View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by DSpace at VU

Genes, Brain and Behavior (2006) **5:** 585–595

Copyright © Blackwell Munksgaard 2006

Genetic contributions to the association between height and intelligence: evidence from Dutch twin data from childhood to middle age

K. Silventoinen[*,†,‡], D. Posthuma[‡], T. van Beijsterveldt[‡], M. Bartels[‡] and D. I. Boomsma[‡]

[†]Department of Public Health, University of Helsinki, Helsinki, Finland, and [‡]Department of Biological Psychology, Free University of Amsterdam, Amsterdam, the Netherlands

[*]Corresponding Author: K. Silventoinen, PhD, Department of Public Health, University of Helsinki, PO Box 41, Mannerheimintie 172, FIN-00014, Helsinki, Finland. E-mail: karri.silventoinen@helsinki.fi

A positive association between intelligence (IQ) and height has been reported previously. It is generally assumed that this association reflects the effect of childhood environment on IQ, but there is still little research supporting directly this hypothesis. We studied the association between height and IQ in 209 Dutch twin pairs at the ages of 5, 7, 10 and 12 years, 208 twin pairs at 16 and 18 years of age and 567 twin pairs and their siblings in adulthood. The heritability of height was high in all cohorts and across all ages (a^2 = 0.93 – 0.96). In adulthood, heritability was also high for full-scale IQ (FSIQ: a^2 = 0.83–0.84) and somewhat lower for verbal IQ (VIQ: a^2 = 0.66–0.84). In early childhood, the heritability was lower, and common environmental factors had a substantial effect on FSIQ and VIQ. A positive association of height and IQ was found in early childhood and adolescence. In adulthood, a correlation was found between height and FSIQ in young adulthood and between height and VIQ in middle age. All correlations could be ascribed to genetic factors influencing both height and IQ. Thus, these results show that the association between height and IQ should not be directly regarded as evidence for childhood living conditions affecting IQ, but the effect of genetic factors affecting independently or interacting with environmental factors should be considered as well.

Keywords: Full-scale IQ, genetics, height, twins, Verbal IQ

Received 19 July 2005, revised 30 December 2005, accepted for publication 1 January 2006

A positive association between height and intelligence (IQ) has been found both in childhood (Wheeler *et al*. 2004) and in adulthood (Humphreys *et al*. 1985; Teasdale *et al*. 1989; Tuvemo *et al*. 1999), including the extremes of height distribution (Teasdale *et al*. 1991). Longitudinal studies have also found that growth velocity is associated with IQ. A large Swedish conscription study found that men who were small at birth but later reached normal adult height had higher IQ than those who were small at birth and short in adulthood (Lundgren *et al*. 2001). Two UK studies revealed that high growth velocity between the ages of 2 and 4 and after age 15 (Richards *et al*. 2002) as well as between the ages of 9 and 13 (Pearce *et al*. 2005) was associated with higher IQ. Short height in middle age has also been found to predict IQ decline in old age (Abbott *et al*. 1998). Thus, short stature seems to be consistently associated with lower IQ over one's life span.

The reason for the association between height and IQ is poorly understood. The most widely accepted explanation is that common environmental factors affect both height and IQ during childhood, and thus adult height can be interpreted as an indicator of childhood living conditions. This is a plausible explanation, because, for example, previous studies have suggested that vitamin and mineral deficiency affect negatively both children's growth (Allen 1994) and IQ (Eysenck & Schoenthaler 1997). The role of environmental factors is also supported by Danish results. Teasdale *et al*. (1989) reported that the association between IQ and height, measured during early adulthood, decreased over birth cohorts after the Second World War. Because the standard of living has increased in Denmark over the postwar period, it is possible that this diminishing association between height and IQ is due to a decrease in the effect of environmental factors.

The possibility that environmental factors are the reasons for the association between height and IQ, however, seems to be in contrast to twin and adoption studies on IQ and height, which find high heritability for both traits. In adulthood, over half of the IQ variation is due to genetic differences between individuals (Plomin & Spinath 2004). For height, the heritability estimates are even higher (80–90% in Caucasian populations: Silventoinen *et al*. 2003b). Thus, the possibility that common genetic factors could cause the height–IQ association should be considered as well. Genetic mediation has been found for brain volume and IQ (Posthuma *et al*. 2002) and for brain volume and height (Posthuma *et al*. 2000a). Thus, there may be genes which affect both body growth and central nervous system

doi: 10.1111/j.1601-183X.2006.00208.x

development, for example, through endocrinological pathways. One candidate is the growth hormone, as the clinical syndrome of growth hormone deficiency is associated with both short stature and mental retardation (van Dam *et al.* 2005). Even so, it is unknown whether there is an association between cognitive development and the secretion of the growth hormone within the normal variation of growth hormone levels.

The main objective of this study is to analyze the associations between IQ assessed by psychometric IQ tests, height during childhood and in adulthood, and growth in Dutch twins and their siblings. The subjects participated in four independent studies on the genetics of cognitive abilities, and their ages ranged from 5 to 71 years. This study allows the decomposition of the common variation between height and IQ into a variation caused by genetic differences and differences in environmental factors between individuals. Due to the age differences between the subjects who participated in the four studies, we can explore the etiology of the association between height and IQ as a function of age.

Materials and methods

Subjects were recruited from the Netherlands Twin Registry (Boomsma *et al.* 2002b), and they participated in ongoing projects on the genetics of cognition. The data include twins who were monozygotic (MZ), same-sex dizygotic (SSDZ) and opposite-sex dizygotic (OSDZ), as well as non-twin siblings. Zygosity was determined using DNA markers. Non-twin siblings were included if available, because the information provided by additional siblings increases the power to detect additive genetic and common environmental effects, as demonstrated previously (Posthuma & Boomsma 2000). Six IQ tests were used: the Revised Amsterdamse Kinder Intelligentie Test (RAKIT) (Bleichrodt *et al.* 1984), the Dutch version of the Wechsler Intelligence Scale for Children (WISC-R) (van Haasen *et al.* 1986), the Raven intelligence test (Raven 1985), the Dutch adaptation of the Wechsler Adult Intelligence Scale (WAIS) (Stinissen *et al.* 1970), a later version of the WAIS, i.e. WAIS-3 intelligence test (WAIS-III 1997), as well as the vocabulary subtest of the Groninger Intelligence Test (GIT) (Snijders *et al.* 1983). We used full-scale IQ (FSIQ) and verbal IQ (VIQ). FSIQ describes general IQ as a summary score of IQ subscales. To test the robustness of the results, we repeated the analyses using VIQ. VIQ was selected, because we assumed that environmental factors, for example education, may have had a stronger effect on VIQ than FSIQ. Additionally, in a large number of subjects, an additional verbal IQ test was available. Four study cohorts – children, adolescents, young adults and middle-aged participants – were participated in this study. The number of study subjects in each of these cohorts 1–4 is summarized in Table 1.

