

5

Current Status and Future Prospects in Twin Studies of the Development of Cognitive Abilities: Infancy to Old Age

D.I. BOOMSMA

Department of Psychonomics, Vrije Universiteit, De Boelelaan 1111,
1081 HV Amsterdam, The Netherlands

ABSTRACT

In this chapter, structural models for the genetic analysis of longitudinal data are introduced and several generalizations discussed that pertain to the estimation of genetic and environmental individual scores and mean trends. Cross-sectional and longitudinal twin and adoption studies of cognitive development are reviewed. The most important changes in the genetic architecture of IQ that can be observed over time are an increase in heritability from infancy to childhood and a decrease in common environmental influences during adolescence. From age 6 onward, heritability for general intelligence is around 50%, and the high longitudinal stability for IQ seems largely mediated by genetic factors.

INTRODUCTION

There may be a priori reasons to expect age-dependent changes in the contributions of genetic and environmental effects to individual IQ differences. Scarr and Weinberg (1978), for example, expect developmental differences in the size of environmental influences. Younger children may resemble their parents more on environmental grounds before they enter schools and other social institutions, and the influence of genetic factors may increase as they grow older. Of course, some want to deny the role of heredity at any age, either for emotional reasons (e.g., John Stuart Mill who wrote "Of all the vulgar modes of escaping from the consideration of the effect of social and moral influences upon the human mind, the most vulgar is that of attributing the

diversities of conduct and character to inherent natural differences” [in Gould 1980]) or for lack of convincing evidence (e.g., Roubertoux and Capron 1990). According to Nash (1990), the absence of genetic influences on intelligence is highly unlikely because of the need to account for the evolution of cognitive processing capabilities. Without some genetic variation, it is impossible to understand how the evolution of functional brain structures involved in cognitive performance could have occurred.

During development, changes in a quantitative trait may be due to distinct subsets of genes turning on and off, whereas continuity may be due to stable environmental causes. Contrary to popular points of view, genetically determined characters are not always stable, nor are longitudinally stable characters always influenced by heredity (Molenaar et al. 1991). In this chapter I discuss developmental models that are concerned with the disentanglement of genetic and nongenetic causes of stability and change. Two important generalizations of the multivariate extension of these models concern (a) the estimation of genetic and environmental time-dependent profiles for individual subjects and (b) the inclusion in the model of genetic and environmental mean trends.

The literature review of genetic studies of cognitive development addresses the following questions:

1. Are heritabilities for cognitive abilities age specific?
2. How are genetic and environmental processes involved in stability and change in individual differences in intelligence and specific cognitive abilities?
3. Are changes in the environmental contributions to individual differences in intelligence informative as to how environment shapes cognitive development?

Twin Analyses in General

The classical twin study of monozygotic (MZ) and dizygotic (DZ) twins does not permit simultaneous estimation of additive (A) and nonadditive or dominant (D) genetic effects, common (C) or between-family environmental and individual (E) or within-family environmental influences (see Hewitt 1989 and references to the work of Eaves and colleagues therein). If nonadditive genetic effects are present, a model in which they are not specified will overestimate A and underestimate C. If there is assortative mating, common environmental effects will be overestimated. However, a two-parameter AE model will be rejected if $C > 2D$ or if $C < 0.5D$. Equally, an environmental EC model will fail in the presence of A or D. Hewitt (1989) summarizes the value of a twin study as follows: “... it leads to testable hypotheses about appropriate variance decomposition for a particular measurement or multivariate set of measures, it permits a test of sex differences in the expression of genetic and environmental influences, allows us to test causal hypotheses for the relationships between variables in both cross-sectional and longitudinal designs, and when augmented by other family members provides the nucleus for exploring issues as wide

ranging as the mechanisms of marital assortment and marital interaction through to the estimation of and control for rater bias.”

Use of Twins in Intelligence Research

Are twins representative of singletons, although it has been claimed that they have a lower average IQ? According to Storfer (1990), lower average IQ scores of (identical) twins can be explained by their lower birth weights. The heavier twin of a pair is likely to have an IQ score equal to that of a nontwin of comparable birth weight and gestational age. Moreover, the twin disadvantage may disappear with age. Kallmann et al. (1951) present results from white twin pairs aged 60 or more who do as well as a standardization group aged 50–54 on Wechsler subtests.

Intelligence Tests

Genetic studies of cognition have almost exclusively used traditional IQ tests, and specific abilities have usually been defined as subtests. Results from tests developed from other perspectives, e.g., Piaget or information-processing, are scarce. The most frequently employed general intelligence tests are the Wechsler, the Stanford-Binet, Raven's Progressive Matrices, and the Bayley scales. The Wechsler consists of three tests: the WPPSI for children aged 4 to 6.5, the WISC for ages 7 to 16, and the adult WAIS. All three have 11 subscales, divided into performance and verbal tests, that are similar but not identical at different age levels. The Stanford-Binet is most suitable for ages 4 to 17 and gives an intelligence score that is heavily weighted with verbal abilities. For infants 1 to 30 months, the Bayley Mental Development Index and the Bayley motor scales are frequently used. A group intelligence test based on figure-analogy that is suitable for both children (from 5 years on) and adults is the Raven's Progressive Matrices test, developed by J.C. Raven and the geneticist L.S. Penrose.

