Development genetic analysis of general cognitive ability from 1 to 12 years in a sample of adoptees, biological siblings, and twins

E.G. Bishop\textsuperscript{a}, Stacey S. Cherny\textsuperscript{b}, Robin Corley\textsuperscript{a}, Robert Plomin\textsuperscript{c}, John C. DeFries\textsuperscript{a}, John K. Hewitt\textsuperscript{a,*}

\textsuperscript{a}Institute for Behavioral Genetics, University of Colorado at Boulder, 447 UCB, Boulder, CO 80309-0447, USA
\textsuperscript{b}Wellcome Trust Centre for Human Genetics, Oxford, UK
\textsuperscript{c}Institute of Psychiatry, King’s College, London, UK

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Abstract

We report a longitudinal genetic analysis that combines twins and adoptive and nonadoptive siblings to investigate continuity and change in the etiology of general cognitive ability from infancy through the transition to adolescence. Research in childhood suggests that heritability increases and shared environmental influence decreases, that genetic factors contribute to change as well as continuity, that shared environment contributes entirely to continuity, and that nonshared environment contributes entirely to change. Twins from the Longitudinal Twin Study (LTS; 224 MZ pairs, 189 same-sex DZ pairs at 1 year) and adoptive (genetically unrelated) and nonadoptive (biological) siblings from the Colorado Adoption Project (CAP; 107 and 87 pairs, respectively, at 1 year) were assessed again at 2, 3, 4, 7, 9, and 10 years, and nontwin siblings were also assessed at 12 years. Longitudinal model fitting supported the above hypotheses derived from research in childhood with two exceptions. Nonshared environmental influences contribute to continuity as well as change in middle childhood. The most striking exception is that during the transition to adolescence, genetic factors no longer contribute to change, just to continuity.

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* Corresponding author. Tel.: +1-303-492-7362.
E-mail address: john.hewitt@colorado.edu (J.K. Hewitt).
1. Introduction

The psychometric approach to the measurement of cognitive abilities occupied researchers for the entirety of the 20th century, beginning with the work of Binet and Simon (1905) and their “holistic” view of intelligence and Spearman’s (1904, 1927) common underlying general factor or $g$, and on through to Cattell’s (1971) crystallized and fluid intelligence model, Sternberg’s componential theory (Sternberg, 1985) and information-processing approaches (Deary, 2000). A hierarchical organization with general cognitive ability at the top of the hierarchy is widely accepted as a psychometric model of cognitive abilities (Carroll, 1993). General cognitive ability is the trait for which we know the most from a behavioral-genetic prospective. Bouchard and McGue (1981) noted over 140 family, twin and adoption studies of general cognitive ability that converge on the conclusion of substantial genetic influence. Model-fitting analyses of these data suggest that genetic factors account for about half of the variance of general cognitive ability (Chipuer, Rovine, & Plomin, 1990; Devlin, Daniels, & Roeder, 1997; Loehlin, 1989).

Behavioral genetic data can go beyond estimating heritability to consider developmental change and continuity. The first developmental question is whether genetic and environmental parameter estimates change during development, a question that intrigued the first researchers in this area (Galton, 1876; Merriman, 1924). Because it is so reasonable to assume that genetic differences become less important as experiences accumulate during the course of life, one of the most interesting findings in this area is that genetic factors become increasingly important for $g$ throughout the life span (McCartney, Harris, & Bernieri, 1990; McGue et al., 1993; Plomin, 1995). Another important developmental finding is that the effects of shared environment are weak because shared environment is estimated indirectly by the twin method, the world’s twin literature indicates that shared environment effects are moderate in childhood but negligible in adulthood (McGue et al., 1993). The most direct test of the importance of shared environment comes from the resemblance of adoptive siblings, pairs of genetically unrelated children adopted into the same adoptive families. In childhood, the average adoptive sibling correlation is .25, but in adulthood, the correlation for adoptive siblings is near zero (Plomin, DeFries, McClearn, & McGuffin, 2000). In summary, from childhood to adulthood, heritability increases and shared environment decreases for general cognitive ability. The second interesting aspect of the developmental process involves age-to-age continuity and change for which longitudinal data are required. Researchers have hypothesized major changes in the structure of mental functioning across development (e.g., McCall, 1979). A pioneering study by Honzik, MacFarlane, and Allen (1948) found that prediction of IQ increased with age. For example, the correlation between ages 2 and 5 was only .32, but this correlation increased to .70 between ages 5 and 8 and .85 between 9 and 12. Other early studies also found little or no correlation between IQ measured in infancy and early and middle childhood (e.g., Anderson, 1939), although cognitive tasks, such as novelty preference, visual information processing, and cross-modal transfer, that emphasize information processing by young infants, may be better predictors of childhood IQ (Rose & Feldman, 1997; Rose, Feldman, & Wallace, 1992; Slater, 1995; Thompson, Fagan, & Fulker, 1991). By early adolescence, the long-term stability of IQ is
remarkable. A recent study of 101 individuals assessed at 11 years of age and again at 77 years yielded a correlation of .63 (Deary et al., 2000).