1 The youngest cohort was measured at the ages of 5, 7, 10 and 12 years old as detailed elsewhere (Bartels *et al.* 2002). The baseline cohort included 418 twin individuals. The RAKIT intelligence test was administrated at ages 5, 7, and 10, and the WISC-R intelligence test at age 12. Height was measured at ages 5 and 7 during the laboratory visit. At ages 10 and 12, height was reported by the mother.

2 The adolescent cohort was measured at ages 16 and 18 years and included 426 twin individuals. At age 16, the participants took the Raven and at age 18 the WAIS intelligence test as described in detail elsewhere (Rijsdijk *et al.* 1998, 2002). Height was measured at both ages during the laboratory visit.

3 The young adult cohort was part of a study of brain function and cognition (Posthuma *et al.* 2001a) and included 405 individuals. The mean age was 26.2 years (SD 4.14 years). The WAIS-3 intelligence test and the GIT vocabulary subtest were administered to 405 subjects. Height was measured during the laboratory visit. The young adult cohort included 103 twin individuals who had previously participated in the WAIS intelligence test at age 18, and thus this cohort partly overlaps with the adolescence cohort.

4 The middle-age adult cohort was part of the same project described under (3). The mean age in the middle-age cohort was 49.5 years (SD 7.14 years). The WAIS-3 intelligence test and the GIT vocabulary subtest were administered to 388 subjects. Additionally, 204 subjects who did not participate in

Table 1: Number of participants (twin individuals and siblings) and families in the study cohorts by sex and zygosity

	Children cohort				Adolescence cohort			Middle-age cohort	
	Age 5	Age 7	Age 10	Age 12	Age 16	Age 18	Young adult cohort	WAIS-3	GIT
Male MZ	82	74	50	56	77	76	62	60	103
Female MZ	94	84	62	68	99	89	64	84	133
Male DZ	129	120	85	88	120	93	76	54	121
Female DZ	113	106	85	90	124	116	94	98	145
Male non-twins	–	–	–	–	–	–	54	42	40
Female non-twins	–	–	–	–	–	–	55	50	50
Families	209	192	144	151	208	189	156	155	411

DZ, dizygotic; MZ, monozygotic.

the WAIS-3 intelligence test took the GIT vocabulary subtest as a part of a cardiovascular project described elsewhere (van den Berg *et al*. 2004; Snieder *et al*. 1999). The mean age of the 204 subjects who only provided data on the GIT vocabulary test was 44.2 years (SD 6.64 years). Thus, the GIT vocabulary test was available to 592 middle-aged subjects. Height was measured during the laboratory visit, both in the cardiovascular and in the brain function study.

The data were analyzed using quantitative genetic modeling based on linear structural equations. Separate analyses were conducted in each of the four cohorts. Quantitative genetic modeling of twin and sibling data allows the decomposition of phenotypic variation and covariation into additive genetic component (A), genetic dominance (D) including the interaction of alleles in the same locus (dominance) and the interaction between the alleles over all relevant loci (epistasis), common environment shared by siblings (C) and unique environment (E) specific to each sibling including measurement error (Boomsma *et al*. 2002a). Our design includes only twins and siblings reared together and does not allow modeling of genetic dominance and common environmental effects simultaneously. The raw data analysis option as available in the Mx statistical package (Neale 2003) was used. First, univariate models for height, FSIQ and VIQ were fitted to the data at each age and in each cohort separately. The best model, identified by comparing the χ^2-goodness-of-fit statistics, was then retained in the subsequent analyses.

Next, the associations between height and IQ were analyzed, using a triangular or Cholesky decomposition (Neale & Cardon 1992). Figure 1 describes the bivariate and trivariate Cholesky decompositions with additive genetic and unique environmental factors. The method assumes that specific factors affect each phenotype (A_1 and E_1–E_3 in the Fig. 1).

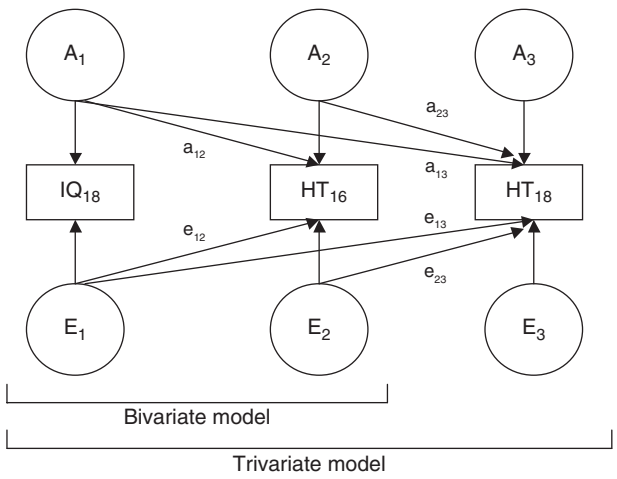

Figure 1: Bivariate and trivariate Cholesky decomposition of phenotypic variances and covariances. Latent factors include additive genetic (A1, A_2 and A_3) and unique environmental variance influences (E1, E_2 and E_3).

However, these factors can also affect the other phenotypes (e.g. a_{12}, a_{13} and a_{23} for genetic common paths). Thus, the Cholesky decomposition allows the phenotypic variation and covariation between height and IQ to be ascribed to common genetic and environmental factors affecting these traits. The Cholesky decomposition is an approach to obtain genetic and environmental correlations between phenotypes. In this study, we present the Cholesky results as genetic and environmental correlations between the phenotypes by standardizing the covariances by the variance components. For example, to calculate the genetic correlation in the bivariate case between IQ_{18} and HT_{16}, we divided the genetic covariance (a_{12}) by the genetic variation of these two phenotypes (the square root of the multiplication of the effect of A_1 on IQ_{18} and A_2 on HT_{16}). Similarly, it is possible to compute common and specific environmental correlations.

We first analyzed the data on height and FSIQ and VIQ. Next, we analyzed in the adolescent cohort whether IQ at age 18 was correlated with height at age 16 when the correlation between IQ and height at age 18 was adjusted for. A statistically significant correlation would suggest that delayed growth is associated with IQ independently of the height at early adulthood. Using the trivariate Cholesky decomposition, we explored whether this correlation is explained by correlated genetic or environmental factors.