Scores for IQ tests are usually constructed to be normally distributed with the same mean and standard deviation in each age group. Information on growth in means and variances is thus lost. Alternative ways of scoring standard tests have sometimes been considered. McArdle (1988) analyzed longitudinal WISC scores from children aged 6, 7, 9, and 11. An analysis of percentage correct scores clearly shows an increase in both means and variances with age.

ARE HERITABILITIES FOR COGNITIVE ABILITIES AGE SPECIFIC?

Bouchard and McGue (1981) have summarized IQ correlations obtained in family and adoption studies. The pattern of correlations strongly suggests polygenic inheritance without consistent sex differences. Comparing parent-offspring and twin correlations

may offer a first suggestion about age-dependent genetic and environmental effects. As explained in Plomin et al. (1988), significant parent-offspring resemblance implies significant heritabilities, both in childhood and adulthood, and a substantial genetic correlation across time. Average weighted MZ ($N = 4672$ pairs) and DZ ($N = 5546$) correlations are 0.86 and 0.60. Doubling the difference between MZ and DZ correlations gives a heritability estimate close to 50%. The parent-offspring correlation for parent and offspring reared in the parental home equals 0.42 ($N = 8433$) and the correlation of adoptive parent and offspring is 0.19 ($N = 1397$). Doubling the difference between these correlations gives an estimate of heritability of 46%, so that at first glance little evidence seems to exist for intergenerational differences in heritability. In the few studies in which parents and offspring received the same IQ test at the same age, correlations are not higher than the average parent-offspring correlation reported by Bouchard and McGue. Vroon and Meester (1986) observe a correlation of 0.34 for Raven's Progressive Matrices in 2847 father-son pairs tested by the Dutch army. McCall (1970) reports IQ correlations on parents and children ($N = 35$) who were both tested from age 3.5 to 11. Parent-offspring correlations are much lower, with a median value of 0.29, than sibling correlations from the same study (median $r = 0.55$, $N = 100$). The twin results are suggestive of common environmental influences. In twin studies, however, these cannot be distinguished from the effect of assortative mating, which is quite substantial for IQ. Bouchard and McGue (1981) report a spouse correlation of 0.33 ($N = 3817$). However, the adoptive parent-offspring correlation of 0.19 also indicates the presence of common environmental influences on IQ.

A developmental meta-analysis of twin similarities in personality and intelligence was published in 1990 by McCartney et al. (1990). The average MZ and DZ correlations for total IQ from 42 studies are 0.81 and 0.57, closely resembling the values reported by Bouchard and McGue (1981). Average correlations for specific cognitive abilities (verbal, quantitative, and performance) show the same pattern as for total IQ, with the exception of perception, where MZ and DZ correlations from 11 studies average 0.55 and 0.45. To study age as a moderator of twin resemblances, mean age from each study was correlated with the intraclass correlations from that same study. For total IQ and specific abilities, except verbal IQ, DZ twins become more dissimilar over time than MZ twins. For total IQ (results from 16 independent studies), the correlations of twin resemblance with age are 0.15 for MZ and -0.25 for DZ twins. Decrease in twin similarities is largest for perception IQ: -0.64 for MZ and -0.79 for DZ twins (results based on five studies). Correlating estimates for heritability, common and unique environment with mean age shows correlations of 0.36, -0.37 , and -0.15 for total IQ (no separate results are given for specific abilities). Excluding studies where mean age is less than 5 years yields even higher correlations (0.52, -0.50 , and -0.28). These last analyses, however, must be viewed with caution, because components of variance are less reliable than the intraclass correlations on which they are based, and the analyses are carried out on few data points (exactly how many is unclear). There is no accepted significance test for these correlations, and according to McCartney et al. (1990) they should be interpreted as effect-size

estimates. An analysis of age contrasts that allows for significance testing, however, shows only one significant age contrast for MZ twins (for performance IQ) and one for DZ twins (for quantitative IQ), although most contrasts for specific abilities are negative. This means that there is decreasing concordance between twins as they get older. For total IQ, only the DZ age contrast is negative. These results suggest that heritability for IQ increases over time, although the effects do not seem very large.

McCartney et al. (1990) recognize that differences between age groups can result from either age or cohort effects. Heath et al. (1985) addressed the question of cohort effects on education data in Norwegian twins born between 1915 and 1960 and their parents. Sundet et al. (1988) analyzed cohort effects on general ability in male twins from the same country, born between 1931 and 1960. For males educational attainment is subject to secular change, showing an increase in heritability. Results for general ability and for females offer no evidence for cohort effects.

