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Genetic Influences on Childhood IQ in 5- and 7-Year-Old Dutch Twins

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To study changes in the genetic architecture of cognitive functioning as children grow up, we measured intelligence in a sample of young Dutch twins at ages 5 and 7 years. By applying a developmental model to the data, we could examine the continuity and change in genetic and environmental influences from age 5 to age 7. Results show that genetic influences persist from age 5 to age 7 and greatly gain in relative importance during this period. There is no evidence of new genetic influences being expressed at age 7. The influence of genetic factors on intelligence at age 5 is relatively small, accounting for 27% of the total phenotypic variance. At age 7, genetic factors explain 62% of the variance in IQ scores. This increase in heritability is due both to an increase in the total genetic variance and to a decrease in the amount of variance explained by shared environmental factors. The shared family background influences IQ in early childhood, and these effects also persist until age 7, but their relative importance becomes smaller. At age 5, the shared family environment accounts for half of the variance in IQ. At age 7, the influence of the family environment decreases to 10%. Unique environment acts age specifically and accounts for roughly a quarter of the total variance in IQ both at ages 5 and 7.

Several recent reviews on the genetics of intelligence and cognitive abilities have paid special attention to the question of developmental stability and change in the genetic and environmental influences underlying individual differences in IQ (e.g., Boomsma, 1993; McGue, Bouchard, Iacono, & Lykken, 1993; Plomin & Rende, 1991; Thompson, 1993). Most studies agree that in infancy the heritability for IQ is low, whereas the influence of the shared family environment is relatively high. From age 6 onward, in adolescence and in adulthood, heritability estimates for IQ

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increase (to 50%–70%) and the influence of common environment becomes negligible.

A meta-analysis of monozygotic (MZ) and dizygotic (DZ) twin similarities in intelligence as a function of age was reported by McCartney, Harris, and Bernieri (1990). Their results support the idea that heritability for IQ increases over time, whereas the importance of a common family background decreases. Analyses of total IQ and specific abilities showed that, except for verbal IQ, DZ twins became more dissimilar over time than did MZ twins. For total IQ (results from 16 independent studies), the correlations of twin resemblance with age were .15 for MZ and –.25 for DZ twins. Correlating estimates for heritability and common and unique environment with age showed correlations of .36, –.37 and –.15, respectively. These correlations indicate that heritability increases with increasing age, whereas the importance of environmental influences in explaining variation in IQ decreases with age. However, these last analyses must be viewed with some caution, because components of variance are less reliable than the correlations on which they are based, and the analyses were carried out on only a few data points.

McCartney et al. (1990) recognized that in cross-sectional studies, heritability differences between age groups can result from either age or cohort effects. Only longitudinal studies can resolve whether the suggested increase in heritability during childhood and adolescence is due to an increase in genetic variance with age, or whether it is associated with cohort effects. More important, however, only longitudinal studies can reveal how genes and environment operate throughout development. For example, is the increase in heritability due to new, additional, genetic factors being expressed as children grow older, or is there an amplification of existing genetic influences?

Eaves, Long, and Heath (1986) applied a general developmental model to longitudinal data from the Louisville twin study (Wilson, 1983). Twins were measured from 3 months through 15 years with the Bayley Mental Scale, the Stanford–Binet Intelligence Scale, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) at 4, 5, and 6 years, and Wechsler Intelligence Scale for Children (WISC) at later ages. Analysis of these IQ measures showed initially small but persisting and accumulating effects of a single set of genes; it also showed appreciable influences of common environmental effects that were persistent, but showed age-specific input as well. Unique environmental influences were more occasion specific and less persistent than were genetic and shared environmental effects.

Loehlin, Horn, and Willerman (1989) analyzed IQ data in adoptees from two occasions approximately 10 years apart. At the first occasion, adoptive children were between 3 and 14 years old. Parents were measured once. A model with phenotypic transmission gave a good fit and reasonable parameter estimates. (Eaves

et al., 1986, noted that such a model is equivalent to genetic and environmental transmission parameters being the same.) At the second occasion, heritability slightly increased, although there was no longer evidence for common environmental effects.