In all analyses, we assumed no gene–environment interaction or correlation, random mating and equal environment for MZ twins, DZ twins and siblings. Previous studies have shown a clear tendency for assortative mating both for IQ (Reynolds *et al*. 1996) and height (Silventoinen *et al*. 2003a). However, in these studies including twins and their spouses, spousal correlations for IQ and height were found to be partly because of social homogamy, i.e. similarities in the background environments of spouses, which do not affect the heritability estimates. The extent to which spousal correlations reflect phenotypic assortment generates a correlation between spouses' genotypes and thus increases the genetic correlations within DZ twins and ordinary siblings above 0.5 as assumed in the used quantitative genetic models, i.e. they share on average more than 50% of their segregating genes. This would inflate the estimates of common environmental variance, which thus may be overestimated in this study. The effect of possible gene–environment interaction is estimated as a part of additive genetic component (Purcell 2002). Thus, the additive genetic component affecting height and IQ may not be solely because of genetic factors but can partly reflect genetic differences in susceptibility to environmental conditions. Finally, passive or active gene–environment correlation may exist. Passive gene–environment correlation exists if, for example, genetically tall or intelligent children tend to live in an environment supporting physical and intellectual growth. In active gene–environment correlation, taller children may create intellectually more simulating environment than shorter children. In the models we used, passive

gene–environment correlation inflates common environmental component and active gene–environment correlation-additive genetic component.

Results

Table 2 summarizes the means of height and the IQ tests in the four cohorts. As different IQ tests were used and not all tests were standardized, the results are not comparable over the cohorts. No systematic differences between MZ twins, DZ twins or non-twin siblings were found in height, VIQ or FSIQ (Posthuma *et al*. 2000b). A clear age effect was present in all of these traits. In the pooled adult data (the young adult cohort and the middle age cohort together), age was negatively correlated with height ($r = -0.10$, $P = 0.030$ in men and $r = -0.33$, $P < 0.001$ in women) and FSIQ ($r = -0.30$, $P < 0.001$ and $r = -0.42$, $P < 0.001$, respectively), whereas the correlation between age and VIQ was positive in men ($r = 0.14$, $P = 0.002$), and no correlation was found in women ($r = 0.04$, $P = 0.25$). The mean height was about the same for boys and girls until age 12 at which age girls were somewhat taller than boys. After age 12, boys grew more than girls, and at age 16, they were already taller than girls. As reported previously (Posthuma *et al*. 2003), in the adolescent and adult cohorts, men had, on average, somewhat higher FSIQ scores than women, whereas no sex difference was found in VIQ. In the children cohort, no systematic sex differences were found in FSIQ or VIQ. Additionally, we found that in the children cohorts, height was negatively correlated with the exact age of measurement. Due to the age and sex differences in mean height and IQ, for subsequent modeling, the effects of age and sex on the means were adjusted for independently in each cohort by including age and sex as covariates in the statistical

Table 2: Means and standard deviations of height and unstandardized IQ measures in the study samples by sex and zygosity*

	Men			Women		
	Height (cm)	Full-scale IQ	Verbal IQ	Height (cm)	Full-scale IQ	Verbal IQ
Children cohort (WISC)						
Age 5						
MZ twins	114 (4.1)	102 (13.6)	106 (13.5)	113 (4.3)	105 (13.5)	105 (14.3)
DZ twins	114 (4.7)	102 (12.9)	103 (12.9)	114 (4.3)	102 (12.9)	102 (13.3)
Age 7						
MZ twins	125 (4.7)	104 (13.4)	102 (13.5)	124 (4.9)	103 (15.7)	96 (14.2)
DZ twins	124 (5.5)	103 (15.4)	98 (14.9)	124 (4.4)	103 (14.0)	95 (13.4)
Age 10						
MZ twins	145 (5.8)	106 (14.9)	104 (13.1)	144 (7.0)	107 (18.1)	102 (16.9)
DZ twins	143 (6.3)	109 (14.1)	106 (13.5)	143 (6.3)	105 (15.3)	101 (15.6)
Age 12						
MZ twins	155 (7.4)	99 (12.6)	98 (13.1)	157 (7.4)	101 (13.8)	97 (14.3)
DZ twins	153 (7.6)	102 (13.1)	100 (12.6)	156 (6.9)	98 (12.9)	94 (11.6)
Adolescence cohort						
Age 16 (Raven)†						
MZ twins	176 (9.1)	49 (7.7)	–	167 (5.6)	48 (5.4)	–
DZ twins	177 (7.2)	50 (5.6)	–	169 (5.1)	51 (5.8)	–
Age 18 (WAIS)						
MZ twins	179 (7.8)	113 (13.2)	109 (14.5)	167 (5.7)	114 (10.8)	108 (12.4)
DZ twins	181 (6.6)	111 (12.1)	111 (11.3)	170 (5.1)	116 (11.0)	111 (10.8)
Young adult cohort† (WAIS-III – % correct per subtest, and GIT)						
MZ twins	182 (7.6)	75 (10.5)	14 (2.4)	166 (5.4)	13 (2.5)	13 (2.5)
DZ twins	181 (7.0)	73 (7.3)	14 (2.2)	166 (7.0)	13 (2.3)	13 (2.3)
Non-twins	181 (5.3)	74 (9.5)	13 (3.0)	166 (6.0)	14 (2.4)	14 (2.4)
Middle aged cohort†,‡ (WAIS-III – % correct per subtest, and GIT)						
MZ twins	181 (7.2)	68 (9.5)	14 (2.7)	165 (5.6)	63 (9.9)	14 (2.6)
DZ twins	180 (6.7)	66 (11.5)	15 (2.6)	166 (6.4)	61 (10.8)	14 (2.8)
Non-twins	181 (5.4)	68 (10.3)	15 (2.5)	166 (6.0)	61 (11.5)	14 (2.6)

DZ, dizygotic; MZ, monozygotic.

*Different intelligence tests were used; hence, results are not comparable over study cohorts.

†IQ scores are unstandardized scores representing the percentage correct per subtest (see Posthuma *et al*. 2001b).

‡Height among subjects who were phenotyped for the verbal intelligence test (GIT).

models. We also tested possible non-linearity in the effect of age but found that higher order polynoms of age did not correlate significantly with height, FSIQ or VIQ in any cohort when age as a linear variable was adjusted for.

Genetic modeling began by fitting the univariate models to height and to IQ data. As reported previously (Bartels *et al.* 2002; Posthuma *et al.* 2001a; Posthuma *et al.* 2001b), our assumptions were confirmed for MZ and DZ twins having equal environments as well as for the generalizability of the twin data to the singleton data. We also tested whether DZ twins and non-twin siblings share the same proportions of environmental variance. We found that in the two adult data sets, non-twin sibling correlations of FSIQ, VIQ or height did not differ significantly or systematically from DZ correlations suggesting that no differences in the shearing of environmental variation were present. The most parsimonious model was used in the subsequent genetic analyses for height, FSIQ and VIQ within each cohort, i.e. in children, adolescents and the two adult cohorts.