DeFries et al. (1976) review studies of specific cognitive abilities. Evidence from six medium-sized U.S. studies and a Swedish twin study suggests that heritability decreases in spatial, vocabulary, word fluency to arithmetic speed, and reasoning abilities, while in parent-offspring studies verbal IQ seems more heritable than performance IQ. Plomin (1986, 1988) summarizes genetic studies of IQ and specific cognitive abilities from infancy to senescence. In infancy, heritabilities are low (15%), while the influence of common environment is large. This result, however, was mainly based on studies with the Bayley Mental Development Index, which some argue is not a good indicator of general intelligence (Bornstein and Sigman 1986; Storfer 1990). On the other hand, animal studies have also found that genetic variation in behavior develops postnatally (Scott 1990). From age 6 onward—for which age Wilson (1983) finds MZ and DZ correlations of 0.86 and 0.59—heritability estimates for IQ are 50% in adolescence and in adulthood. Heritabilities for specific abilities may increase during adolescence. Fischbein (1979) applied verbal and inductive tests to male twins at ages 12 and 18 and mathematical tests to twins of both sexes at ages 10 and 13. Although the same twins were measured twice, no bivariate analyses are given. Correlations for verbal ability increase slightly from 0.70 to 0.78 for MZ and decrease from 0.60 to 0.50 for DZ. For inductive tests, correlations increase for both MZ (0.59 to 0.78) and DZ twins (0.46 to 0.56). For mathematical abilities differences between MZ and DZ correlations increase (from 0.08 to 0.21 for boys and from -0.04 to 0.14 for girls). For boys, heritabilities thus seem to increase from around 20% to 40%. It has been suggested that heritabilities increase in old age; however, only three studies of aging twins have been conducted. Kallmann et al. (1951) studied 120 twin pairs aged 60 or more on WAIS subtests, Stanford-Binet Vocabulary, and a tapping test. Their between/within pair F ratios may easily be converted into intraclass correlations. Most measures show heritabilities around 50%, with the possible exception of memory tasks, where heritabilities seem lower. Plomin (1986) reports a similar result for memory tests in childhood and adolescence. Swan et al. (1990) studied 267 aging male twins (mean age 63 years). Two cognitive screening tests were administered: the Iowa Screening Battery for Mental Decline ($r_{MZ} = 0.47$, $r_{DZ} = 0.36$) and

the Mini-Mental State examination ($r_{MZ} = 0.51$, $r_{DZ} = 0.24$), which correlates reasonably highly with verbal IQ. Subjects also received the Digit Symbol subtest from the WAIS ($r_{MZ} = 0.72$, $r_{DZ} = 0.50$). Heritability estimates that take into account differences in variances between zygositys were 22%, 38%, and 76%, respectively. Pedersen et al. (1992) report resemblances in Swedish twins with an average age of 65 years. The sample consisted of MZ pairs reared together ($N = 67$) and apart ($N = 46$, separated before 11 years of age), and DZ twins together ($N = 89$) and apart ($N = 100$). For a principal component score based on 13 subtests, MZa and MZt correlations were 0.78 and 0.80, DZa and DZt correlations were 0.32 and 0.22. These correlations suggested a broad heritability of 80%, which includes nonadditive genetic variance. Correlations for subtests are lower, and average heritabilities for verbal, spatial, perceptual speed, and memory were 58%, 46%, 58%, and 38%. Taken together, these studies do not suggest large changes in heritabilities as people grow older.

Correlates of Intelligence

Galton (1883) was the first one to propose that reaction time (RT) is correlated with general intelligence and may be used as a measure of it. Vernon (1991) reports correlations of -0.44 between IQ and RT from 2 studies that also measured nerve conduction velocity (NCV), which is a measure of the speed with which electrical impulses are transmitted by the peripheral nervous system. Correlations of RT and NCV were -0.28 and -0.18 , while IQ and NCV correlated at $r = 0.42$ and 0.48 . No twin studies of NCV are available; however, Reed (1984) found NCV heritable in mice. He suggests that this forms a sufficient basis for asserting that there is genetic determination of variation in human intelligence. Twin studies suggest heritabilities for RT that are of the same magnitude as those for IQ. McGue and Bouchard (1989) observed heritabilities of 54% and 58% for basic and spatial speed factors in a sample of MZ ($N = 49$) and DZ ($N = 25$) twins reared apart. For a general speed factor based on eight complex RT tests, Vernon (1989) found a heritability of 49% in 50 MZ and 52 DZ twins. Vernon also found that RT tests requiring more complex mental operations show higher heritabilities. A bivariate analysis of these data with IQ in 50 MZ and 32–SS DZ pairs (15 to 57 years) was reported by Baker et al. (1991). Phenotypic correlations of verbal and performance IQ with general speed were both -0.59 and were entirely mediated by genetic factors. Genetic correlations were estimated to have absolute values of 0.92 and 1.0. Rose et al. (1981) estimated heritability as 76% for a perceptual speed measure in 74 MZ and 127 DZ college-aged twins and genetic half-siblings (MZ twin offspring). Boomsma and Somsen (1991) measured RT in 12 MZ and 12 DZ adolescent twins. For choice RT higher heritabilities (20%) were seen for shorter than for longer (7%) interstimulus intervals. Heritabilities of almost 50% were seen for RT measured in double task trials. Ho et al. (1988) analyzed WISC-IQ and speed measures in 30 MZ and 30 DZ pairs (8–18 years). Speed measures were rapid automatic naming and symbol-processing factors. Heritabilities for these factors are 0.52 and 0.49. Multivariate results indicate that the correlation

between IQ and speed measures (both r 's 0.42) is mainly due to genetic correlations of 0.46 and 0.67. Willerman et al. (1979) correlated problem-solving speed in parents with WISC subtests and IQ scores of adopted (average age 8.3) and natural (average age 9.9) children. Mother-child correlations were low; however, father–natural-child correlations were always higher than father–adopted-child correlations: for block design these correlations are 0.24 and 0.17; for object assembly 0.27 and 0.16; and for PIQ 0.31 and 0.18. Other biological correlates of intelligence that are at least partly genetically determined include evoked potentials, glucose uptake in the brain, pupillary dilatation during mental activity, myopia, allergies and other immune disorders, left-handedness, and uric acid levels (Storfer 1990).