DeFries, Plomin, and LaBuda (1987) combined IQ data of twins from the Louisville twin study who were measured at ages 1, 2, 3, and 4 years and data on scholastic abilities in young adult twins with IQ data from adoptive and control children at ages 1, 2, 3, and 4 years, and their parents in a longitudinal analysis. Estimates for the influence of a twin shared environment were high and transmission from parental phenotype to child's environment was not significant. The heritability estimate for IQ in adults was 55%; in children it increased from 10% at age 1, to 17% at age 2, to 18% at age 3, and to 26% at age 4. Estimates for the genetic stability parameter from childhood to adulthood increased from .67 to around .90 from ages 1 to 4. These results lend support to the developmental amplification model proposed by DeFries (in Plomin & DeFries, 1985). In this model, the effects of genes that are relevant to mental development during infancy and childhood are amplified during adulthood.

Cardon, Fulker, DeFries, and Plomin (1992) analyzed IQ data from adopted and nonadopted siblings at ages 1 through 7 years and from twins at ages 1 through 3 years. Children were measured at ages 1 and 2 (Bayley MDI), at ages 3 and 4 (Stanford–Binet Intelligence Scale), and at age 7 (Wechsler Intelligence Scale for Children–Revised [WISC–R]). Higher heritabilities than those obtained in the DeFries et al. study (1987) were found. There was evidence for common environmental influences on IQ in siblings as well as in twins. Heritabilities were estimated at 55% at age 1, 68% at age 2, 59% at age 3, 53% at age 4, and 52% at age 7. The influence of common environment was 10% at each age. The genetic part of the model showed increasing transmission parameters and substantial genetic innovations at all ages, except at age 4. Common environment functioned as a single, constant background factor. Cherny and Cardon (1994) analyzed these same data from adopted and nonadopted siblings and included a first principal component score from a telephone-administered cognitive abilities test battery at age 9. They did not find a significant contribution of shared environment but replicated the finding of a large amount of new genetic variance at age 7. At age 9, a significant part of the genetic variance also was due to new genetic factors. The unique environmental influences were time specific.

A complication in studying intelligence in early life is that changes in heritability and low stability, as evident in small transmission parameters, may be related to changes in IQ tests that are employed during infancy and later on. Longitudinal studies using traditional infant and childhood intelligence tests provide little support for stability between IQ measures during infancy and later ages (e.g., Boomsma, 1993; Bornstein & Sigman, 1986), and this lack of stability may be due to changes

in the IQ tests that are administered at different ages. For example, the relatively large influence of new genetic factors on IQ at ages 7 and 9 reported by Cherny and Cardon (1994) may reflect a genuine new source of genetic influence, or it may reflect merely different IQ tests measuring different aspects of intelligence.

We administered the same IQ test, at ages 5 and 7, to a sample of Dutch twins to study changes in the relative importance of genetic and environmental influences on IQ, and to study the components of phenotypic stability. This second objective addresses the question to what extent phenotypic stability in IQ is due to the same genes being expressed at different ages and to what extent phenotypic stability is due to the same environmental influences being of importance. Contrary to popular points of view, genetically determined characters need not be stable, nor are longitudinally stable characters always influenced by heredity (Molenaar, Boomsma, & Dolan, 1991). A Dutch child intelligence test was used that was recently developed to measure IQ in 4- to 11-year-old children (Bleichrodt, Drenth, Zaal, & Resing, 1987).

