused by dominance (D), the effects of the shared environment (C), and the effects of the unshared environment (E).[23] Because monozygotic and dizygotic twin pairs differ in their degree of genetic relatedness, this information was used to estimate components of variance due to the aforementioned factors. Four models were estimated: AE, CE, ACE, and ADE. Chi-square statistics were used to evaluate which model best fit the data.

The best model was used to examine the change in the parameters of the model by birth cohort. Year of birth was divided into 4 broad cohorts: (1) those born in 1928 or earlier (i.e., before the Great Depression), (2) those born in 1929 through 1938, (3) those born in 1939 through 1946 (i.e., during World War II and the subsequent period of rationing), and (4) those born in 1947 through 1957 (i.e., the postwar years when the standard of living steadily increased and reached a relatively high level in Finland).

In the preliminary analysis (Table 1), intraclass correlations were used. Because of the dependence of twin pairs, the means between monozygotic and dizygotic pairs were analyzed with generalized estimating equations[24] by means of PROC GENMOD in SAS (SAS Institute, Inc, Cary, NC). The correlations and standard deviations calculated for the total population were age-adjusted with PROC REG. Differences in the variances between monozygotic and dizygotic twins were analyzed with the assumption of dependence, as suggested by Christian et al.,[25] by means of the TWINAN90 statistical package.[26] LISREL[23] and Mx[27] statistical packages were used in genetic modeling. In comparisons between sexes, mean and variance were standardized by sex and year of birth (5-year groups) with PROC STANDARD.

TABLE 1—Distribution, Mean, and Variance of Body Height; Intraclass Correlation, by Zygosity, Sex, and Birth Cohort; and Statistical Significance of Difference in Mean and Variance Between Monozygotic (MZ) and Dizygotic (DZ) Twins[a]

	No. of Pairs	Mean	Variance	Intraclass Correlation
Men				
1928 or earlier				
MZ	311	171.3	6.00	0.86
DZ	654	171.6	6.30	0.48
P		.396	.264	
1929–1938				
MZ	250	173.0	6.07	0.90
DZ	663	173.6	6.43	0.52
P		.279	.288	
1939–1946				
MZ	314	175.4	6.30	0.93
DZ	735	175.6	6.12	0.52
P		.868	.518	
1947–1957				
MZ	686	176.1	6.29	0.92
DZ	1540	176.6	6.29	0.52
P		.118	.976	
All				
MZ	1561	174.7	6.25[b]	0.91[b]
DZ	3592	175.0	6.29[b]	0.51[b]
P		.066	.283	
Women				
1928 or earlier				
MZ	433	158.7	5.54	0.79
DZ	910	159.5	5.79	0.48
P		.012	.224	
1929–1938				
MZ	267	161.1	5.48	0.86
DZ	610	161.3	5.61	0.48
P		.509	.900	
1939–1946				
MZ	355	162.2	5.28	0.88
DZ	732	162.8	5.55	0.48
P		.062	.652	
1947–1957				
MZ	850	163.0	5.25	0.90
DZ	1606	163.6	5.65	0.51
P		.004	.349	
All				
MZ	1905	161.7	5.52[b]	0.87[b]
DZ	3858	162.2	5.68[b]	0.50[b]
P		.0001	.214	

[a]Data were derived from Finnish Twin Cohort Study.
[b]Adjusted by age.

Results

The average body height increased steadily over the 4 broad birth cohorts born between 1900 and 1957 (Table 1). In pooled data (monozygotic and dizygotic twins), the average height was 171.4 cm among men and 159.2 cm among women in the cohort born in 1928 or earlier. In the youngest cohort, born in 1947 through 1957, the average height was 176.6 cm and 163.5 cm, respectively. The age distribution was similar for monozygotic and dizygotic twins.

The comparison of different genetic models indicated that the fit of the AE model was poor (χ^2 = 30.7 for men and 31.8 for women) (data not shown). The fit of the CE model also was poor (χ^2 = 1109.1 for men and 1017.5 for women). The fit of the ACE model was good among men (χ^2 = 3.7) and fairly good among women (χ^2 = 8.4). However, in the ADE model, the estimate of the genetic effect caused by dominance was very close to zero, and the χ^2 value of the model was similar to that of the AE model. Subsequent analyses used the ACE model.

Table 2 presents the parameters of the ACE model matched to the data by birth cohort. The fit of the model was lowest in the oldest birth cohort but still reasonably good (χ^2 = 9.4 for men and 15.4 for women; *P* = .024 and .002, respectively). In the younger cohorts, the fit of the model was better, with χ^2 values varying between 2.1 and 6.1.

Among men, the relative effect of genetic factors increased from the oldest cohort born in 1928 or before (0.76; 95% confidence interval [CI] = 0.65, 0.88) to the cohort born in 1939 through 1946 (0.82; 95% CI = 0.72, 0.92) but remained at a similar level in the youngest cohort, those born in 1947 through 1957 (0.81; 95% CI = 0.73, 0.87). Among women, the ratio increased steadily from the oldest cohort (0.66; 95% CI = 0.55, 0.77) to the youngest cohort (0.82; 95% CI = 0.75, 0.89). In the entire data set, heritability was 0.77 (95% CI = 0.73, 0.82) among men and 0.76 (95% CI = 0.71, 0.80) among women.