Genetic and environmental factors can contribute to change as well as continuity during development. Because there are so few longitudinal behavioral genetic studies of cognitive abilities, little is known about the etiology of genetic and environmental origins of change and continuity. Much of what is known comes from two major developmental studies conducted at the Institute for Behavioral Genetics, University of Colorado, Boulder: the Colorado Adoption Project (CAP; DeFries, Plomin, & Fulker, 1994; Plomin & DeFries, 1985; Plomin, DeFries, & Fulker, 1988), Colorado Adoption Project and the Longitudinal Twin Study (LTS; Benson, Cherny, Haith, & Fulker, 1993; DiLalla et al., 1990; Emde et al., 1992; Plomin et al., 1990, 1993). The general picture that emerges from analyses of these and other longitudinal data for general cognitive ability is that genes contribute to change as well as to continuity during childhood, that shared environment contribute almost entirely to change (Cardon, Fulker, DeFries, & Plomin, 1992; Cherny & Cardon, 1994; Cherny et al., 1994; Cherny, Fulker, & Hewitt, 1997; Fulker, Cherny, & Cardon, 1993). Similar inferences have been drawn from the Louisville Twin Study (Wilson, 1983). Combining longitudinal twin and adoption study data permits the most powerful applications of structural equation modeling to be brought to bear on the issue of behavioral development. The most recent analysis applied a longitudinal genetic model to these twin and adoptive sibling data from infancy to middle childhood and found evidence for genetic change especially at two important development transitions (Cherny et al., 1997). The first is the transition from infancy to early childhood, an age when cognitive ability rapidly changes as language develops. The second is the transition from early to middle childhood, around 7 years of age, which theories of cognitive development recognize as time of transition associated with the development of concrete operational thought, in Piagetian terms, accompanied by the increasing importance of formal academic tasks as the typical child completes his or her first year of formal schooling.

Twin and sibling comparisons are especially informative for analyzing age-to-age change and continuity because twins are the same age and siblings can be assessed at the same age. Parent–offspring comparisons provide a very different view of developmental processes because parents are assessed as adults and their offspring as children. For this reason, parent–offspring resemblance is diluted by any genetic change from childhood to adulthood. Nonetheless, parent–offspring analyses are consistent with the picture that emerges from the twin and sibling data. For example, in the longitudinal CAP, parent–offspring correlations for general cognitive ability increase from infancy through adolescence (Plomin, DeFries, McClearn, & Rutter, 1997). Correlations between parents and children for control (non-adoptive) families increase from <.20 in infancy to about .20 in middle childhood and to about .30 in adolescence. The correlations between biological mothers and their adopted-away children follow a similar pattern, thus indicating that increasing parent–offspring resemblance is due to increasing influence of genetic factors that are stable from childhood to adulthood. Parent–offspring correlations for adoptive parents and their adopted children hover around zero, which suggests that family environment shared by parents and offspring
does not contribute importantly to parent–offspring resemblance for \( g \). In contrast, twin and sibling adoption analyses indicate the early influence of family environment shared by children growing up in the same family.

The present study extends the combined twin and adoptive sibling longitudinal genetic analysis of Cherny et al. (1997) past middle childhood to adolescence. Because adolescence is uncharted territory, strong hypotheses about what might be found in the transition to adolescence are not possible. However, tentative hypotheses can be extracted from results obtained in childhood. For example, shared environmental influence might be expected to continue to decline in adolescence. What shared environment that exists might be expected to contribute to continuity rather than change in adolescence. Heritability might be expected to continue to increase and genetic factors seem likely to contribute to change as well as continuity. Whether the transition to adolescence yields any major genetic change like the transition from early to middle childhood is a question of major interest.

