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Heritability of Verbal and Performance Intelligence in a Pediatric Longitudinal Sample

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The longitudinal stability of IQ is well-documented as is its increasing heritability with age. In a longitudinal twin study, we addressed the question to what extent heritability and stability differ for full scale (FSIQ), verbal (VIQ), and performance IQ (PIQ) in childhood (age 9–11 years), and early adolescence (age 12–14 years). Genetic and environmental influences and correlations over time were evaluated in an extended twin design, including Dutch twins and their siblings. Intelligence was measured by the Wechsler Intelligence Scale for children — Third version (WISC III). Heritability in childhood was 34% for FSIQ, 37% for VIQ, and 64% for PIQ, and increased up to 65%, 51%, and 72% in early adolescence. The influence of common environment decreased between childhood and early adolescence from explaining 43% of the phenotypic variance for FSIQ to 18% and from 42% for VIQ to 26%. For PIQ common environmental influences did not play a role, either in childhood or in early adolescence. The stability in FSIQ and VIQ across the 3-year interval (r_p) was .72 for both measures and was explained by genetic and common environmental correlations across time (FSIQ, r_g = .96, r_c = 1.0; VIQ, r_g =.78, r_c = 1.0). Stability of PIQ (r_p =.56) was lower and was explained by genetic influences (r_g = .90). These results confirm the robust findings of increased heritability of general cognitive abilities during the transition from childhood to adolescence. Interestingly, results for PIQ differ from those for FSIQ and VIQ, in that no significant contribution of environment shared by siblings from the same family was detected.

■ **Keywords:** intelligence quotient (IQ), verbal IQ, performance IQ, childhood, adolescence, longitudinal model, development

General cognitive ability, or intelligence, is one of the most studied domains in the fields of psychology and behavior genetics. Twin and adoption studies have explored the genetic and environmental influences on general cognitive abilities at different ages. An intriguing aspect of the etiology of general cognitive ability is the increase in genetic influences with increasing age, while common environment influences decrease (Deary et al., 2009; Davis et al., 2009; Haworth et al., 2010). In early childhood, genetic influences explain around 20–30% of the total variance of general cognitive ability. In middle childhood the relative importance of genetic influences increases up to around 40–50% (Bartels et al., 2002; Davis et al., 2008; Bishop et al., 2003). This increase in the relative influence of genetic factors continues and results in heritability estimates of 70% in young adolescence (Bartels et al., 2002), and 70–80% in adulthood (Bartels et al., 2002; Posthuma et al., 2001; Rijsdijk et al., 2002).

Psychometric IQ is remarkably stable across the lifespan (Deary et al., 2000; Livingston et al., 2003; Bartels et al., 2002; Hoekstra et al., 2007; Lyons et al., 2009). Longitudinal twin studies have generally found that genetic influences explain this stability (Davis et al., 2008; Bartels et al., 2002; Davis et al., 2009; Lyons et al., 2009; Boomsma & van Baal, 1998), that is, variation in IQ is

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explained by the same genetic factors at different ages, although in childhood there also is a contribution of shared environment (Bartels et al., 2002; Boomsma & van Baal, 1998; Davis et al., 2009) to stability.

General cognitive abilities are often assessed by extracting a first unrotated principal component from a series of test batteries (the g-factor), or by an intelligence quotient measure from a standardized intelligence test (i.e., full scale IQ; FSIQ). Usually these measures combine verbal and non-verbal tests. When verbal (VIQ) and non-verbal (performance IQ; PIQ) were analyzed separately in a longitudinal twin study of 5 to 18 year olds, some differences in genetic architecture emerged (Hoekstra et al., 2007). An increase in heritability was found for both scales. For VIQ, 28% of the variance was explained by common environment at the age of 5 years and this decreased to 6% at age 12. However, common environmental influences did not contribute to the variance of PIQ at any age. Stability in PIQ was entirely explained by genetic influences, while stability in VIQ was influenced by both genetic and common environmental factors (Hoekstra et al., 2007).

In the present longitudinal study 9-year-old twin pairs and their full siblings were assessed on general cognitive ability by standardized IQ tests. When the twins were 12 years old, they and their siblings returned at the follow-up. With this extended, longitudinal twin design an increase in power to detect sources of variance due to genetic and common environment influences was realized (Posthuma & Boomsma, 2000). In addition it allowed an examination of the causes of stability in full scale IQ, verbal and performance IQ.