METHOD

Participants

Five-year-old twins were recruited from the Netherlands Twin Register (Boomsma, Orlebeke, & van Baal, 1992). The twins took part in a longitudinal study of brain development and came to the laboratory together, accompanied by one or two of their parents. Whereas electroencephalogram (EEG) and event-related potential (ERP) were measured in one child in each twin pair, the other child completed an IQ test. Children were usually tested by the same examiner. At age 5, 209 twin pairs participated. At age 7, 192 twin pairs came back for a second assessment. There was no significant IQ difference at age 5 between children who participated and children who did not participate the second time. For most twins, zygosity was determined by blood and DNA typing (see van Baal, de Geus, & Boomsma, this issue). At age 5 there were 47 monozygotic female (MZF) twin pairs, 37 dizygotic female (DZF) twin pairs, 43 monozygotic male (MZM) twin pairs, 42 dizygotic male (DZM) twin pairs, and 39 dizygotic twin pairs of opposite sex (DOS; see Table 1 for the number of pairs that participated at age 7). The average age at the first IQ test was 5 years and 3 months ($SD = 0.19$), at the second IQ test the average age was 6 years and 10 months ($SD = 0.20$). Nearly all Dutch children start school (kindergarten) in the month after their 4th birthday and begin formal education at age 5. At the time of first testing all twins were in their 1st year of formal education.

Procedure

Children were tested with the Revisie Amsterdamse Kinder Intelligentie Test (RAKIT) that was developed to assess IQ in children ages 4 to 11 years (Bleichrodt,

TABLE 1
Maximum Likelihood Estimates of Means and Standard Deviations for IQ at Ages 5 and 7 for First- and Second-Born Twins

		Twin 1–IQ 5		*Twin 1–IQ 7*		*Twin 2–IQ 5*		*Twin 2–IQ 7*	
	N[a]	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
MZM	43/38	102.8	13.7	102.4	15.1	101.0	13.2	101.8	13.1
DZM	42/40	99.3	12.3	100.4	15.5	102.0	11.9	101.0	14.7
MZF	47/42	103.5	13.2	101.8	15.1	105.2	14.0	103.3	16.2
DZF	37/34	101.3	14.1	101.7	13.6	102.1	12.6	101.9	11.9
DOS	39/38	107.2	13.2	106.5	15.1	102.4	11.5	104.7	15.8

Note. In opposite-sex twins, boys are listed first.
[a]Number of twin pairs that participated the first and second time.

Drenth, et al., 1987). The short version of the RAKIT was used, which takes 1 hour to administer. It consists of six subtests: three subtests that measure perceptual and spatial IQ (hidden figures, abstract reasoning, and visual analysis and motor speed), and three subtests that assess verbal IQ (word recognition, verbal learning, and verbal fluency). The correlation in a normative sample between the short version and the complete RAKIT was 0.93 both in 5-year-olds ($N = 204$) and in 7-year-olds ($N = 196$). The correlation between the RAKIT and the Dutch translation of the WISC–R in 116 children between the ages of 6;2 years and 7;2 years was 0.81; after correction for attenuation, the correlation was 0.86 (Bleichrodt, Resing, Drenth & Zaal, 1987).

Genetic Analysis

Bivariate genetic models were fitted to the raw IQ data obtained at ages 5 and 7. Three sources of variation were considered: genetic effects (G), common or shared family environment (C), and unique environment (E). Two sets of latent factors consisting of these three influences were specified at ages 5 and 7. The covariance between IQ scores at ages 5 and 7 was modeled by latent G, C, and E factors that influenced IQ at both age 5 and age 7 (see Figure 1). A second set of latent factors accounted for age-specific genetic and environmental effects at age 7.

The resemblance between twin siblings could be due to correlated genetic factors, common environmental factors, or both. For identical twins, the correlation between their genotypes is 1.0, and for DZ twins, it is 0.5. For both types of twins, the correlation between their common environmental factors is 1.