TABLE 2—Path Coefficients and Standard Errors of Parameter, Heritability Estimates, and Statistical Significance of the ACE Model, by Birth Cohort and Sex[a]

	A	C	E	h^2	χ^2	*df*	*P*
Men							
1928 or earlier	5.417 (0.2109)	2.058 (0.5560)	2.282 (0.0915)	0.76	9.4	3	.024
1929–1938	5.700 (0.2009)	2.023 (0.5764)	1.965 (0.0880)	0.80	2.1	3	.554
1939–1946	5.526 (0.1750)	1.983 (0.5083)	1.711 (0.0683)	0.82	3.4	3	.341
1947–1957	5.643 (0.1250)	2.022 (0.3617)	1.807 (0.0488)	0.81	2.2	3	.689
Parameter fixed (model: men)					1171.6	21	<.0001
Women							
1928 or earlier	4.639 (0.1955)	2.168 (0.3908)	2.556 (0.0866)	0.66	15.4	3	.002
1929–1938	4.859 (0.2034)	1.795 (0.5366)	2.100 (0.0907)	0.76	3.6	3	.314
1939–1946	4.909 (0.1725)	1.430 (0.5837)	1.891 (0.0709)	0.81	5.5	3	.139
1947–1957	4.990 (0.1107)	1.629 (0.3453)	1.709 (0.0414)	0.82	6.1	3	.109
Parameter fixed (model: women)					1190.5	21	<.0001
Parameter fixed (model: men and women)[b]					1035.8	36	<.0001

Note. $h^2 = A^2/(A^2 + C^2 + E^2)$. A^2 = additive genetic variance; C^2 = shared environmental variance; E^2 = unshared environmental variance.
[a]Data were derived from Finnish Twin Cohort Study.
[b]In this model, mean and variance parameters were standardized.

The statistical significance of the differences between birth cohorts was studied by fixing the parameter in the 4 birth cohorts to similar values. The fit of the model was poor (χ^2_{21} = 1171.6 for men and 1190.5 for women), suggesting a statistically significant change in the parameters over the birth cohorts. The similarity of change in the parameters between men and women was studied by fixing parameters among men and among women to similar values. The fit of the model also was poor (χ^2_{36} = 1035.8), showing a statistically significant sex difference.

Discussion

A secular trend of increasing body height was found for Finnish twins. This finding probably reflects the continuous improvement in the standard of living over the 20th century, including the years during World War II. The results are consistent with the increase in the gross national product in Finland.[16]

As expected, not only the genetic factors but also the shared environmental factors contributed to the similarity of relatives' body height. This finding is shown by the fact that only the ACE model fits well to the data. As further expected, the relative effect of genetic factors was stronger in the younger birth cohorts. However, this trend leveled off in the cohort born from 1939 through 1946. This finding was unexpected because the increasing secular trend of body height continued in the youngest cohort born in 1947 through 1957.

Environmental determinants were relatively more important for body height among women than among men. The relative effect of genetic factors was lowest in the oldest female cohort but similar to that in men in the younger cohorts. This finding also was unexpected because women's growth is considered to be more resistant to environmental influences than that of men.[18] The estimated heritability in the entire data set approaches that found by Pearson and Lee[8] and Stunkard et al.[10]

A potential source of bias is assortative mating found in previous studies.[28,29] A high regional mobility in the later cohorts may have increased assortative mating, because opportunities to find a partner were better. This bias should weaken the estimated heritability, because it overestimates the effect of environmental factors by strengthening the correlation within dizygotic twins. Thus, the bias is not likely to explain the stronger heritability coefficients in the younger cohorts.

Another potential bias may result from the interaction between genetic and environmental factors or from the lack of identical environments between monozygotic and dizygotic twins.[23] Mothers may have fed undersized babies more frequently than other babies. It is also possible, but unlikely, that monozygotic twin children were fed more similar food than were dizygotic twins.

A special problem in cross-sectional studies is selective mortality in older birth cohorts. According to previous studies, mortality is selective by height.[30,31] This is an obvious bias but unlikely to explain the results. Dissimilarity in body height within twin pairs suggests an environmental effect. Shorter twins should have a higher mortality risk, and correlations in the oldest cohorts should consequently be stronger, which was not the case. This kind of bias has a conservative effect.

This study showed that changing environmental factors affect the heritability of height. The heritability is higher when the standard of living is better. Thus, the value of body height as an indicator of childhood living conditions varies in time and probably between communities. The observed effect is relatively slight but useful to consider in studies of body height. □

Contributors

K. Silventoinen planned the study design, analyzed the data, and drafted the paper. J. Kaprio and M. Koskenvuo collected the data. J. Kaprio and E. Lahelma contributed to the planning and writing of the paper. J. Kaprio assisted in the statistical analyses. All authors contributed to the final draft.

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