Materials and Methods

Subjects

Participants were recruited from the Netherlands Twin Register (NTR; Boomsma et al., 2006). They took part in an ongoing longitudinal study into the development of cognition and brain maturation (van Leeuwen et al., 2008; Peper et al., 2008). Families were invited for participation around the 9th birthday of the twins. Exclusion criteria consisted of chronic use of medication, any known major medical or psychiatric history, or participation in special education. In total 112 families were included and one older sibling with a maximum age of 14 years was also invited to participate ($N = 103$). Twins and sibs were invited for a follow-up study around the 12th birthday of the twins. In total 89 families participated at follow-up, including 83 siblings. Table 1 provides their mean age and *SD*.

Zygosity of same-sex twin pairs was determined by DNA polymorphisms. The twin sample at baseline and follow-up consisted of 23/20 monozygotic male twin pairs (MZM), 25/20 monozygotic female twin pairs (MZF), 23/17 dizygotic male twin pairs (DZM), 21/17 dizygotic female twin pairs (DZF), and 20/15 dizygotic twin pairs of opposite sex (DOS).

At baseline there were 46 siblings who were close to the 9-year-old twins in age (mean age was 10.9 years, with a range of 9.9 up to 11.5 years, 20 girls and 26 boys). Their data were analyzed with the baseline data of the twins. In total 69 siblings were close in age to the 12-year-old twins (mean age was 12.8 years, with a range of 11.5 up to 14.0 years, 38 girls and 31 boys). These sibling data had either been collected during the first visit to the lab ($N = 50$), or at follow-up ($N = 19$).

Written informed consent was obtained from all subjects and their parents and the study was approved by the Dutch Central Committee on Research involving Human Subjects (CCMO). Parents were financially compensated for travel expenses and the children received a small gift each.

Measures

Participants were individually tested in separate rooms by experienced test administrators. At baseline the full version of the Wechsler Intelligence Scale for children — Third version (WISC III; Wechsler et al., 2002) was used, including six verbal (information, similarities, arithmetic, vocabulary, comprehension, and digit span), and six non-verbal subtests (picture completion, coding, picture concepts, block design, picture assembly, and symbol search). At follow-up a shortened version of the WISC III was administered, including four verbal subtests (similarities, arithmetic, vocabulary, and digit span), and two nonverbal subtests (picture completion, and block design). Test scores were corrected for the different number of subtests for verbal and nonverbal separately. For full scale, these corrected scores were summed, giving equal weight of verbal and nonverbal subtests to full scale performance. Raw scores were standardized according the age of the child at moment of testing, based on a population sample of same-aged subjects in the Netherlands (Wechsler et al., 2002), giving full scale intelligence quotient (FSIQ), as well as a verbal (VIQ), and nonverbal quotient (PIQ).

Genetic Analyses

Monozygotic (MZ) twins are genetically identical and share (nearly) 100% of their genetic material, while dizygotic (DZ) twins and full siblings share on average 50% of their segregating genes. By comparing the MZ, DZ and twin-sibling covariance structures for a univariate or longitudinal phenotype, one can estimate the relative influences of genes and environment on phenotypic variation and on covariation among phenotypes. Additive genetic factors (A) represent the influences on the phenotype of multiple alleles at different loci on the genome that act additively. The proportion of variance in a trait that can be attributed to genetic factors is termed heritability. Common environmental influences (C) include all environmental factors that make twins and siblings who grow

up in the same family resemble each other. Environmental factors not shared with other family members are referred to as unique environmental influences (E), and also include measurement error (Falconer & Mackay, 1996).

All data analyses were carried out with structural equation modeling (SEM) in the software package Mx (Neale et al., 2006). All available data were analyzed, that is, regardless of whether subjects participated once or twice in the study. Parameters were estimated by full-information maximum likelihood. Tests of significance of parameters were carried out by comparing the model fits of a model including that parameter to a model in which the parameter estimate was constrained at zero. The goodness of fit of different models was evaluated by comparing differences in log-likelihood. Twice the difference between log likelihoods is chi-squared distributed with degrees of freedom (df) equal to the difference in the number of parameters estimated in the two models.

First, phenotypic correlations over time, MZ, DZ and twin-sibling correlations and cross-twin cross-time correlations were estimated for FSIQ, VIQ and PIQ. Equality of means and variances for twins and siblings and of DZ and twin-sibling correlations were tested.