In the children cohort, the additive genetic/unique environment (AE) model fitted adequately for height at the ages of 5, 7, 10 and 12, and the dominance genetic or common environmental component could be excluded from the model without significantly worsening that fit. The AE model also offered an adequate fit for FSIQ and VIQ at the ages of 10 and 12. However, at ages 5 and 7, a common environmental component was statistically significant for FSIQ ($\Delta\chi^2_1 = 14.5$, $P < 0.001$ at age 5 and $\Delta\chi^2_1 = 3.73$, $P = 0.05$ at age 7) and VIQ ($\Delta\chi^2_1 = 7.55$, $p = 0.006$ and $\Delta\chi^2_1 = 11.24$, $p = 0.001$, respectively), and thus the additive genetic/common environment/unique environment (ACE) model was used. In the adolescent cohort, the AE model offered adequate fit for height and VIQ at 16 and 18 years. For FSIQ, the AE model gave the best fit at age 18, whereas at age 16, both AE and CE model offered an adequate fit. In the adult cohorts, the AE model offered the best fit for VIQ in the young adult and middle-age cohorts and for height and FSIQ in the middle-age cohort. In the young adult cohort, the additive genetic/dominance genetic/unique environment (ADE) model fitted statistically significantly better for height ($\Delta\chi^2_1 = 4.02$, $P = 0.05$) and FSIQ ($\Delta\chi^2_1 = 7.04$, $p = 0.008$) compared with the AE model. However, as the dominance effect was relatively small, we assumed that this inconsistency is rather due to a type 1 error than reflects a real difference in the genetic architecture of height and FSIQ between the cohorts. We therefore decided to use an AE model for these phenotypes in the young adult cohort as well. The AE model was also the best model if the two adult cohorts were analyzed together.

Thus, to summarize the results for the initial genetic modeling, the AE model was used in the subsequent modeling for height in all cohorts and for FSIQ and VIQ in the adolescent and adult cohorts. In the children cohort, the ACE model was used for measuring FSIQ and VIQ at ages 5 and 7 and the AE model for FSIQ and VIQ at ages 10 and 12. The comparisons of the fit of the models are described in detail in previous publications (Bartels *et al.* 2002; Posthuma *et al.* 2001a; Posthuma *et al.* 2001b).

Table 3 summarizes the proportions of the phenotypic variance of height and the IQ measures explained by the additive genetic and environmental factors in the best models. The heritability of height was high and similar in all cohorts and at all ages ($a^2 = 0.93 - 0.96$). For FSIQ and VIQ, the heritability estimates were lower in the children cohort at ages 5 and 7 than at older ages; this result is expected, because common environmental factors were important at these ages. Otherwise, the heritability estimates were high and varied from 0.61 (the adolescence cohort at age 16) to 0.84 (the young adult cohort) for FSIQ and from 0.61 (the middle age cohort) to 0.84 (the children cohort at age 12 and the adolescent cohort at age 18) for VIQ. Generally, the heritability estimates were somewhat higher for FSIQ than for VIQ.

Table 4 summarizes the results for bivariate analyses of height and IQ. Height was weakly associated with FSIQ in young childhood (ages 5 and 7) and more strongly in adolescence (ages 16 and 18) and young adulthood. No association between height and FSIQ was found in late childhood (age 10 and 12) or in middle age. For VIQ, the age pattern was similar to that found for FSIQ in childhood and adolescence: a statistically significant correlation was found in young childhood and adolescence but not in late childhood. In adulthood, the pattern was different than found for FSIQ, and the association was found in middle age but not in young adulthood. In all cohorts where we observed a statistically significant phenotypic correlation between height and IQ, the entire correlation was explained by the additive genetic correlation. The magnitude of the additive genetic correlations between height and FSIQ or VIQ varied from 0.16 to 0.65; the additive genetic correlations were about the same magnitude for FSIQ and VIQ. The highest genetic correlation was found for VIQ at age 7; this result was expected, because at this age, the relative effect of genetic factors on VIQ was much lower than in other ages, i.e. 0.08, and thus a larger genetic correlation was needed to explain the statistically significant phenotypic correlation.

We finally studied how IQ was associated with growth. In the children cohort, we did not find statistically significant correlations between growth and IQ, and thus we conducted the analyses only for the adolescence cohort. In this cohort, we studied whether height at age 16 was associated with FSIQ or VIQ at age 18 when height at age 18 was adjusted for. The correlation between height at age 16 and 18 was 0.99 in women and 0.92 in men. The mean difference between these two height measures was 3.4 cm among men and 0.6 cm among women. The partial correlation between height at age 16 and FSIQ at age 18 adjusted for height at age 18 was statistically significant among men ($r = 0.16$, $P = 0.04$) but not among women ($r = -0.09$, $P = 0.22$). For VIQ, the correlations were 0.16 ($P = 0.04$) and -0.08 ($P = 0.25$), respectively. Because of the large

Table 3: Proportion of variation of height, full-scale IQ and verbal IQ accounted for variation in additive genetic (a^2), common environmental (c^2) and unique environmental factors (e^2) in the best model with 95% confidence intervals by age and study cohort

	Children cohort				Adolescence cohort			
	Age 5	Age 7	Age 10	Age 12	Age 16	Age 18	Young adult cohort	Middle-age cohort
Height								
a^2	0.94 (0.91–0.96)	0.93 (0.90–0.95)	0.94 (0.91–0.96)	0.96 (0.94–0.97)	0.93 (0.90–0.95)	0.94 (0.92–0.96)	0.95 (0.93–0.97)	0.95 (0.93–0.96)
e^2	0.06 (0.04–0.09)	0.07 (0.05–0.10)	0.06 (0.04–0.09)	0.04 (0.03–0.06)	0.07 (0.05–0.10)	0.06 (0.04–0.08)	0.05 (0.03–0.07)	0.05 (0.04–0.07)
Full-scale IQ								
a^2	0.24 (0.01–0.49)	0.39 (0.06–0.72)	0.79 (0.69–0.85)	0.83 (0.76–0.88)	0.61 (0.49–0.70)	0.83 (0.75–0.87)	0.84 (0.76–0.89)	0.83 (0.75–0.88)
c^2	0.52 (0.29–0.71)	0.30 (0.00–0.55)	–	–	–	–	–	–
e^2	0.24 (0.17–0.33)	0.31 (0.23–0.44)	0.21 (0.15–0.31)	0.17 (0.12–0.24)	0.39 (0.30–0.51)	0.17 (0.13–0.25)	0.16 (0.11–0.24)	0.17 (0.12–0.25)
Verbal IQ								
a^2	0.37 (0.13–0.65)	0.08 (0.00–0.41)	0.77 (0.66–0.84)	0.84 (0.77–0.89)	–	0.84 (0.77–0.88)	0.66 (0.48–0.78)	0.61 (0.50–0.70)
c^2	0.39 (0.13–0.60)	0.52 (0.24–0.66)	–	–	–	–	–	–
e^2	0.23 (0.17–0.32)	0.40 (0.29–0.53)	0.23 (0.16–0.34)	0.16 (0.11–0.23)	–	0.16 (0.12–0.23)	0.34 (0.12–0.23)	0.39 (0.29–0.50)