CONTINUITY AND CHANGE IN INDIVIDUAL IQ DIFFERENCES

Special issues of the journals *Child Development* (1983, vol. 54, nr. 2) and *Behavior Genetics* (1986, vol. 16, nr. 1) give overviews of the most important current longitudinal studies with twins and adoptees and of developments in the field of longitudinal structural equation modeling.

Developmental Genetic Models

In longitudinal studies, the same set of variables is measured repeatedly over time on the same subjects. The correlation matrix of such data often displays a simplex pattern, that is, a simple order of complexity, where correlations are maximal near the main diagonal (i.e., among adjoining occasions) and decrease as the time between measurements increases. Such a structure was already noticed around the turn of this century by Pearson and co-workers with respect to physical space relations and was called the “Rule of Neighborhood” (Guttman and Guttman 1965). A simplex pattern can be generated by a first-order autoregressive process, where the partial correlation $r_{ik,j} = 0$, whenever $i < j < k$.

In addition to autoregressive models, so-called growth curve models may be used to analyze repeated measures. Growth curve models often include both the mean trend and the covariance structure. From the perspective of the covariance structure (discarding the mean trend), the growth curve model can be viewed as a confirmatory common factor model in which individual scores are determined by a constant base of “true” or common factor scores (Kenny and Campbell 1989). The growth curve model has the following form:

$$y(t) = \eta(0) + \lambda(t) \times \eta(1) + \varepsilon(t) , \quad (5.1)$$

where $y(t)$ is the observed score at occasion t . The scores on the latent common factors $\eta(0)$ and $\eta(1)$ represent the true scores that are constant over time.

In autoregressive models, by contrast, random change within the true score is introduced at each time. The true score continually changes—either increasing or decreasing—making adjacent time periods more similar than more remote ones. It can be written as (discarding the subject subscript to ease presentation):

$$y(t) = \eta(t) + \varepsilon(t), \text{ and } \eta(t) = \beta(t) \times \eta(t-1) + \zeta(t), \quad (5.2)$$

where $y(t)$ is the observed score measured from its mean at time t , $\beta(t)$ is the autoregressive coefficient, which is the correlation between $\eta(t)$ and $\eta(t-1)$ if the variables are standardized. $\eta(t)$ is the true score at occasion t that is subject to change over time because it depends both on the previous occasion and on a random innovation term $\zeta(t)$. The stability of individual differences over time, which can be expressed as the correlation between the variables $\eta(t)$ and $\eta(t+1)$, equals

$$\text{cor} [\eta(t), \eta(t+1)] = [\beta(t+1)] \times \text{var} [\eta(t)] / SD [\eta(t)] \times SD [\eta(t+1)]. \quad (5.3)$$

Thus stability depends on $\beta(t+1)$, and on the variances of $\eta(t)$ and $\zeta(t+1)$, because $\text{var} [\eta(t+1)] = \beta^2(t+1) \times \text{var} [\eta(t)] + \text{var} [\zeta(t+1)]$.

It is important to realize that, if variables are not standardized, β may be greater than one and this may lead to misleading interpretations of stability. In this model, parameters are invariant across persons, but may change over time. In a time-series model, where a single person or a few people are measured at many occasions, parameters may differ between persons, but are invariant across time. Only in this last model must the absolute value of β be less than or equal to one.

The autoregressive simplex model can be generalized to the genetic analysis of longitudinal data (Boomsma and Molenaar 1987). Let Equation 5.2 define the latent genetic and environmental time series, and let the basic genetic model for the observations be:

$$P(t) = \lambda(t) \eta(t) + \varepsilon(t) = \lambda_g(t) G(t) + \lambda_e(t) E(t) + \varepsilon(t), \quad (5.4)$$

where $t = 1, \dots, T$ are the number of time points that need not be equidistant; P is the observed phenotype that can be univariate or multivariate; $G(t)$ and $E(t)$ are series of genetic and environmental factor scores that are uncorrelated; the λ s are loadings of observed variables on latent factors; and ε represents influences unique to each variable and individual. Estimates of λ , β , and the variances of ζ and ε can be used to construct individual genetic and environmental profiles across time by means of Kalman filtering (Boomsma et al. 1991). Such individual profiles enable the attribution of individual phenotypic change to changes in the underlying genetic or environmental processes. Simulations have shown that these individual estimates can be reliably obtained. Estimation of $G(t)$ and $E(t)$ permits identification of sources of underlying deviant development in individual subjects.

The role of genetic and environmental influences on average growth does not usually feature in behavior genetic studies. McArdle (1986) was one of the first to model phenotypic means in a longitudinal analysis of twin data. His model can be viewed as a restricted common factor model in which variation in level (L) and shape (S) factors is decomposed into second order, but zero mean, latent genetic and environmental factors. Means are modeled as:

$$E [P(t)] = E [L(t)] + w(t) E [S(t)] , \quad (5.5)$$

where $w(t)$ is the factor loading of $P(t)$ on $S(t)$ at occasion t . Application of this model to data from the Louisville twin study (Bayley scale at 6, 12, 18, and 24 months) shows a strong, largely linear change over age, strong common environmental and small genetic effects.