Second, a longitudinal ACE model was fitted to the data (see Figure 1). The latent A, C and E factors in this model are standardized to have zero mean and unit variance. Path coefficients a_{11}, c_{11}, and e_{11} represent the influences of A_1, C_1, and E_1 on IQ in childhood. Path coefficients a_{21}, c_{21}, and e_{21} represent the influences of A_1, C_1, and E_1 on IQ in early adolescence. The path coefficients a_{22}, c_{22}, and e_{22} represent the influences of A_2, C_2, and E_2 on IQ in early adolescence (i.e. the part that is not influenced by A_1, C_1 or E_1). The total variance of IQ in childhood is given by the sum of the squares of a_{11}, c_{11}, and e_{11} (Vp= $a_{11}^2 + c_{11}^2 + e_{11}^2$). Heritability in childhood is given by $a_{11}^2 / (a_{11}^2 + c_{11}^2 + e_{11}^2)$. The total variance of IQ in early adolescence is given by the sum of the squares of a_{21}, a_{22}, c_{21}, c_{22}, e_{21}, and e_{22} (Vp= $(a_{21}^2 + a_{22}^2) + (c_{21}^2 + c_{22}^2) + (e_{21}^2 + e_{22}^2)$). As a result, the heritability in early adolescence is given by $(a_{21}^2 + a_{22}^2) / (a_{21}^2 + a_{22}^2 + c_{21}^2 + c_{22}^2 + e_{21}^2 + e_{22}^2)$.

The covariance between IQ in childhood and IQ in early adolescence is derived from multiplying the path coefficients that define the association of childhood and early adolescence. The genetic covariance is given by $(a_{11} * a_{21})$, the common environmental covariance by $(c_{11} * c_{21})$, and the unique environmental covariance by $(e_{11} * e_{21})$. The total covariance is a summation: $(a_{11} * a_{21}) + (c_{11} * c_{21}) + (e_{11} * e_{21})$. The extent to which genetic influences in childhood and early adolescence overlap can be calculated as the genetic correlation $r_g = (a_{11} * a_{21}) / \sqrt{(a_{11}^2 * (a_{21}^2 + a_{22}^2))}$. In a similar way the common environmental and unique environmental correlations can be obtained. All correlations were tested to establish if they significantly contribute to the stability of intelligence measures across age by constraining a_{21}, c_{21}, or e_{21} at zero. In addition, all genetic and environmental correlations over time were tested whether they were significant different from 1.00, by constraining the path of a_{22}, c_{22}, and e_{22} at zero (implicating complete overlap of factors between the ages). As the sample is relatively small and sex differences in genetic architecture of IQ based measures have not or very seldom been reported (e.g. Haworth et al., 2010) we pooled the data for boys and girls.

Results

Mean age at testing and IQ scores for twins and siblings are given in Table 1. Full scale (FSIQ), verbal (VIQ), and performance IQ (PIQ) were normally distributed in all groups. Mean IQ scores were higher for siblings than for twins in both childhood and early adolescence for FSIQ ($\chi^2 = 18.58$, $df = 1$, $p < .01$), VIQ ($\chi^2 = 19.47$, $df = 1$, $p < .01$) and PIQ ($\chi^2 = 6.96$, $df = 1$, $p = .01$). For all IQ measures, variances in twins did not differ by birth order or zygosity. For VIQ, a scalar was included in the model to account for a larger variance in siblings ($\chi^2 = 6.20$, $df = 2$, $p = .04$).

Table 2 gives the phenotypic correlations across time, MZ and DZ twin, and twin-sibling correlations for FSIQ, VIQ and PIQ. Phenotypic correlations over time (r_p) were high for FSIQ ($r_p = .72$) as well as for VIQ ($r_p = .72$). PIQ showed a somewhat lower phenotypic correlation over time ($r_p = .56$). For all three IQ measures, MZ correlations were higher than DZ and twin-sibling correlations, indicating genetic influences. For FSIQ and PIQ, MZ correlations increased with age, while for VIQ, the MZ correlations remained approximately similar. The higher DZ correlations for VIQ imply that common environment is of importance for both age groups. DZ correlations were not different from the twin-sibling correlations in childhood (FSIQ, $\chi^2 = .56$, $df = 1$, $p = .45$; VIQ, $\chi^2 = .20$, $df = 1$, $p = .65$; PIQ, $\chi^2 = 3.03$, $df = 1$, $p = .08$), or in early adolescence (FSIQ, $\chi^2 = .43$, $df = 1$, $p = .51$; VIQ, $\chi^2 = 3.21$, $df = 1$, $p = .07$; PIQ, $\chi^2 = .68$, $df = 1$, $p = .41$).