2. Methods

2.1. Subjects

2.1.1. CAP families

The adopted children are Caucasians with no known disabilities who were placed in foster care immediately after release from the hospital. The infants remained in the foster homes until the legal requirements concerning relinquishment were fulfilled; they were placed in the adoptive homes at an average of 28.5 days. Control families were matched to the adoptive families on the basis of the sex of the proband, number of children in the family, age of the father (±5 years), rating of father (±8 points) on an occupational rating scale developed by Duncan (Reiss et al., 1961), and years of education of the father (±2 years). The CAP adoptive and nonadoptive families are similar in terms of socioeconomic status and education. In addition, using national norms for the US white labor force, the CAP sample was found to be somewhat above the national average in terms of occupation, but nearly representative in terms of variance. In the CAP, selective placement for educational attainment, socioeconomic status, IQ, and other measures is negligible. Greater detail about the CAP sample is available elsewhere (DeFries et al., 1994; Plomin & DeFries, 1985). The number of adoptive and nonadoptive sibling pairs at each age are listed later (Table 2).

2.1.2. Twin families

The twin sample was ascertained by the Twin Infant Project component of the LTS (Emde et al., 1992). The Colorado Department of Health provided a monthly listing of all twin births in which both twins survived. Letters soliciting participation were sent to parents of twins via the Department of Health. About 40% of the contacted families agreed to take part in the study—clearly an element of self-selection took place, raising the question of representativeness of the parents in the sample. There is some selection in this volunteer sample by ethnicity—the percentages of minority families is 8% as compared to 15% in the population.
from which these families were drawn. This ethnicity is comparable to the CAP sample; from
the point of view of combining an existing adoption and sibling sample with a sample of twins,
this comparability is advantageous. The mothers in the twin sample have a mean educational
level of 14.1 years, with a range of 9–19; for the fathers, the mean is 15.0, with a range of 9–21.
National Opinion Research Center (NORC; Hauser & Featherman, 1977) norms for occu-
pertional status are not available for the state of Colorado, but the US Caucasian average is 41.7,
compared to 43.8 in our sample. Finally, the mean WAIS IQ for the twin parents is 108.4
(S.D.=12.6), with the wide range of 77–141. Thus, while the twin sample is probably biased
upwards compared to the national population on all these demographic variables, the bias is
small. Most important, however, is the wide range of the parental variables and the
comparability to the CAP sample. The number of twin pairs are listed later (Table 2).

2.2. Measures

For both CAP and LTS, the tests used were the Bayley Mental Development Index
(Bayley, 1969) at ages 1 and 2, the Stanford–Binet IQ test (Terman & Merrill, 1973) at ages
3 and 4, a composite based on the first principal components from cognitive battery
including the Wechsler Intelligence Scale for Children—Revised (WISC-R; Wechsler,
1974) administered in person at age 7, and a composite based the first principal component
from a telephone-administered cognitive test battery at ages 9 and 10, and the WISC-Rat age
12. (LTS data are not yet available at age 12.) The telephone battery was designed to assess
verbal, spatial, perceptual speed, and memory abilities. The telephone tests follow those
described by Kent and Plomin (1987) and have similar reliabilities to in-person versions of
the same tests, which were used in the Hawaii Family Study of Cognition (DeFries,
Vandenberg, McClearn, Kuse, & Wilson, 1974). The present battery also has similar factor
structure to an in-person version (Cardon, Corley, DeFries, Plomin, & Fulker, 1992).

2.3. Analysis

Although longitudinal correlations and twin and nontwin sibling correlations are reported,
the focus of the analysis is on longitudinal genetic model fitting. The model-fitting and
estimation procedures are implemented using the program Mx (Neale, 1999), a program
specifically developed for analyzing genetically informative twin and family data. Continuity is
modeled by allowing two developmental processes to be formalized in the structural equations.
First, genetic and environmental effects expressed in the child’s phenotype at one age may have
direct consequences for the phenotype expressed at later ages; this is sometimes referred to as
the simplex component of the model. Second, genes or environments may be consistent in the
nature of their influence during development independent of phenotypic transmission; this is
the common factor component of the model. In addition to developmental continuity, genetic
and environmental influences can change with age. Such changes are incorporated into the
developmental model by allowing independent genetic and environmental innovations, that is,
sources of variance specific to each age. These different developmental processes can be
resolved empirically because they lead to different correlations between relatives across ages.
The behavioral genetic model presently employed recognizes four sources of individual differences that are depicted schematically in Fig. 1.