The model proposed by McArdle does not result in the decomposition of longitudinal means into a genetic and environmental part. Dolan et al. (1991) suggested an alternative that involves estimating the contribution of genetic and environmental factors to changes in means over time. The mean structure of a univariate time series is decomposed by the following model:

$$E [P(t)] = v + E [G(t)] + E [E(t)] , \quad (5.6)$$

where v is a constant intercept term. The latent means are in part attributable to the preceding occasion and in part independent thereof:

$$\begin{aligned} E [G(t)] &= \beta_g(t) E [G(t-1)] + \Delta_g \text{var} [\zeta_g(t)] \quad \text{and} \\ E [E(t)] &= \beta_e(t) E [E(t-1)] + \Delta_e \text{var} [\zeta_e(t)] . \end{aligned} \quad (5.7)$$

The same factors (G and E) contribute to both means and individual differences as is expressed in the dual function of the autoregressive coefficients (β) and the residual variance components ($\text{var} [\zeta]$). The Δ s are time-invariant coefficients of proportionality that relate the mean to the standard deviation of the innovations. The model can be tested by fitting it to the covariance structure with and without including the means. If the goodness of fit does not decline and the parameter estimates are stable, this is taken as support for the validity of the model.

Applications of Developmental Models to Longitudinal Data

In general, analyses of longitudinal data do not allow observed time series to be decomposed into more than one underlying series. Twin data are unique in that they do allow such a decomposition. An interesting feature of such data is also the possibility of simultaneously fitting factor and simplex structures to the data, e.g., a factor structure for common environmental influences and a simplex for the genetic process.

Eaves et al. (1986) present a general developmental model in which both factor and simplex processes are incorporated together with the possibility of phenotypic transmission. More general versions of this model have been presented subsequently (e.g., in Hahn et al. 1990). Application of the original model to cognitive data from the Louisville twin study (3 months through 15 years) shows initially small but persisting and accumulating effects of a single set of genes and an appreciable influence of common environmental effects that are also persistent as well as age-specific input. Unique environmental influences are occasion-specific.

Loehlin et al. (1989) tried to apply the Eaves et al. model to IQ data from two occasions approximately 10 years apart. On the first occasion, adoptive children were between 3 and 14 years old. Correlations for repeated testing were 0.66 for 258 adoptive and 0.70 for 93 biological children. Parents were measured once. A model with only phenotypic transmission gave a good fit and reasonable parameter estimates (Eaves et al. 1986; note that such a model is equivalent to specifying genetic and environmental transmission parameters to be the same). On the second occasion, no evidence for common environment was found. Heritability at time 1 was 26%, at time 2, 37%; however, Loehlin et al. (1989) do not want to put too much emphasis on numerical estimates. In fitting the model the genetic correlation of parents and offspring equals 0.5 at both times 1 and 2. This seems correct only if the genetic correlation between both occasions equals unity.

Wilson (1983) analyzes the pattern of spurts and lags in mental development by analysis of variance of repeated measures in twins. Two correlations are obtained from this analysis: one for the sum of the repeated measures (which is, however, seriously affected by autocorrelation) and one for the pattern of changes over time (i.e., the interaction of pairs \times occasions). Results from the Louisville twin study show that heritability for developmental profiles increases with age. The MZ-DZ difference at 3 to 12 months is only 0.07, but increases to 0.32 at years 8 to 15. This result does not reveal, however, how genes operate throughout development, as did the model-fitting approach of Eaves et al. (1986).

Plomin et al. (1988) combine IQ data from the Louisville study of twins measured at ages 1, 2, 3, and 4 and data on scholastic abilities in young adult twins from a study by Loehlin and Nichols with IQ data from adoptive and control children aged 1, 2, 3, and 4 and their parents in a longitudinal analysis. Estimates for twin-shared environment are high, yet transmission from parental phenotype to a child's environment is not significant. The heritability estimate in adults is 50%; in children it increases from 10%, 17%, and 18% to 26% at 1, 2, 3, and 4 years. Estimates for the genetic stability parameter from childhood to adulthood also increase from around 0.60 to 1 from ages 1 to 4. These results lend support to the developmental amplification model proposed by DeFries (in Plomin and DeFries 1985). In this model the effects of genes that are relevant to mental development during infancy and childhood are amplified during adulthood. Cardon et al. (1992) analyze Bayley, Stanford-Binet and Wechsler data from adopted and nonadopted siblings measured from 1 to 7 years and twins measured from 1 to 3 years. They find higher heritabilities than Plomin et al. (1988) and also

evidence for common environmental influences in siblings as well as twins. Heritabilities are estimated at 55%, 68%, 59%, 53%, and 52% for ages 1, 2, 3, 4, and 7 years; the influence of common environment is 10% at each age. The genetic part of the model shows increasing transmission parameters and substantial genetic innovations at all ages, except age 4. Common environment functions as a single, constant background factor.

Carey (1988) warns against simple interpretations of genetic correlations in terms of sets of genes common or specific to variables or times. Two occasions may have all their genes in common and show low genetic correlations, while systems with only a few genes in common can have high genetic correlations. Genetic correlations depend on the rank order of genic effects and the type of polygenic system. Carey suggests that we must distinguish between biological pleiotropism, in which the same genes physically underlie different traits and statistical pleiotropism, and in which allelic effects on one trait predict allelic effects on other characters.