Parameters estimates from the longitudinal ACE models are given in Table 3. Each of the path coefficients

TABLE 1

Mean (*SD*) of Age at Testing, Full Scale (FSIQ), Verbal (VIQ), and Performance (PIQ) Intelligence Scores for All Subjects in Childhood and Early Adolescence

	Childhood		Adolescence	
	Twins *(N = 224)*	Siblings *(N = 46)*	Twins *(N = 177)*	Siblings *(N = 69)*
Age at test (years)	9.1 (0.1)	10.9 (0.4)	12.1 (0.3)	12.8 (0.9)
FSIQ	99.9 (13.5)	105.7 (16.4)	100.3 (14.1)	106.5 (16.3)
VIQ	99.6 (14.5)	106.2 (17.7)	102.3 (12.5)	106.6 (15.8)
PIQ	100.1 (12.3)	103.5 (14.5)	98.3 (17.6)	106.7 (15.6)

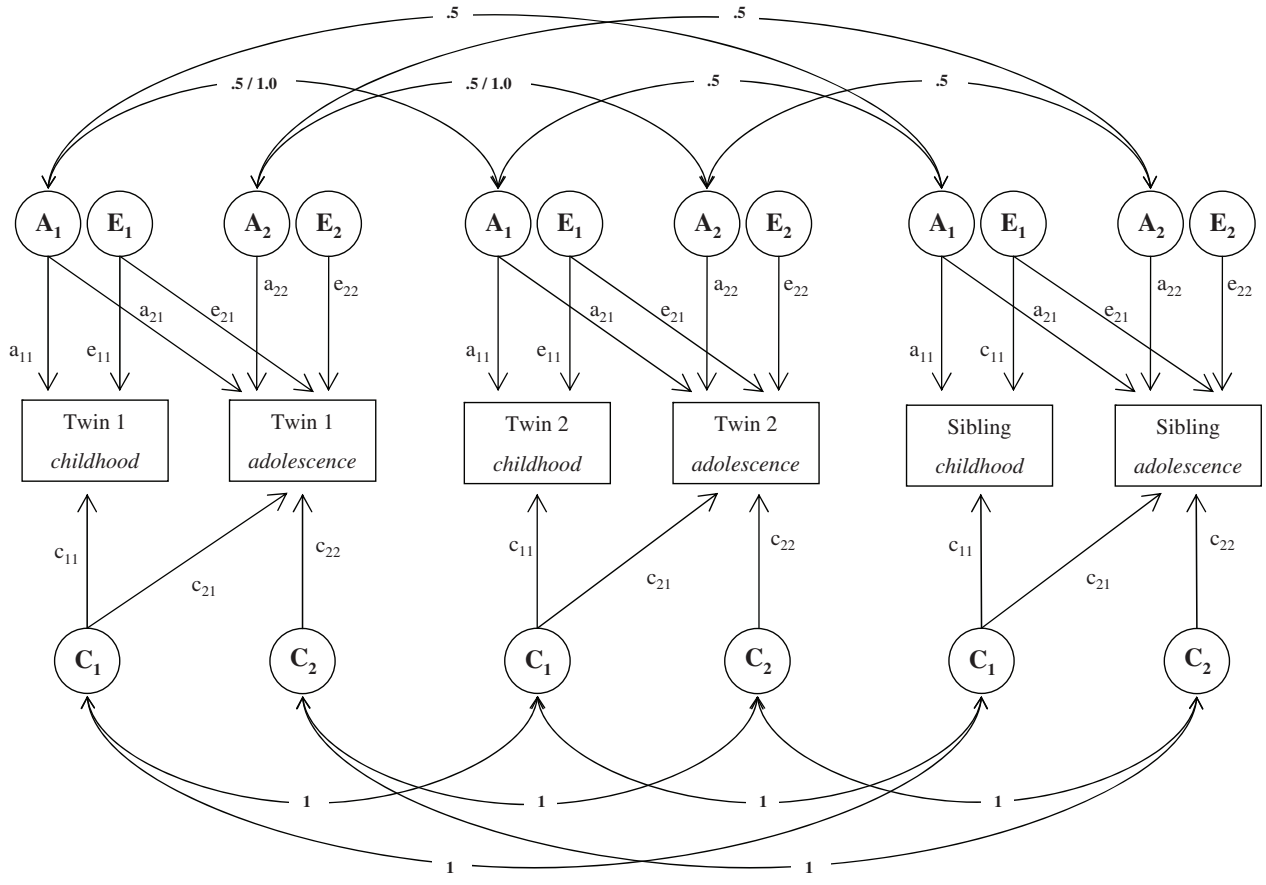

FIGURE 1

Path diagram representing the longitudinal genetic model fitted to the intelligence scores in childhood and early adolescence. Squares represent the measured variables (i.e. intelligence score), and the circles represent latent, unobserved factors. Double-headed arrows represent correlations between the latent factors. The influence of the first set of latent factors on intelligence in childhood is represented by the path coefficients (one-headed arrows) a_{11}, c_{11}, and e_{11}, and in early adolescence by a_{21}, c_{21} and e_{21}. The second set of latent factors influence intelligence in early adolescence only, and is represented by the path coefficients a_{22}, c_{22} and e_{22}. Following path tracing rules; Var ($IQ_{childhood}$) = (a_{11}^2) + (c_{11}^2) + (e_{11}^2), and Var ($IQ_{adolescence}$) = ($a_{21}^2 + a_{21}^2$) + ($c_{21}^2 + c_{22}^2$) + ($e_{21}^2 + e_{22}^2$). In MZ twins the genetic factors are correlated 1.0 within twin pairs and .5 for DZ and siblings. In MZ, DZ and twin-siblings, the common environmental factors are correlated 1.0, and the unique environmental factors are uncorrelated.

was tested for significance. The model fitting statistics of the full ACE model and the submodels are given in Table 4.