First, we tested if there were gender differences in the relative influences of G, C, and E on the phenotype; second, we tested whether either G or C could be omitted from the model. Leaving out G implies that all resemblance between twins can be attributed to the common family environment shared by twins. Leaving out C

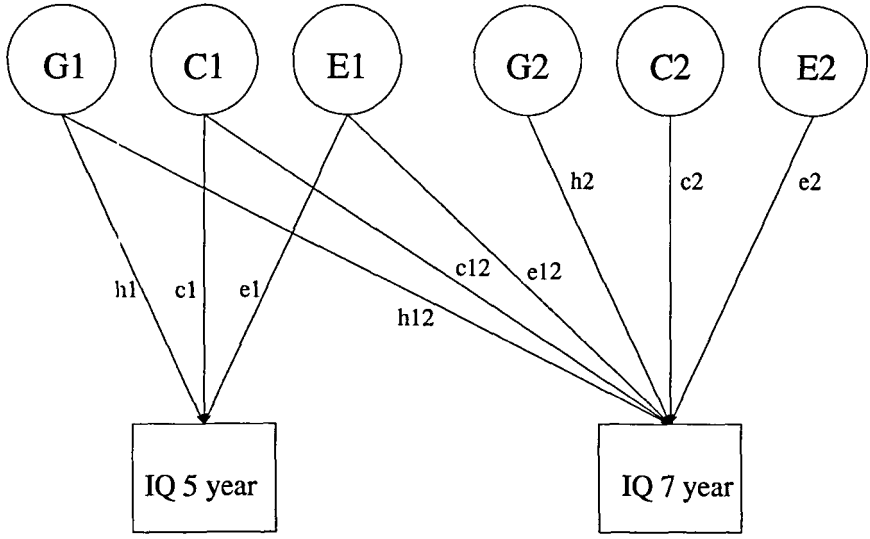

FIGURE 1 Path diagram depicting the bivariate genetic model for a single individual that was fitted to the IQ data assessed at age 5 and age 7. Squares represent measured variables; circles represent latent, unobserved, factors. G1 and G2 are the genetic influences on IQ at age 5 and 7, C1 and C2 are the common environmental factors, and E1 and E2 are the unique environmental factors. Path coefficients h1, c1, e1 and h2, c2, e2 represent the relative influences on IQ of the latent factors at Times 1 and 2, respectively; h12, c12, and e12 are path coefficients that represent the relative influence of G1, C1, and E1 on the second IQ assessment at age 7. In MZ twins, the genetic factors are correlated 1.0 within twin pairs; in DZ twins, the genetic factors are correlated 0.5 on average. In both MZ and DZ twins, the common environmental factors are correlated 1.0, and the unique environmental factors are uncorrelated.

implies that familial resemblance is entirely caused by shared genes. Next, it was tested if the latent G, C, and E factors that are expressed only at age 7 could be omitted from the model (i.e., no new genetic or environmental influences at age 7) and whether the genetic or environmental covariances could be constrained at zero (i.e., genetic and environmental influences are occasion specific).

Parameters h, c, and e, representing the influence of G, C, and E on the phenotype, were estimated by maximum likelihood, using the computer program Mx (Neale, 1997). With missing data, as for the 17 twin pairs that did not participate the second time, the data cannot be summarized in dispersion matrices, but the model has to be fitted directly to the raw data. Mx allows the likelihood of each pedigree to be calculated separately and maximized over all available pedigrees. The likelihood of the bivariate genetic models described previously was compared to the likelihood of a saturated model in which for all five Sex × Zygosity groups, a 4 × 4 covariance matrix and a 4 × 1 vector of mean IQ scores was estimated. Goodness of fit of models estimating fewer parameters than the 70 parameters that

were specified in the saturated model was assessed by likelihood-ratio chi-squared tests. Submodels were compared by subtracting –2*log-likelihood for the full model from that for a reduced model. This difference is distributed as a chi-square. The degrees of freedom for this test are equal to the difference between the number of estimated parameters in the full model and the number in the submodel.