New Infant Measures of General Intelligence

Bornstein and Sigman (1986) challenge the belief that there is little (phenotypic) association between cognitive performance in infancy and adulthood. Part of this belief stems from the fact that one of the most frequently used measures of infant IQ, the Bayley Mental Development Index, is a poor predictor of later IQ scores. The Bayley motor scales and the Gesell infant development scales also show no association with later IQ (Storfer 1990). Promising new measures of infant cognitive function are decrement of attention or habituation and recovery of attention or novelty preference. Bornstein and Sigman report 15% and 22% (r^2) common variance for cognition in childhood with habituation and novelty preference, respectively, in a large number of studies. Individual differences in habituation and novelty preference date from the earliest months of life; however, few studies have analyzed these differences. Bornstein and Sigman refer to an unpublished study in which maternal IQ correlated with attention at term in preterm infants. DiLalla et al. (1990) obtained measures of novelty preference in twins 7, 8, and 9 months. Midtwin scores were regressed on midparent WAIS-IQ. Significant regressions were observed for novelty preference at 9 but not at 7 months (β of 0.22 and 0.33 for immediate and retest at 9 months; these regressions are analogous to heritability estimates). A Bayley composite measure showed regressions of 0.13 and 0.06 at 7 and 9 months.

CHANGES IN ENVIRONMENTAL CONTRIBUTIONS AND SHAPING OF COGNITIVE DEVELOPMENT

Gottfried and Gottfried (1986) review ten longitudinal studies of home environment and cognition. They consider it an empirical fact that family environment correlates with young children's cognitive development. An advantaged home environment is

associated with higher SES, with being first-born, but not with sex of the child. Later, as compared to earlier, home environment measures are more highly correlated with cognitive development. After SES has been controlled for, correlations between home factors and cognitive development persist. However, SES also correlates with cognitive development independent of home factors. Storfer (1990) discusses environmental factors that may shape IQ in the upward direction. These include having an older father, being a single child, and child-rearing practices of Jewish and Japanese families. Plomin and DeFries (1985) compare relationships between environmental measures and Bayley scores in adoptive and control families. If heredity affects this relationship, correlations will be larger in control than in adoptive families. At 24 months, relationships are stronger than at 12 months and are mediated environmentally to a significant extent, although there also is genetic mediation. Environmental measures at 12 and 24 months predict Bayley scores at 24 months almost equally well. Plomin and DeFries find that heredity is not involved in this longitudinal relationship.

Gottfried and Gottfried (1986) also report high correlations between early home environment and academic achievement. This seems difficult to reconcile with the absence of common environmental influences in adoption studies of adolescents and adults. At ages 4 to 7, Scarr and Weinberg (1977) find correlations of 0.39 for adopted siblings ($N = 53$), 0.30 for adopted-natural pairs ($N = 134$) and 0.42 for biological siblings ($N = 107$). No resemblance in IQ was found, however, in adoptees aged 16–22 (Scarr and Weinberg 1978). A correlation of -0.03 was observed in 84 adopted sibling pairs, while the correlation for biological siblings was 0.35 ($N = 168$). Teasdale and Owen (1984) report data on adult (18–26 years) adoptees from a Danish adoption register. They find genetic but no common environmental influences on intelligence, whereas for educational attainment both factors are of importance. Loehlin et al. (1989) also conclude that in early adulthood there is no influence of common environment on IQ.

CONCLUSIONS AND FUTURE PROSPECTS

Heritability for cognition differs as a function of age. It increases from around 15% in infancy to around 50% at age 6 but does not seem to change very much during adolescence and adulthood. Not much can be said about genetic influences on IQ in old age because few studies are available. A genetic analysis of individual IQ differences in elderly subjects raises interesting problems because of the possibility of dealing with selected samples in which the selection process is directly associated with the dependent variable. In contrast to the increasing influence of heredity on IQ, the large influence of the shared family environment that is seen early in life rapidly decreases in adolescence. Wilson (1983), in his longitudinal study of twins, finds that common environment accounts for 70% of the variance at age 3 and for 20% at age 15.

The developmental course of genetic and nongenetic influences on cognition has been studied in infancy and childhood and results indicate that genetic factors are stable across time and that their influences are possibly amplified as children grow older. Beyond childhood we do not know what causes stability in intelligence, although parent-offspring data are suggestive of high genetic correlations across age. Most tests of specific cognitive abilities indicate significant genetic influence, with some evidence that heritability for memory is lower than for other abilities. A few studies suggest that intercorrelations between tests arise because of genetic covariance (e.g., Labuda et al. 1987). Martin et al. (1984), however, show that phenotypic correlations between subtests of the National Merit Scholarship Qualifying Test also arise because of a single underlying between-families environmental factor, and they suggest that a single dimension of mate selection or cultural inheritance accounts for a significant part of the phenotypic covariance.

It is almost completely unknown to what extent the relations between IQ and its biological correlates are genetically or environmentally mediated, and there is a clear need for multivariate analyses of twin data in this area (Vernon 1991).

Application of the techniques of molecular genetics are now being considered to look for multiple loci that affect quantitative traits such as intelligence (Plomin and Neiderhiser 1991). Twin data may be of use in this respect, as they offer the possibility to estimate individual genetic and environmental scores (Boomsma et al. 1990). Genetic scores can be used to investigate their relationship with RFLPs and other genetic markers. It is conceivable that the power of these types of analyses will greatly increase when genetic instead of phenotypic scores can be used.