Table 4: Trait correlations between height and the IQ measurements and the correlations between additive genetic factors affecting these traits (the best model) with 95% confidence intervals by age and study cohort*

	Trait correlation†		Additive genetic correlation†,‡	
	r	95% CI	r_A	95% CI
Correlation between height and full-scale IQ				
Children cohort				
Age 5	0.09	0.00–0.18	0.22	0.01–1.00
Age 7	0.11	0.01–0.21	0.21	0.03–0.56
Age 10	−0.03	−0.15–0.09	–	
Age 12	−0.01	−0.12–0.11	–	
Adolescence cohort				
Age 16	0.15	0.02–0.26	0.16	0.02–0.30
Age 18	0.22	0.12–0.31	0.23	0.10–0.35
Young adult cohort	0.22	0.13–0.31	0.18	0.06–0.30
Middle-age cohort	0.05	−0.05–0.15	–	
Correlation between height and verbal IQ				
Children cohort				
Age 5	0.13	0.03–0.22	0.21	0.04–0.46
Age 7	0.16	0.07–0.26	0.65	0.17–1.00
Age 10	0.03	−0.09–0.15	–	
Age 12	0.08	−0.04–0.19	–	
Adolescence cohort				
Age 18	0.23	0.13–0.32	0.26	0.13–0.38
Young adult cohort	0.08	−0.02–0.18	–	
Middle-age cohort	0.14	0.06–0.22	0.20	0.08–0.31

*Additive genetic correlations obtained from Cholesky decomposition.

†Correlations are adjusted for age and sex.

‡Correlation is presented only if statistically significant phenotypic correlation was found.

difference between men and women, we conducted trivariate analyses separately for men and women and presented the results only for men; for women, the results were statistically insignificant because of the near unit correlation between height at age 16 and at age 18.

Figure 2 shows the trivariate results for the associations between height at ages 16 and 18 and FSIQ at age 18 in men. The unique environmental correlations of FSIQ at age 18 with height at ages 16 or 18 were not statistically significant ($\Delta\chi^2_2 = 2.75$, $P = 0.25$) and were excluded from the final model. Both additive genetic ($r_A = 0.93$, 95% CI 0.90–0.96) and unique environmental components ($r_E = 0.75$, 95% CI 0.58–0.86) affecting height at ages 16 and 18 were strongly correlated. The additive genetic component affecting FSIQ at age 18 was statistically significantly correlated with both the additive genetic component affecting height at age 16 ($r_A = 0.26$, 95% CI 0.05–0.45) and 18 ($r_A = 0.29$ 95% CI 0.08–0.47). We conducted similar trivariate analyses for height at age 16 and 18 and VIQ at age 18 (data not shown). The results were very similar to the trivariate analyses for FSIQ. The unique environmental correlations of VIQ at age 18 with height at ages 16 or 18 were not significant ($\Delta\chi^2_2 = 4.43$, $P = 0.10$) and were thus excluded from the final model. In the final model, the additive genetic

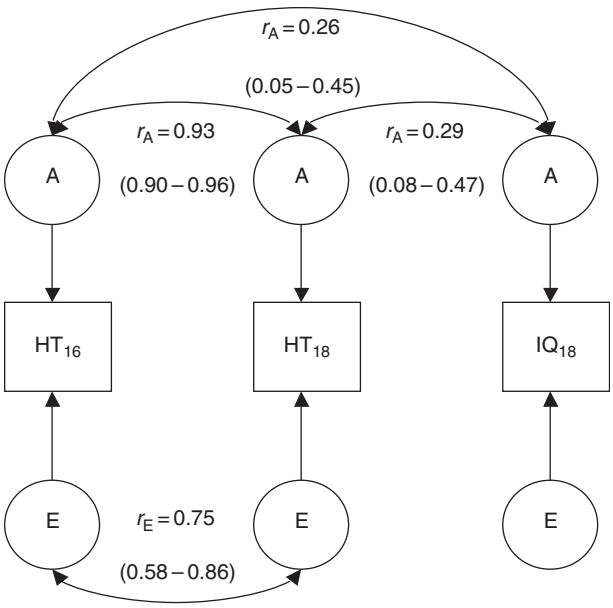

Figure 2: The final trivariate model for height at age 16, height at age 18, and Full Scale IQ (FSIQ) at age 18 in men (additive genetic and unique environmental correlations are obtained from the Cholesky decomposition).

component affecting VIQ at age 18 was significantly correlated with both the additive genetic component affecting height at ages 16 ($r_A = 0.26$, 95% CI 0.05–0.45) and 18 ($r_A = 0.29$ 95%, CI 0.08–0.48).

Discussion

Our results on the association between height and IQ in adulthood suggest that this phenotypic correlation is due to common genetic factors affecting these two traits. The genetic architecture of this association was similar regardless of whether FSIQ or VIQ was used. These results differ from those in the studies which have suggested that this association would be due to common environmental factors affecting height and IQ (Abbott *et al*. 1998; Teasdale *et al*. 1989). Yet, these studies have merely provided indirect evidence on the background of this association. In contrast, our current study, which exploited data on twins, was able to actually decompose the covariance of height and IQ into genetic and environmental components and thereby give more direct evidence for the mechanism behind this association. We also found that delayed growth at age 16 was associated with lower IQ at age 18 among men, and this association was caused by common genetic factors. One explanation is that IQ is associated with genes affecting the timing of the onset of puberty, but also other factors delaying growth in adolescence may lie behind this association. However, the genetic correlation was modest, i.e. 0.26, suggesting that 7% of the genetic variation is common to height at age 16 and IQ at age 18. Among girls, we could not study this question, because no or only very little growth happened between the ages of 16 and 18 years. In these Dutch-birth cohorts, the average age of the onset of puberty is almost 1 year lower in girls (10.72 years of age) than in boys (11.45 years of age), and thus at age 16, most of the girls had already gone through puberty (Fredriks *et al*. 2000).