RESULTS

Table 1 gives mean IQ scores and standard deviations for first- and second-born twins (for boys and girls for opposite-sex twins) at ages 5 and 7 for each Sex × Zygosity group. There were no significant differences between boys and girls in total IQ at either age 5 or age 7. There also were no significant differences in intelligence between MZ and DZ twins or between first- and second-born twins. There was no effect of age on average IQ, but at age 7 the variance in IQ scores was somewhat larger than at age 5 (at age 5, $SD = 13.2$; at age 7, $SD = 14.7$; $\Delta\chi^2 = 6.03$ with 1 *df*). The estimate for IQ in the total sample was 102.7 at age 5 and 102.3 at age 7. In a normative sample of 5-year-old singleton children, the short version of the RAKIT had a mean of 102.6 ($SD = 14.2$). In 7-year-old singletons, the mean RAKIT score also was 102.6 ($SD = 13.8$). The mean IQ scores for twins in our sample clearly did not differ from those in singletons.

Resemblances between MZ and DZ twins for IQ measured at ages 5 and 7 are presented in Table 2, which gives both the correlations between twins and the 80% confidence intervals around these correlations (Miller & Neale, in press). The resemblances between MZ twins were larger than those between DZ twins, but for female twins at age 5, the difference between the MZ and the DZ correlation was small. For twins of opposite sex, the correlations were of the same magnitude as

TABLE 2
Maximum-Likelihood Estimates of Twin Correlations, Twin Cross Correlations, and Within-Person Correlations for IQ at Ages 5 and 7

	Twin Correlations				*Twin Cross Correlations*[a]		*Within-Person Correlations*[b]	
	5 Years		*7 Years*					
MZM	.76	(.66–.83)	.59	(.44–.71)	.65	(.53–.74)	.65	(.54–.74)
DZM	.52	(.35–.65)	.42	(.24–.57)	.31	(.16–.45)	.59	(.48–.68)
MZF	.77	(.68–.83)	.77	(.68–.84)	.65	(.55–.74)	.69	(.60–.77)
DZF	.72	(.61–.81)	.51	(.33–.66)	.46	(.31–.59)	.67	(.56–.75)
DOS	.58	(.43–.70)	.55	(.39–.68)	.45	(.31–.58)	.62	(.51–.71)

Note. Eighty percent confidence intervals around estimates are given within parentheses.
[a]IQ 5 is measured in one child and IQ 7 is measured in the other child. [b]IQ 5 with IQ 7.

those for DZ same-sex twin pairs, indicating that the same genetic and shared environmental influences are expressed in boys and girls. A different picture is offered by the cross-correlations of IQ measured at age 5 in one member of a pair with IQ measured at age 7 in the other twin. The MZ cross-correlations were larger than the DZ cross-correlations, indicating that most of the covariance between IQ assessed at ages 5 and 7 must be genetically mediated. In fact, the cross-correlations in MZ twins were of the same magnitude as the within-persons correlation between IQ measured at ages 5 and 7.

Model fitting to the raw twin data, summarized in Table 3, showed that in comparison with the fully saturated model, the more parsimonious bivariate genetic model provided a good fit to the data. There were no gender differences in genetic architecture, as can be seen in Table 3 by comparing the likelihood for Model 3 (GCE, no gender differences) with the likelihood for Model 2 (GCE, gender differences). Leaving out C (Model 4, no common environmental influences) and leaving out G (Model 5, no genetic influences) lead to significantly worse fitting models, indicating that both genetic and common environmental effects were required to explain familial resemblances in IQ. Unique environmental influences were age specific, as was indicated by the nonsignificant chi-square for Model 6, in which the path-coefficient representing the influence of the first unique environmental factor on the second IQ assessment was constrained at zero. In contrast, constraining the genetic or common environmental covariances at zero (Models 7 and 8) gave a significant chi-square, indicating that the genetic and common environmental factors that influenced IQ at age 5 also were of importance at age 7. At age 7, no new genetic or common environmental effects were expressed. This is indicated by the nonsignificant increase in chi-square for the models that specified