In addition, knowledge about the reasons why certain subjects exhibit high phenotypic scores may be of practical interest. Risk assessment may be improved by the knowledge that a high phenotypic score is caused by a high genetic or a high environmental deviation.

ACKNOWLEDGEMENT

I would like to thank P.A. Vernon, C.V. Dolan, P.C.M. Molenaar, and N.G. Martin for their helpful comments.

REFERENCES

- Baker, L.A., P.A. Vernon, and H.Z. Ho. 1991. The genetic correlation between intelligence and speed of information processing. *Behav. Genet.* **21**:351–368.
- Boomsma, D.I., and P.C.M. Molenaar. 1987. The genetic analysis of repeated measures. I. Simplex models. *Behav. Genet.* **17**:111–123.
- Boomsma, D.I., P.C.M. Molenaar, and C.V. Dolan. 1991. Individual latent growth curves: Estimation of genetic and environmental profiles in longitudinal twin studies. *Behav. Genet.* **21**:241–253.

- Boomsma, D.I., P.C.M. Molenaar, and J.F. Orlebeke. 1990. Estimation of individual genetic and environmental factor scores. *Genet. Epidemiol.* **7**:83–91.
- Boomsma, D.I., and R.J.M. Somsen. 1991. Reaction times measured in a choice reaction time and a double task condition: A small twin study. *Pers. Individ. Diff.* **12**:519–522.
- Bornstein, M.H., and M.D. Sigman. 1986. Continuity in mental development from infancy. *Child Devel.* **57**:251–274.
- Bouchard, T.J. Jr., and M. McGue. 1981. Familial studies of intelligence: A review. *Science* **212**:1055–1059.
- Cardon L., D.W. Fulker, J.C. DeFries, and R. Plomin. 1992. Continuity and change in general cognitive ability from 1 to 7 years of age. *Devel. Psychol.* **28**:64–73.
- Carey, G. 1988. Inference about genetic correlations. *Behav. Genet.* **18**:329–338.
- DeFries, J.C., S.G. Vandenberg, and G.E. McClearn. 1976. Genetics of specific cognitive abilities. *Ann. Rev. Genet.* **10**:179–207.
- DiLalla, L.F., L.A. Thompson, R. Plomin, K. Phillips, J.F. Fagan, M.M. Haith, L.H. Cyphers, and D.W. Fulker. 1990. Infant predictors of preschool and adult IQ: A study of infant twins and their parents. *Devel. Psychol.* **26**:759–769.
- Dolan, C.V., P.C.M. Molenaar, and D.I. Boomsma. 1991. Simultaneous genetic analysis of longitudinal means and covariance structure in the simplex model using twin data. *Behav. Genet.* **21**:49–65.
- Eaves, L.J., J. Long, and A.C. Heath. 1986. A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behav. Genet.* **16**:143–162.
- Fischbein, S. 1979. Intra-pair similarity in IQ of monozygotic and dizygotic male twins at 12 and 18 years of age. *Ann. Hum. Bio.* **6**:495–504.
- Galton, F. 1883. *Inquiries into Human Faculty and Its Development*. London: MacMillan.
- Gottfried, A.W., and A.E. Gottfried. 1986. Home environment and children's development from infancy through school entry years: Results from contemporary longitudinal investigations in North America. *Child. Envir.* **3**:3–9.
- Gould, S.J. 1980. *Ever since Darwin: Reflections in Natural History*, 2nd ed. Harmondsworth: Penguin.
- Guttman, R., and L. Guttman. 1965. A new approach to the analysis of growth patterns: The simplex structure of intercorrelations of measurements. *Growth* **29**:219–232.
- Hahn, M.E., J.K. Hewitt, N.D. Henderson, and R. Benno, eds. 1990. *Developmental Behavior Genetics: Neural, Biometrical and Evolutionary Approaches*. Oxford: Oxford Univ. Press.
- Heath, A.C., K. Berg, L.J. Eaves, M.H. Solaas, L.A. Corey, J. Sundet, P. Magnus, and W.E. Nance. 1985. Education policy and the heritability of educational attainment. *Nature* **314**:734–736.
- Hewitt, J. 1989. Of biases and more in the study of twins reared together. *Behav. Genet.* **19**:605–608.
- Ho, H.Z., L.A. Baker, and S.N. Decker. 1988. Covariation between intelligence and speed of cognitive processing: Genetic and environmental influences. *Behav. Genet.* **18**:247–261.
- Kallmann, F.J., L. Feingold, and E. Bondy. 1951. Comparative adaptional, social and psychometric data on the life histories of senescent twin pairs. *Am. J. Hum. Genet.* **3**:65–73.
- Kenny, D.A., and D.T. Campbell. 1989. On the measurement of stability in over-time data. *J. Pers.* **57**:445–481.
- Labuda, M., J.C. DeFries, and D.W. Fulker. 1987. Genetic and environmental covariance structures among WISC-R subtests: A twin study. *Intelligence* **11**:233–244.
- Loehlin, J.C., J.M. Horn, and L. Willerman. 1989. Modeling IQ change: Evidence from the Texas adoption project. *Child Devel.* **60**:993–1004.