Our results suggesting that the association between IQ and height is exclusively because of correlated genetic factors are in contrast to a previous Norwegian twin study based on a conscription register and thus including only same-sex males (Sundet & Tambs 2005). This study reported also phenotypic IQ–height correlation but found that it was mainly explained by correlated common environmental factors. One possible explanation for this inconsistency is that in the Norwegian study, the heritability estimates both for IQ and height were lower and the estimates for the effect of common environmental factors higher than in our study as well as in many previous studies on height (Silventoinen 2003; Silventoinen *et al*. 2003b) and IQ (Plomin & Spinath 2004). The Norwegian study covered two birth cohorts born 1915–1960 and 1967–1979. It is possible that especially in the older birth cohort, environmental factors may have had a stronger effect on both height and IQ than in our cohorts leading to lower heritability estimates. In a previous Finnish study, it was found that heritability estimates for height have increased from the beginning of the 20th century to the 1950s (Silventoinen *et al*. 2000). However, because cohort-specific results were not presented, this hypothesis cannot be verified. It is noteworthy that in the Norwegian study, mean IQ score and height were lower in MZ and DZ twins than in singletons. Because twin-singleton difference is not found in other studies in IQ (Posthuma *et al*. 2000b) or in height (Andrew *et al*. 2001), this difference is probably not because of biological reasons related to twinning. It may indicate stronger environmental stress in this population, which has especially affected twins, for example because of the extra burden caused by two same-age children in a family. Nevertheless, these different results suggest that the genetic architecture of the IQ–height correlation may vary between populations, for example because of differences in environmental stress.

The heritability estimates found in this study are among the highest reported for both height (Silventoinen 2003; Silventoinen *et al*. 2003b) and IQ (Plomin & Spinath 2004). We did not detect any differences in the heritability estimates for FSIQ, VIQ or height between the young adult and middle-age cohorts. For height, heritability estimates remained high from early childhood to middle age, and the common environmental factors showed no effect. This lack of notable differences in the heritability of IQ or height is interesting, because we found that both FSIQ and height showed a strong negative association with age in the two adult data sets. Increase in mean adult height (Silventoinen *et al*. 2001) and IQ, known as the Flynn effect (Flynn 1987), over birth cohorts during the 20th century is well documented. Our results do not support a previous hypothesis that this increase would be because of a decreasing effect of environmental factors on height and IQ. Unique environmental factors affecting height at ages 16 and 18 were strongly correlated. This environmental component can include external factors, such as smoking, but may also be caused by purely idiopathic factors affecting growth, for example differences in hormonal secretion, or epigenetic heritability (Wong *et al*. 2005). As reported previously (Bartels *et al*. 2002), common environmental factors in early childhood have a substantial effect on FSIQ, and the heritability estimates were lower than at later ages. We also found that the heritability of VIQ followed a rather similar age pattern, but the heritability estimates were somewhat lower than for FSIQ.

It is noteworthy that our study population represents an affluent and egalitarian society, even by the standards of industrialized nations. This is also reflected in this study by the high heritability of height, which shows that environmental factors have only a small effect on height differences within our study cohorts. It is possible that in populations which are living under more severe environmental stress, the background of the association between height and IQ is different. However, previous studies on the heritability of IQ have not provided evidence that common environmental

factors affect IQ in adulthood (Plomin & Spinath 2004). In childhood, the effect of common environmental factors is more substantial, but we did not find that environmental factors explained the association between height and IQ. Our results suggest that common genetic factors have an effect on the association between height and IQ, and thus this phenotypic association should not be interpreted as evidence on the effect of environmental factors on IQ. In most of the cohorts we studied, the genetic correlations varied between 0.20 and 0.26, indicating that 4–7% of the genetic variation is common to height and IQ. Even when this can be regarded as a modest shared variation, it still completely explained the phenotypic correlations between height and IQ. Interestingly, a previous study on the association between height and education, which utilized large twin cohorts from the USA and Finland, reported that this association was mainly because of common environmental factors and to a lesser extent because of unique environmental factors (Silventoinen *et al*. 2004). This difference between the genetic architectures of the association of height with education and IQ in ethnically homogeneous populations strongly suggests that results on education should not be generalized to IQ, even if these two phenotypes would ostensibly behave similarly.

When we analyzed the association between height and IQ, we discovered some age differences. Both VIQ and FSIQ were associated with height in early childhood, but these associations disappeared in late childhood and resurfaced during adolescence. In adulthood, we found the association between height and FSIQ only for young adults but not for those who were middle aged. Previous Danish results suggested that the correlation between height and IQ in early adulthood has decreased rather than increased over cohorts born after the Second World War (Teasdale *et al*. 1989), and thus this difference may be associated more with age than with the birth cohort. In contrast, we found an association between height and VIQ only in the middle-aged cohort but not in the young adult cohort.

The age differences we found are not easy to explain. It is possible that because VIQ represents a more crystallized form of IQ than FSIQ, the association between height and VIQ becomes stronger during aging. This may be because of an active gene–environment correlation, i.e. persons who have higher IQ during childhood generate an intellectually stimulating environment, which leads to increasing VIQ during aging. We found that VIQ was positively associated with age in men, and no association was found in women, whereas a clear negative association was found between age and FSIQ in both men and women. There is also some previous evidence that verbal and non-verbal cognitive abilities become more unidimensional during cognitive development. Increasing correlation between additive genetic factors affecting verbal and non-verbal IQ has been reported from infancy (Price *et al*. 2000) to childhood (Rietveld *et al*. 2003) and further to early adulthood (Rijsdijk *et al*. 2002). We tested this by computing sex-adjusted correlations between FSIQ and VIQ and found that this correlation was slightly higher in the middle-age cohort ($r = 0.63$) than in the young adult cohort ($r = 0.57$), but this difference was statistically insignificant ($P = 0.19$ in a two-tailed test). However, in general, the age differences were small and were associated with the magnitude of the phenotypic correlation between height and IQ rather than with the genetic architecture of this association. It is very possible that some of the statistically non-significant correlations occurred because of random variation, i.e. type 1 error. Further, it is noteworthy that different IQ tests were used at different ages. It is possible that these IQ tests cover slightly different aspects of IQ and thus have produced heterogeneity in our results in an unknown way. Heritability estimates for height and IQ are also only moderate, which can cause instability for the estimates of genetic correlations. Finally, at ages 10 and 12, height was reported by a mother and not measured such as at other ages. The genetic architecture of height at these ages is very similar than at other ages suggesting that there is not considerable bias in the maternal reports. However, it is possible that there are age differences between the measures of IQ and the maternal reports of height which may explain why IQ–height difference was not found at these ages. Thus, further studies are needed to confirm whether or not the age differences found in this study are due to real developmental differences.

One explanation for the association between height and IQ in our data is the variation in brain volume. Previous studies have shown a common genetic component behind brain volume and height (Posthuma *et al*. 2000a) and behind brain volume and IQ (Posthuma *et al*. 2002) in Dutch data. Thus, it is possible that a part of the common genetic component between height and IQ is common to brain volume as well. Nevertheless, the correlations between brain volume, IQ and height were not so high that they are likely to explain the correlation between height and IQ. The correlation between FSIQ and brain volume was only slightly higher ($r = 0.25$ for brain gray matter and 0.24 for brain white matter) than found in this study between FSIQ and height. It is also possible that assortative mating for height and IQ contributes to the results. Assortative mating for IQ (Reynolds *et al*. 1996) and height (Silventoinen *et al*. 2003a) has been demonstrated. Assortative mating tends to inflate DZ correlations, leading to an overestimation of common environmental factors and thus cannot directly explain the results in this study, because we did not find any evidence on the effect of common environment on height or IQ after early childhood. However, it is possible that if height and IQ are correlated in spouse selection, for example, if tall women tend to select more intelligent men, it may create a genetic correlation between height and IQ in their offspring. Further twin studies including information on height and IQ of twins and their spouses and/or parents are needed to study this question in detail.