TABLE 3
Model Fitting of Twin IQ Data Measured at Ages 5 and 7

Model	*–2*Log-Likelihood*	*Estimated Parameters*	*Tested Against*	χ^2	*df*
1. Saturated model	6059.06	70			
2. GCE, gender differences	6101.89	22	1	42.83	48
3. GCE, no gender differences	6107.88	13	2	5.99	9
4. GE, no gender differences	6126.15	10	3	18.27[a]	3
5. CE, no gender differences	6119.93	10	3	12.05[a]	3
6. No E covariance of ages 5, 7	6109.92	12	3	2.04	1
7. No G covariance of ages 5, 7	6142.94	11	6	33.02[a]	1
8. No C covariance of ages 5, 7	6116.35	11	6	6.43[a]	1
9. No new G at age 7	6109.93	11	6	0.01	1
10. No new G, C at age 7	6112.08	10	9	2.15	1

Note. G = genetic; C = common environmental; E = unique environmental.
[a]Significant increase in –2*log-likelihood, implying a significantly worse fitting model.

no new genetic variance at age 7 (Model 9) and no new common environmental variance at age 7 (Model 10). The best model thus specified the stability of IQ assessed at ages 5 and 7 to be due to stability of both genetic and common environmental influences. In fact, there was no evidence for new genetic or new common environmental influences being expressed at age 7. The unique environmental factors, which also include measurement error, acted occasion specifically.

Parameter estimates from the best-fitting model (Model 10) are given in Table 4. From the estimates for the components of variance, it is clear that the increase in heritability at age 7 as compared to age 5 is due both to an increase in the total genetic variance as well as to a decrease in the amount of variance explained by common environmental factors. Table 4 also gives the standardized parameter estimates and their 80% confidence intervals. By comparing the heritabilities and their confidence intervals at ages 5 and 7, it may be seen that the relative influences of genetic factors are indeed different at these ages. Although the heritability at age 5 may be as high as 42%, and the heritability at age 7 as low as 50%, the nonoverlapping confidence intervals indicate a significantly larger heritability at age 7. Likewise, the relative influence of the shared family background is smaller at age 7 than it is at age 5. The correlation between intelligence scores at ages 5 and 7 was 0.65 in our sample. In the bivariate model, the covariance in IQ between ages 5 and 7 was partitioned into a genetic and an environmental part. The estimates in Table 4 show that the largest part (64%) of the covariance is explained by stable genetic influences; the smaller part (36%) is explained by the stability of shared environmental background.

TABLE 4
Genetic, Common Environmental, and Unique Environmental Variances; Genetic and Common Environmental Covariances; and Standardized Parameter Estimates From Best-Fitting Model

	G		*C*		*E*	
Total (co)variances						
Variance, 5 Years	46.81	(28.1–72.6)	85.83	(57.3–112.3)	40.74	(35.1–47.4)
Variance, 7 Years	134.87	(108.3–162.0)	22.42	(4.3–48.3)	60.75	(51.2–72.4)
Covariance, 5–7 Years	79.46	(56.8–105.59)	43.86	(15.9–72.2)	—	
Standardized estimates						
Variance, 5 Years	.27	(.16–.42)	.50	(.35–.60)	.23	(.20–.28)
Variance, 7 Years	.62	(.50–.71)	.10	(.02–.21)	.28	(.23–.34)
Covariance, 5–7 Years	.64	(.45–.86)	.36	(.14–.55)	—	

Note. Eighty percent confidence intervals around estimates are given within parentheses. Covariances are measured between ages 5 and 7. The best model indicated no covariance due to unique environmental (E). G = genetic; C = common environmental.