- Martin, N.G., R. Jardine, and L.J. Eaves. 1984. Is there only one set of genes for different abilities? A reanalysis of the National Merit Scholarship Qualifying Test (NMSQT) data. *Behav. Genet.* **14**:355–370.
- McArdle, J.J. 1986. Latent variable growth within behavior genetic models. *Behav. Genet.* **16**:163–201.
- McArdle, J.J. 1988. Dynamic but structural equation modeling of repeated measures data. In: *Handbook of Multivariate Experimental Psychology*, ed. J.R. Nesselroade and R.B. Cattell, pp. 561–614. New York: Plenum.
- McCall, R.B. 1970. Intelligence quotient pattern over age: Comparison among siblings and parent-child pairs. *Science* **170**: 644–648.
- McCartney, K., M.J. Harris, and F. Bernieri. 1990. Growing up and growing apart: A developmental meta-analysis of twin studies. *Psychol. Bull.* **107**:226–237.
- McGue, M., and T.J. Bouchard. 1989. Genetic and environmental determinants of information processing and special mental abilities: A twin analysis. In: *Advances in the Psychology of Human Intelligence*, vol. 5., ed. R. Sternberg, pp. 7–45. Hillsdale, NJ: Lawrence Erlbaum.
- Molenaar, P.C.M., D.I. Boomsma, and C.V. Dolan. 1991. Genetic and environmental factors in a developmental perspective. In: *Problems and Methods in Longitudinal Research: Stability and Change*, ed. D. Magnusson, L.R. Bergman, G. Rudinger, and B. Thorestad, pp. 250–273. Cambridge: Cambridge Univ. Press.
- Nash, R. 1990. *Intelligence and Realism: A Materialist Critique of IQ*. London: Macmillan.
- Pedersen, N.L., R. Plomin, J.R. Nesselroade, and G.E. McClearn. 1992. A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychol. Sci.* **3**:346–353.
- Plomin, R. 1986. *Development, Genetics and Psychology*. Hillsdale, NJ: Lawrence Erlbaum.
- Plomin, R. 1988. The nature and nurture of cognitive abilities. In: *Advances in the Psychology of Human Intelligence*, vol. 4., ed. R.J. Sternberg, pp. 1–33. Hillsdale, NJ: Lawrence Erlbaum.
- Plomin, R., and J.C. DeFries. 1985. *Origins of Individual Differences in Infancy: The Colorado Adoption Project*. New York: Academic.
- Plomin, R., J.C. DeFries, and D. Fulker. 1988. *Nature and Nurture during Infancy and Early Childhood*. Cambridge: Cambridge Univ. Press.
- Plomin, R., and J. Neiderhiser. 1991. Quantitative genetics, molecular genetics, and intelligence. *Intelligence* **15**:369–387.
- Reed, T.E. 1984. Mechanism for heritability of intelligence. *Nature* **311**:417.
- Rose, R.J., J.Z. Miller, and D.W. Fulker. 1981. Twin-family studies of perceptual speed ability: II. Parameter estimation. *Behav. Genet.* **11**:565–575.
- Roubertoux, P.L., and C. Capron. 1990. Are intelligence differences hereditarily transmitted? *Cahiers Psychol. Cogn.* **10**:555–594.
- Scarr, S., and R.A. Weinberg. 1977. Intellectual similarities within families of both adopted and biological children. *Intelligence* **1**:170–191.
- Scarr S., and R.A. Weinberg. 1978. The influence of “family background” on intellectual attainment. *Am. Sociol. Rev.* **43**:674–692.
- Scott, J.P. 1990. Foreword. In: *Developmental Behavior Genetics: Neural, Biometrical and Evolutionary Approaches*, ed. M.E. Hahn, J.K. Hewitt, N.D. Henderson, and R. Benno. Oxford: Oxford Univ. Press.
- Storfer, M.D. 1990. *Intelligence and Giftedness: The Contributions of Heredity and Early Environment*. San Francisco: Jossey-Bass Publ.
- Sundet, J.M., K. Tambs, P. Magnus, and K. Berg. 1988. On the question of secular trends in the heritability of intelligence test scores: A study of Norwegian twins. *Intelligence* **12**:47–59.

- Swan, G.E., D. Carmelli, T. Reed, G.A. Harshfield, R.R. Fabsitz, and P.J. Eslinger. 1990. Heritability of cognitive performance in aging twins. *Arch. Neur.* **47**:259–262.
- Teasdale, T.W., and D.R. Owen. 1984. Heredity and family environment in intelligence and educational level: A sibling study. *Nature* **309**:620–622.
- Vernon, P.A. 1989. The heritability of measures of speed of information-processing. *Pers. Individ. Diff.* **10**:573–576.
- Vernon, P.A. 1991. Studying intelligence the hard way. *Intelligence* **15**:389–395.
- Vroon, P.A., and A.C. Meester. 1986. Distributions of intelligence and educational level in fathers and sons. *Br. J. Psychol.* **77**:137–142 .
- Willerman, L., J.C. Loehlin, and J.M. Horn. 1979. Parental problem solving speed a correlate of intelligence in parents and their adopted and natural children. *J. Ed. Psychol.* **71**:627–634.
- Wilson, R.S. 1983. The Louisville twin study: Developmental synchronies in behavior. *Child Devel.* **54**:298–316.