For a pair of siblings measured on a particular phenotype $P$, $G$ represents additive genetic differences among individuals, $C$ represents common environmental influences shared by children reared together in the same home, $T$ represents environmental influences shared only by twins over and above shared sibling influences, and $E$ represents nonshared environmental influences unique to the individual. The coefficient of relationship ($r_G$) between genotypes for pairs of individuals varies in the present samples from 0 in the case of adoptive siblings, to 1/2 for nonadoptive siblings and DZ twins, to unity in MZ twins. The correlation ($r_C$) between the shared environment of sibling 1 with that of sibling 2 is, by definition, unity. The correlation ($r_T$) between twin environments is, by definition, 1 for twin pairs and 0 for nonadoptive and adoptive siblings. The correlation ($r_E$) between nonshared environmental influences is, by definition, zero.

The impacts of these four sources of variation—genetic, shared environmental, additional shared twin environmental, and nonshared environmental—are denoted as $h$, $c$, $t$, and $e$, respectively, and the variance explained by each is the square of these quantities, $h^2$, $c^2$, $t^2$, and $e^2$. The quantity $h^2$ is referred to as the narrow-sense heritability. In the absence of genetic dominance or epistasis (gene×gene interaction), this parameter describes the total variation due to genetic differences between individuals. Should sources of nonadditive genetic variation be important, the study of twins and siblings permits their detection through DZ twin or sibling correlation less than half the corresponding MZ twin correlations.

For the present analysis, the developmental model that was fitted to these data was one first proposed by Eaves, Long, and Heath (1986) and represents a combination of a single general factor present at all ages and a simplex model of age-to-age transmission effects, illustrated in Fig. 2 for genetic effects but applied in the same way to the shared environmental, additional shared twin environmental, and nonshared environmental latent factors.
The general factor implies a static process where influences are global across all ages. The simplex implies a more dynamic process in which new variation arises at each age and persists to the next age. A simplex model implies a matrix of correlations (between measures taken across time) where measures taken closer together are more highly correlated than measures taken further apart in time. The matrix would then have higher correlations nearer the diagonal and decreasing correlations further from the diagonal. This is typically observed with longitudinal data and is the case for our studies of cognitive ability.

The full longitudinal behavioral genetic model involves a fourfold expansion of the developmental model just described, to allow for genetic and shared, twin and nonshared environmental levels of variation and covariation. The expected covariance matrices for MZ and DZ twin pairs and nonadoptive and adoptive siblings implied by the full model can be derived using three parameter matrices adapted from the LISREL model (Jöreskog & Sörbom, 1989), \( B \), \( \Gamma \), and \( \Psi \), at each of the genetic shared, twin, and unique environmental levels, or 12 parameter matrices in total. The \( B \) matrices contain the age-to-age transmission parameters, the \( \Gamma \) matrices contain the common factor loadings, and the \( \Psi \) matrices contain the time-specific variances or the new variation at each age. The parameters in \( \Psi \) are constrained to be positive, as is appropriate for variances. The expectations for the genetic component of covariance, \( G \), are given by:

\[
G = (I - B_G)^{-1} (\Gamma_G \Gamma_G + \Psi_G) (I - B_G)^{-1}.
\]

The expectations for the shared, twin, and unique environmental components of covariance, \( C \), \( T \), and \( E \), respectively, are obtained in an analogous manner. In summary, the model has three types of parameters to explain continuity and change: (1) the loadings of the
common set of genes or environmental influences influencing the measures \( P_i \) at all ages, symbolized by \( \gamma_i \); (2) new genetic or environmental influences or innovations appearing at each age \( \psi_i \); and (3) age-to-age transmissions of genetic and environmental influences \( \beta_i \).