The contributions of genetic, common environment and unique environmental influences to the stability of IQ measures between childhood and adolescence were tested for significance by constraining a_{21}, c_{21}, and e_{21} at zero. The genetic covariance was significant for FSIQ (χ^2 = 18.41, df = 1, $p < .01$), VIQ (χ^2 =10.72, df = 1, $p = < .01$), and PIQ (χ^2 = 19.36, df = 1, $p < .01$). Common environmental influences were contributing to stability over time for FSIQ (χ^2 = 7.75, df = 1, $p = < .01$), and VIQ (χ^2 = 9.65, df = 1, $p = < .01$), but not for PIQ (χ^2 = 2.07, df = 1, p =.08). Unique environmental influences did not contribute to the stability of any of the IQ scales, indicating that these influences were not transmitted over time. Table 5 gives the proportion of variance that could be attributed to genetic, common and unique environmental influences on all three IQ scales in childhood and early adolescence. Genetic, common and unique environmental correlations are also displayed in Table 5.

Both the unstandardized genetic variance and the heritability increased for all IQ scales. Heritability of FSIQ increased from 34% in childhood to 65% in early adolescence. Common environmental influences on the other hand, decreased with age for FSIQ, namely from 43% in childhood down to 18% in early adolescence. For VIQ a similar pattern of genetic and environmental influences was observed. Heritability increased from 37% in childhood up to 51% in adolescence, while the contribution of common environmental factors decreased from 42% down to 26%. For PIQ, heritability increased from 46% up to 72%, and in contrast to the other IQ scales, common environment influences did not reach significance for PIQ in both age groups. When common environment was excluded from the model (χ^2 = 1.21, *df* = 3, p = .75), heritability was 64% in childhood and 72% in early adolescence (Table 5).

TABLE 2

Phenotypic (r_p) and Twin Correlations, With Their 95% Confidence Intervals of Full Scale (FSIQ), Verbal (VIQ) and Performance (PIQ) Intelligence Scores in Childhood and Early Adolescence

		Childhood			Adolescence		
	r_p	rMZ	rDZ	rTwin-sibling	rMZ	rDZ	rTwin-sibling
FSIQ	0.72 (.64–.79)	0.75 (.63–.84)	0.54 (.34–.68)	0.6 (.45–.72)	0.81 (.69–.88)	0.55 (.34–.70)	0.47 (.29–.61)
VIQ	0.72 (.63–.79)	0.8 (.69–.87)	0.62 (.44–.74)	0.58 (.41–.70)	0.76 (.62–.85)	0.65 (.45–.78)	0.44 (.26–.59)
PIQ	0.56 (.45–.65)	0.61 (.43–.64)	0.23 (-.02–0.45)	0.45 (.28–.59)	0.74 (.58–.84)	0.27 (.00–.49)	0.38 (.19–.54)