DISCUSSION

We found differences in heritability and in the influence of the shared family environment on individual differences in IQ measured at ages 5 and 7 years. At age 5, the influence of genetic factors was relatively small, and the influence of common environment accounted for half of the variance in IQ. At age 7, the influence of genetic factors increased, and the influence of common environment decreased. Genetic influences persisted from age 5 to age 7, without evidence of new genetic influences being expressed, and greatly gained in relative importance during this period. The shared family background influenced IQ in early childhood, and these effects also persisted until age 7, but their relative importance became much smaller. Unique environmental influences were occasion specific and accounted for roughly a quarter of the total variance in IQ at ages 5 and 7.

As far as we know, all developmental genetic analyses of childhood IQ have been carried out in samples of U.S. twins and adoptees. The results reported in this article provide an addition to the existing literature in a new, non-United States sample. This study of intelligence in Dutch twins is, for the most part, consistent with findings from American studies. IQ data from the longitudinal Louisville twin study (Eaves et al., 1986; Wilson, 1983) also indicate stability of the genetic influences as children grow older and find that the effects of genes that influence IQ during infancy are amplified later in life. Adoption studies are in agreement with twin studies in demonstrating a decreasing influence of shared environment on IQ and intellectual attainment as children grow up (Loehlin et al., 1989; Scarr & Weinberg, 1978). These results differ somewhat from those reported by Cherny and Cardon (1994). In the Colorado adoption study, no effects of family background were found, and an important contribution of new genetic influences to IQ variance was seen at age 7. It might be that part of these genetic innovations stem from a change in the IQ test that was employed at age 7.

Not all behaviors in infancy and childhood show low heritabilities or a substantial contribution of family environment. For example, in a large group of twins from the Netherlands Twin Register, we found that problem behaviors in 3-year-old twins are highly heritable (Van den Oord, Verhulst, & Boomsma, 1996). The influence of a shared family background was small for externalizing behaviors and completely absent for internalizing problems. In adolescent Dutch twins (Boomsma & Koopmans, 1994) and international adoptees (Van den Oord, Boomsma & Verhulst, 1994), heritability for the same dimensions of behavioral problems was lower than in childhood.

The environment surrounding a child continually enlarges as the child grows older. Apparently, for some traits this has the effect of decreasing their heritability, whereas for other traits such as IQ, heritability increases in spite of a broader range of environmental influences. Animal studies also have found that genetic variation in some behaviors develops postnatally (Scott, 1990). Scarr and Weinberg (1983) interpreted the results for intelligence as meaning that as children grow older, they

escape the influence of their parents and select their own environments. This process may be viewed as an active form of genotype–environment interaction, in which choices between environments are influenced by genetic differences between individuals. At age 7, genetic differences between individuals were the most important source of phenotypic differences in IQ; at age 5, the most important influence on intelligence in our sample consisted of shared environmental factors. These may include the influence of perinatal effects and gestational age on IQ. It is not unlikely that the importance of these influences on IQ diminishes after early childhood.

Differences in behavior, including cognition and intelligence, are to varying degrees the result of differences in nervous system functioning. In this same sample of twins, we have shown that individual differences in background EEG (van Baal, De Geus, & Boomsma, 1996) and in event-related brain potentials (ERP–P3) are highly heritable both at ages 5 and 7 (van Baal et al., this issue). Although it is difficult to relate neural and behavioral functioning (Benno, 1990), these results might suggest that high heritabilities of indexes of nervous system functioning precede the high heritabilities that are observed for IQ later in life. Plomin (1986) suggested that slight differences in neuroanatomy or neurophysiology early in life could cascade into increasingly larger behavioral differences among children as they grow older. We found evidence that for IQ, the same genes are expressed at ages 5 and 7, and that genetic differences that have a relatively small influence at age 5 are magnified as development proceeds. This finding supports the amplification theory for cognitive development (DeFries et al., 1987), in which genetic differences among infants at an early age only result in small functional differences due to, for example, their limited information-processing capabilities and are manifested to a greater extent as children get older.

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