Our results do not exclude the possibility that environmental factors could contribute to the association between height and IQ but instead suggest that if this is the case, environmental factors do not affect independently but rather interplay with genetic factors. It is possible that active gene–environment correlation may play a role, for example, if tall children generate intellectually more stimulating environments than short children. Moreover, gene–environment interaction may also exist. In this case, some persons are genetically more resistant to the effect of adverse environmental factors, and therefore their physical and mental development is not affected so much as those who are genetically more vulnerable in the presence of the same environmental conditions. In the genetic model used in this study, both active gene–environment correlation and gene–environment interaction inflates the effect of additive genetic factors (Purcell 2002), and thus a part of the effect of additive genetic component may in reality be because of the interplay between environmental and genetic factors. Further studies with direct measurements of environmental factors are needed to clarify this question.

In conclusion, our results suggest that the association between height and IQ is due to common genetic factors, whereas environmental factors do not contribute to this association, at least in this affluent and egalitarian Caucasian population. Thus, the association between height and IQ should not be regarded as evidence for the effect of childhood living conditions on intelligence, because genetic factors can also contribute to this association independently or in interaction with environmental factors.

References

Abbott, R.D., White, L.R., Ross, G.W., Petrovitch, H., Masaki, K.H., Snowdon, D.A. & Curb, J.D. (1998) Height as a marker of childhood development and late-life cognitive function: the Honolulu-Asia Aging Study. *Pediatrics* **102**, 602–609.

Allen, L.H. (1994) Nutritional influences on linear growth: a general review. *Eur J Clin Nutr* **48**, S75–S89.

Andrew, T., Hart, D.J., Sneider, H., de Lange, M., Spector, T.D. & McGregor, A.J. (2001) Are twins and singletons comparable? A study of disease-related and lifestyle characteristics in adult women. *Twin Res* **6**, 399–408.

Bartels, M., Rietveld, M.J.H., van Baal, G.C.M. & Boomsma, D.I. (2002) Genetic and environmental influences on the development of intelligence. *Behav Genet* **32**, 237–249.

van den Berg, S.M., Posthuma, D. & Boomsma, D.I. (2004) A longitudinal genetic study of vocabulary knowledge in adults. *Twin Res* **7**, 284–291.

Bleichrodt, N., Drenth, P.J.D., Zaal, J.N. & Resing, W.C.M. (1984) *Revisie Amsterdamse Kinder Intelligentie Test [Revised Amsterdam Child Intelligence Test]*. Swets and Zeitlinger B. V., Lisse, the Netherlands.

Boomsma, D., Busjahn, A. & Peltonen, L. (2002a) Classical twin studies and beyond. *Nat Rev Genet* **3**, 872–882.

Boomsma, D.I., Vink, J.M., van Beijsterveldt, T.C.E.M., de Geus, E.J.C., Beem, A.L., Mulder, E.J.C.M., Derks, E.M., Riese, H., Willemsen, G.A.H.M., Bartels, M., van der Berg, M. Kupper, N.H.M., Polderman, T.J.C., Posthuma, D., Rietveld, M.J.H., Stubbe, J.H., Knol, L.I. Stroet, T. & van Baal, G.C.M. (2002b) Netherlands Twin Register: a focus on longitudinal research. *Twin Res* **5**, 401–406.

van Dam, P.S., de Winter, C.F., de Vries, R., van der Grond, J., Drent, M.L., Lijffijt, M., Kenemans, J.L., Aleman, A., de Haan, E.H.F. & Koppeschaar, H.P.F. (2005) Childhood-onset growth hormone deficiency, cognitive function and brain N-acetylaspartate. *Psychoneuroendocrinology* **30**, 357–363.

Eysenck, H.J. & Schoenthaler, S.J. (1997) Raising IQ level by vitamin and mineral supplementation. In Sternberg, R.J. & Grigorenko, E. (eds), *Intelligence, Heredity, and Environment*. Cambridge University Press, Cambridge, pp. 363–392.

Flynn, J.R. (1987) Massive IQ gains in 14 nations: what IQ tests really measure. *Psychol Bull* **101**, 171–191.

Fredriks, A.M., van Buuren, S., Burgmeijer, R.J.F., Meulmeester, J.F., Beuker, R.J., Brugman, E., Roede, M.J., Verloove-Vanhorick, S.P. & Wit, J.-M. (2000) Continuing positive secular growth change in the Netherlands 1955–97. *Pediatr Res* **47**, 316–323.

van Haasen, P.P., de Bruyn, E.E.J., Pijl, Y.J., Poortinga, Y.H., Lutje-Spelberg, H.C., Vander Steene, G., Coetsier, P., Spoelders-Claes, R. & Stinissen, J. (1986) *Wechsler Intelligence Scale for Children – Revised, Dutch Version*. Swets & Zetlinger B. V., Lisse, the Netherlands.

Humphreys, L.G., Davey, T.C. & Park, R.K. (1985) Longitudinal correlation analysis of standing height and intelligence. *Child Dev* **56**, 1465–1478.

Lundgren, E.M., Cnattingius, S., Jonsson, B. & Tuvemo, T. (2001) Intellectual and psychological performance on males born small for gestational age with and without catch-up growth. *Pediatr Res* **50**, 91–96.

Neale, M.C. & Cardon, L.R. (1992) *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic Publishers, Dordrecht.

Neale, M.C. (2003) *Mx: Statistical Modeling*, 2nd edn. Box 710 MCV. Department of Psychiatry, Richmond, VA, USA.

Pearce, M.S., Deary, I.J., Young, A.H. & Parker, L. (2005) Growth in early life and childhood IQ at age 11 years: the Newcastle Thousand Families Study. *Int J Epidemiol* **34**, 673–677.

Plomin, R. & Spinath, F.M. (2004) Intelligence: genetics, genes, and genomics. *J Pers Soc Psychol* **86**, 112–129.

Posthuma, D. & Boomsma, D.I. (2000) A note on the statistical power in extended twin designs. *Behav Genet* **30**, 147–158.

Posthuma, D., de Geus, E.J., Neale, M.C., Hulshoff Pol, H.E., Baare, W.E.C., Kahn, R.S. & Boomsma, D. (2000a) Multivariate genetic analysis of brain structure in an extended twin design. *Behav Genet* **30**, 311–319.