TABLE 3

Unstandardized Parameter Estimates for Additive Genetic (*a*), Common Environmental (*c*), and Unique Environment (*e*) Path Coefficients from the Longitudinal Model

		a_{11}, a_{12} and a_{22}		c_{11}, c_{12} and c_{22}		e_{11}, e_{12} and e_{22}	
		Childhood	Adolescence	Childhood	Adolescence	Childhood	Adolescence
FSIQ	Childhood	8.15		9.16		6.78	
	Adolescence	11.53	3.55	6.25	0	0.42	6.21
VIQ	Childhood	8.97		9.58		6.68	
	Adolescence	6.99	5.52	6.33	-0.01	1.3	5.99
PIQ	Childhood	8.51		5.12		7.72	
	Adolescence	14.4	0	1.92	0	-1.72	8.7

TABLE 4

Model Fit Results for the Longitudinal ACE Model and the Nested Submodels for Full Scale (FSIQ), Verbal (VIQ), and Performance (PIQ) IQ

	FSIQ				VIQ				PIQ			
Model	-2LL	*df*	χ^2	*p*	-2LL	*df*	χ^2	*p*	-2LL	*df*	χ^2	*p*
Full ACE longitudinal model	3919.68	503			3917.14	501			4062.4	503		
1. No genetic covariance (a_{21})	3938.09	504	18.41	< .01	3927.87	502	10.72	< .01	4081.77	504	19.36	<.01
2. No common environmental covariance (c_{21})	3927.43	504	7.75	< .01	3926.79	502	9.65	< .01	4062.67	504	0.27	0.30
3. No unique environmental covariance (e_{21})	3919.9	504	0.23	0.32	3919.63	502	2.49	0.06	4064.48	504	2.07	0.08
4. No genetic influence through a_{11}	3938.09	504	18.41	< .01	3928.35	502	11.21	< .01	4081.77	504	19.36	< .01
5. No genetic influence through a_{22}	3919.77	504	0.09	0.38	3919.27	502	2.13	0.07	4062.4	504	0.00	1.00
6. No common environmental influence through c_{11}	3930.01	504	10.33	< .01	3927.98	502	10.84	< .01	4063.61	504	1.21	0.14
7. No common environmental influence through c_{22}	3919.68	504	0	1	3917.14	502	0	1	4062.4	504	0.00	1.00

Note: Model fit is given by -2 *log-likelihood (-2LL), and the degrees of freedom (df). All nested models are compared to the full ACE model.

Genetic correlations across time were .96 for FSIQ, .78 VIQ, and .90 for PIQ and all genetic correlations were found to be not significantly different from 1.00, because the path of a_{22} could be constrained at zero without a significant worse fit of the data. Common environmental correlations across time were 1.00 for both FSIQ and VIQ.

Discussion

Genetic and environmental influences on the stability of full scale (FSIQ), verbal (VIQ) and nonverbal (PIQ) intelligence scores in children (9–11 years) and in young adolescents (12–14 years) were explored in a sample of twins and siblings. The main finding was that the stability of FSIQ and VIQ was explained by genetic and to a smaller extent by common environmental influences, while stability of PIQ was completely explained by genetic influences during this important developmental period of the transition from childhood to early adolescence.

The influence of genes and common environment on the stability of FSIQ over time is in line with previous longitudinal studies in this age group (Boomsma & van Baal, 1998; Bartels et al., 2002; Davis et al., 2008; Davis et al., 2009; Spinath et al., 2003; Bishop et al., 2003). In addition to stable genetic and environmental influences some studies also observed age-specific genetic and common environmental influences (Davis et al., 2008; Davis et al.,

TABLE 5

Proportion of Total Variance (and 95% Confidence Interval) That Can Be Attributed To Genetic (A), Common Environment (C), and Unique Environmental Influences (E) for Full Scale (FSIQ), Verbal (VIQ), and Performance (PIQ) IQ and Genetic (r_g), Common Environmental (r_c), and Unique Environmental (r_e) Correlations Across Time

		Childhood			Adolescence					
	Model	A	C	E	A	C	E	r_g	r_c	r_e
FSIQ	ACE	0.34 (.14–.61)	0.43 (.18–.61)	0.23 (.16–.34)	0.65 (.43–.82)	0.18 (.02–.38)	0.17 (.11–.28)	0.96 (.74–1.00)	1 (.69–1.00)	0.07 (-.20–.34)
VIQ	ACE	0.37 (.11–.64)	0.42 (.18–.63)	0.21 (.13–.32)	0.51 (.25–.72)	0.26 (.06–.46)	0.24 (.15–.37)	0.78 (.53–1.00)	1 (.71–1.00)	0.21 (-.05–.46)
PIQ	ACE	0.46 (.20–.73)	0.17 (.00–.39)	0.38 (.26–.51)	0.72 (.43–.82)	0.01 (.00–.25)	0.27 (.18–.41)	1 (.73–1.00)	1 (-1.00–1.00)	-0.19 (-.42–.09)
	AE	0.64 (.51–.75)		0.36 (.25–.49)	0.72 (.58–.82)		0.28 (.18–.42)	0.9 (.74–1.00)		-0.19 (-.42–.07)

2009; Bishop et al., 2003). In longitudinal studies intelligence measures often are based on different age appropriate test instruments. This potentially may introduce age-specific influences caused by the different test instruments used at different ages. In our study IQ measures were based on the WISC III at both test occasions.

For verbal and nonverbal cognitive abilities, a different pattern of results between the two domains emerged. Stability of VIQ was influenced by both genetic and common environmental factors, while stability of PIQ was explained by genetic influences only. The number of longitudinal studies that separate the verbal and non-verbal cognitive abilities, using the VIQ and PIQ scales is limited. Similar results were observed in an independent longitudinal twin study (Hoekstra et al., 2007). In that study a larger number of ages (5, 7, 10, 12 and 18 year) was included and this enabled the testing of a transmission model. In this type of model, influences from earlier to later ages and innovation terms unique for each age can be tested (Hoekstra et al., 2007). This study and the present study both contribute to the finding that during childhood and early adolescence verbal and nonverbal cognitive domains develop in different ways (Hoekstra et al., 2007).

When looking at the heritability estimates in childhood and early adolescence, an increase in heritability was observed for all IQ scales, which was due an increase in genetic variance. Heritability of FSIQ increased from 34% in childhood to 65% in early adolescence. Common environmental influences on FSIQ decreased in importance from 43% in childhood down to 18% in early adolescence. Heritability of VIQ increased from 37% in childhood to 51% in early adolescence. Common environmental influences on VIQ decreased from 42% in childhood down to 26% in early adolescence. Heritability of PIQ was 64% in childhood and increased up to 72% in early adolescence. There was no contribution of common environmental influences on PIQ in childhood or in early adolescence. This pattern of increasing genetic and decreasing common environmental influences with age are in line with other large studies (Davis et al., 2009; Bartels et al., 2002; Bishop et al., 2003; Hoekstra et al., 2007; Haworth et al., 2010).

However, an increase in the influences of genetic factors as seen for IQ is not the general rule for childhood behavioral traits. For example, heritability of externalizing problem behavior was generally stable across the ages 3 up to 12, while a slight decrease in heritability was observed for internalizing behaviors across this age range (Bartels et al., 2004). A decrease in heritability by age was also observed in studies exploring anxious and depression symptoms, and withdrawn behavior in childhood (Hoekstra et al., 2008; Boomsma et al., 2005). During adolescence heritability on depression and anxious symptoms and withdrawn behavior were found to remain around 50% (Lamb et al., 2010). For attention problems, a trait which is associated with IQ (Polderman et al., 2006), heritability is uniformly high between ages 3 to 12 years (Rietveld et al., 2004). Between ages 13 and 25, an increase in heritability was observed in a large meta-analysis of externalizing behavior, anxiety and depression symptoms (Bergen et al., 2007).

The increase in the heritability of intelligence may be the result of several processes. Genetic amplification has been previously suggested (DeFries et al., 1987), and this is what was observed in the present study. Also, as children grow older they are more likely to select or maybe even partly create their own environment, driven by their genetic disposition, resulting in an increased expression of their genetic potential (Plomin et al., 1977). Simultaneously, common environmental influences that are present in childhood diminish with increasing age. The increase of genetic and decrease of common environmental influences on IQ could be a result of children becoming more independent from their familial environmental and parental influences (Scarr & Mccartney, 1983). We now show that this is mainly driven by the verbal counterpart of intelligence, and not by nonverbal abilities.