Posthuma, D., de Geus, E.J.C., Bleichrodt, N. & Boomsma, D.I. (2000b) Twin-singleton differences in intelligence. *Twin Res* **3**, 83–87.

Posthuma, D., de Geus, E.J.C. & Boomsma, D. (2001a) Perceptual speed and IQ are associated through common genetic factors. *Behav Genet* **31**, 593–602.

Posthuma, D., Neale, M.C., Boomsma, D.I. & de Geus, E.J.C. (2001b) Are smarter brains running faster? Heritability of alpha peak frequency, IQ and their interrelation. *Behav Genet* **31**, 567–579.

Posthuma, D., de Geus, E.J., Baare, W.F., Hulshoff Pol, H.E., Kahn, R.S. & Boomsma, D.I. (2002) The association between brain volume and intelligence is of genetic origin. *Nat Neurosci* **5**, 83–84.

Posthuma, D., Baare, W.F., Hulshoff Pol, H.E., Kahn, R.S., Boomsma, D.I. & de Geus, E.J. (2003) Genetic correlations between brain Volumes and the WAIS-III dimensions of verbal

comprehension, working memory, perceptual organization, and processing speed. *Twin Res* **6**, 131–139.
Price, T.S., Eley, T.C., Dale, P.S., Stevenson, J., Saudino, K. & Plomin, R. (2000) Genetic and environmental covariation between verbal and nonverbal cognitive development in infancy. *Child Dev* **71**, 948–959.
Purcell, S. (2002) Variance components models for gene–environment interaction in twin analysis. *Twin Res* **5**, 554–571.
Raven, J.C. (1985) *Standard Progressive Matrices: Sets A, B, C, D and E*. University Printing House, London, M.K. Lewis & Co, Cambridge.
Reynolds, C.A., Baker, L.A. & Pedersen, N.L. (1996) Models of spouse similarity: applications to fluid ability measured by twins and their spouses. *Behav Genet* **26**, 73–88.
Richards, M., Hardy, R., Kuh, D. & Wadsworth, M.E.J. (2002) Birth weight, postnatal growth and cognitive function in a national UK birth cohort. *Int J Epidemiol* **31**, 342–348.
Rietveld, M.J.H., Dolan, C.V., van Baal, G.C.M. & Boomsma, D.I. (2003) A twin study of differentiation of cognitive abilities in childhood. *Behav Genet* **33**, 367–381.
Rijsdijk, F.V., Vernon, P.A. & Boomsma, D.I. (1998) The genetic basis of the relation between speed-of-information-processing and IQ. *Behav Brain Res* **95**, 77–84.
Rijsdijk, F.V., Vernon, P.A. & Boomsma, D.I. (2002) Application of hierarchical genetic models to Raven and WAIS subtests: a Dutch twin study. *Behav Genet* **32**, 199–210.
Silventoinen, K. (2003) Determinants of variation in adult body height. *J Biosoc Sci* **35**, 263–285.
Silventoinen, K., Kaprio, J., Lahelma, E. & Koskenvuo, M. (2000) Relative effect of genetic and environmental factors on body height: differences across birth cohorts among Finnish men and women. *Am J Public Health* **90**, 627–630.
Silventoinen, K., Lahelma, E., Lundberg, O. & Rahkonen, O. (2001) Body height, birth cohort and social background in Finland and Sweden. *Eur J Public Health* **11**, 124–129.
Silventoinen, K., Kaprio, J., Lahelma, E., Viken, R.J. & Rose, R.J. (2003a) Assortative mating by body height and BMI: Finnish twins and their spouses. *Am J Hum Biol* **15**, 620–627.
Silventoinen, K., Sammalisto, S., Perola, M., Boomsma, D.I., Cornes, B.K., Davis, C., Dunkel, L., de Lange, M., Harris, J.R., Hjelmborg, J.V.B., Luciano, M., Martin, N.G., Mortensen, J., Nisticò, L., Pedersen, N.L., Skytthe, A., Spector, T.A., Stazi, M.A., Willemsen, G. & Kaprio, J. (2003b) Heritability of adult body height: a comparative study of twin cohorts in eight countries. *Twin Res* **6**, 399–408.
Silventoinen, K., Krueger, R.F., Bouchard, T.J.Jr, Kaprio, J. & McGue, M. (2004) Heritability of body height and educational attainment in an international context: comparison of adult twins in Minnesota and Finland. *Am J Hum Biol* **16**, 544–555.
Snieder, H., Boomsma, D.I., van Doornen, L.J. & Neale, M.C. (1999) Bivariate genetic analysis of fasting insulin and glucose levels. *Genet Epidemiol* **16**, 426–446.
Snijders, J.T., Luteijn, F. & Verhage, F. (1983) *Groninger Intelligence Test (GIT)*. Swets Test Publishers, Lisse, The Netherlands.
Stinissen, J., Willems, P.J. & Coetsier, P. & Hulsman, W.L.L. (1970) *Manual for the Dutch translated and adapted version of the Wechsler Adult Intelligence Scale (WAIS)*. Swets & Zeitlinger B.V., Lisse, the Netherlands.
Sundet, J.M. & Tambs, K. (2005) Resolving the genetic and environmental sources of the correlation between height and intelligence: a study of nearly 2600 Norwegian male twin pairs. *Twin Res Hum Genet* **8**, 307–311.
Teasdale, T.W., Sørensen, T.I.A. & Owen, D.R. (1989) Fall in association of height with intelligence and educational level. *BMJ* **298**, 1292–1293.
Teasdale, T.W., Owen, D.R. & Sørensen, T.I.A. (1991) Intelligence and educational level in adult males at the extremes of stature. *Hum Biol* **63**, 19–30.
Tuvemo, T., Jonsson, B. & Persson, I. (1999) Intellectual and physical performance and morbidity in relation to height in a cohort of 18-year-old Swedish conscripts. *Horm Res* **52**, 186–191.
WAIS-III. (1997) *Dutch Version: Manual*. Swets and Zeitlinger, Lisse.
Wheeler, P.G., Bresnahan, K., Shephard, B.A., Lau, J. & Balk, E.M. (2004) Short stature and functional impairment: a systematic review. *Arch Pediatr Adolesc Med* **158**, 236–243.
Wong, A.H.C., Gottesman, I.I. & Petronis, A. (2005) Phenotypic differences in genetically identical organisms: the epigenetic perspective. *Hum Mol Genet* **14**, R11–R18.

Acknowledgments

This study was supported by the European Commission under the programme 'Quality of Life and Management of the Living Resources' of 5th Framework Programme (no. QLG2-CT-2002–01254), by the Human Frontiers of Science Program (HFSP) (D.I.B rg0154/1998-B) and by the Universitair Stimulerings Fonds (USF) (D.I.B 96/22). K. Silventoinen was additionally supported by the Academy of Finland (grants 204694 and 108297).