Children undergo considerable improvements in their cognitive abilities throughout childhood and adolescence. Differences between children in verbal intelligence are

explained not only by genetic differences but apparently also by the common environment. For example, maternal education, other parental influences, neighbourhood characteristics, and social economic status all tend to be associated with intelligence, verbal ability and reading skills in childhood and adolescents (Leventhal & Brooks-Gunn, 2000), which are all factors shared by children from the same home. An additional explanation for a contribution in twin studies of common environment is assortative mating. For intelligence it has been found that spouses resemble each other in IQ scores. When resemblance of the parents is caused by phenotypic assortment, this can induce genetic similarity between the parents, which in turn affects the genetic similarities between siblings and increases resemblance in DZ twins and siblings. As a consequence the heritability can be underestimated and the estimates of common environmental influence inflated (Cavalli-Sforza & Bodmer, 1971). For IQ based on the Raven test, phenotypic assortment was found in the parents of the participating offspring who were tested at the baseline assessment of the present sample (van Leeuwen et al., 2008).

The increase in genetic variance found in all IQ scales holds valuable information for the fields of molecular genetics and of neuroscience, studying brain maturation and the associations of general cognitive abilities with brain changes during development. Despite the increase in heritability of intelligence up to relatively high estimates in adulthood, gene finding studies have not yet provided consistent results pointing to genetic variants that are associated with intelligence (Posthuma et al., 2005; Bochdanovits et al., 2009), although there are genetic variants that are associated with disorders where cognitive functioning is in some way affected (Flint, 1999; Deary et al., 2009). With respect to individual differences in intelligence in healthy samples, large genome-wide association studies may very well provide more information on this in the near future. That individual differences in verbal and performance IQ each exhibit different etiology is an indication that the separation of these two intelligence domains can be informative. Although higher genetic influences are no guarantee for success in genome-wide association studies (Manolio et al., 2009), the presence of genetic influences on a trait, and the contribution of the same genetic influences on stability of a trait over time is a preferred characteristic and reassuring when IQ scores across different ages are pooled for an increase in power in gene-finding studies.

How the increase in genetic influences on intelligence is associated with developmental brain changes that occur around the period of transition from childhood to adolescence is still not fully understood. Measures for brain anatomy are under large genetic influences throughout early infancy (Gilmore et al., 2010; Smit et al., 2010), childhood and adolescence (Smit et al., 2010; Peper et al., 2009; Yoon et al., 2010; Brouwer et al., 2010; Schmitt et al., 2007; Wallace et al., 2006), and adulthood (Peper et al., 2007; Baare et al., 2001). While the genetic influences that explain individual differences in brain size seem to be generally stable at different ages, the human brain itself is a highly dynamic organ, and undergoes considerable developmental changes during development from infancy up to adulthood (Giedd et al., 1999). The genetic influences found for variation in brain anatomy showed an overlap with genetic influences on intelligence in adults (Posthuma et al., 2002; Hulshoff Pol et al., 2006), and in childhood and adolescence (van Leeuwen et al., 2009; Betjemann et al., 2010; Wallace et al., 2010). The amount of brain changes in cortical thickness in adults is under genetic influences, and partly overlap with genetic influences on IQ (Brans et al., 2010). There are indications that the level of intelligence is associated with developmental trajectories of the human cortex during adolescence (Shaw et al., 2006).

The mechanisms behind the associations between brain anatomy and function, and the developmental brain changes with the level of general cognitive abilities are not fully understood. Recent research is also focussing on the efficiency of the brain, by means of network analyses. A higher level of functional connectivity was found to be associated with higher levels of IQ (van den Heuvel et al., 2009). Furthermore, there are indications that distinct associations with brain anatomy are present when verbal and nonverbal cognitive abilities are explored separately (Betjemann et al., 2010; Wallace et al., 2010). There is more research needed to understand the etiology behind the stability of IQ over time and the possible relationship with brain changes, especially in this period in development.

There were some limitations of the extended twin design used in the present study. The mean IQ score of the siblings was higher compared to their own co-twins in both age groups. This could be partly explained by studies that report that birth order within a family was associated with higher IQ score (Boomsma et al., 2008; Zajonc & Sulloway, 2007). In the majority of the included families the participating sibling is the oldest child in the family. Furthermore, the phenotypic correlation of PIQ across time was relatively low. One reason could be the use of a shortened version of the subscales used to measure performance IQ on the second test occasion.

To summarize, our main finding is that stability in verbal IQ is influenced by genetic and common environmental influences, while stability of performance IQ is driven by genetic influences. These findings contribute to the existing literature that verbal and nonverbal domains have different developmental trajectories. Children undergo considerable changes in their environment, but also in brain anatomy and function during this period in life, and the rates of these changes are most likely linked together in a complex manner. How the developmental trajectories of cognitive abilities are related with brain development has to be explored further in more detail.

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