The full MZ, DZ, nonadoptive, and adoptive expected covariance matrices take a special form whereby they are partitioned into four equal quadrants. The top left and bottom right quadrants contain the within-pair variances and covariances and therefore contain the phenotypic variances and covariances. The other two quadrants contain the cross-sibling variances and covariances. These are expected to differ across the four groups of varying genetic and environmental similarity. The expected covariance matrices are estimated as:

\[
\begin{align*}
\sum &= \begin{pmatrix}
G + C + T + E & r \otimes G + C + t \otimes T \\
G \otimes C + t \otimes T & G + C + T + E
\end{pmatrix} \\
\end{align*}
\]

where \( r \) is equal to 1 for MZ pairs, 1/2 for DZ pairs and nonadoptive siblings, and 0 for adoptive siblings, and \( t \) is equal to 1 for twin pairs and 0 for sibling pairs.

Due to the incomplete nature of data from developmental studies, where individuals may be missing an assessment at one or more ages, we fit the model directly to the raw data rather than to observed covariance matrices. The data were first standardized within each age, across all individuals as a single group. This standardization procedure effectively eliminated age differences in means and variances, which may be due in part to using different tests at different ages, while preserving MZ, DZ, adoptive, nonadoptive, sibling 1, and sibling 2 variance differences. Fitting to the standardized raw data (rather than summary variance–covariance statistics), we maximize the log-likelihood function:

\[
\text{LL} = \sum_{i=1}^{N} \left[ -\frac{1}{2} \ln |\Sigma_i| - \frac{1}{2} (x_i - \mu_i)^T \Sigma_i^{-1} (x_i - \mu_i) \right]
\]

where \( x_i \) is the vector of scores for pair \( i \), \( \Sigma_i \) is the expected covariance matrix appropriate to the type of sibling pair, \( N \) is the total number of pairs, and \( \mu_i \) is the vector of mean scores for a particular type of sibling pair. We fit this model using maximum likelihood estimation procedure for raw data analysis implemented in Mx (Neale, 1999). Testing the relative fit of nested models (e.g., Models A and B) makes use of the fact that 2(LLA – LLB) is distributed as a \( \chi^2 \) statistic with degrees of freedom equal to the difference in the number of free parameters in the models.

3. Results

Table 1 gives the phenotypic correlations for general cognitive ability based on pooled CAP and LTS samples through age 12 years (although twin data are not presently available for analysis at age 12). As previously noted, general cognitive ability was assessed using the Bayley Mental Development Index (Bayley, 1969) at ages 1 and 2 years, the Stanford–Binet (Terman & Merrill, 1973) at ages 3 and 4. The first principal component for tests of specific
cognitive ability at ages 7, 9, and 10 years, and the WISC-R (Wechsler, 1974) at age 12 years. As can be seen, these correlations generally indicate a simplex pattern with increasing stability.

Table 2 shows the mean, standard deviation, and total N for the individual standardized tests at ages 1, 2, 3, 4, 7, and 12 years for the CAP sibling and LTS twin samples. As can be seen, the average scores are, if anything, slightly higher than for normative samples. An analysis of attrition found that CAP subjects who participated at age 12 years scored, on average 0.293 standard deviations higher at ages 1 through 7 than those who did not ($t=2.53, df=457, P<.05$). Twin subjects who participated at age 7 scored 0.502 standard deviations higher at ages 1 through 4 than those who did not ($t=4.29, df=883, P<.01$). Because of missing data in longitudinal studies and the likelihood of attrition biases in complete cases, we analyzed all available scores by fitting our models directly to the individual observations. Providing that it is reasonable to assume that missingness is conditional on the other observations (i.e., is predictable from the observed scores, as it appears to be in this study), then this procedure can provide unbiased parameter estimates even in the presence of selective attrition (Little & Rubin, 1978).

In Table 3, the correlations at each age are given for MZ, DZ, nonadoptive sibling (biological siblings living together), and adoptive sibling (nonbiological siblings living together) pairs with complete data at all years. Genetic influence is suggested at each age

Table 2
Longitudinal correlations for tests of general mental ability

<table>
<thead>
<tr>
<th>Age in years</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>7</th>
<th>9</th>
<th>10</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.00 (1576)</td>
<td>.39 (1395)</td>
<td>.27 (1312)</td>
<td>.26 (1287)</td>
<td>.18 (1290)</td>
<td>.09 (1003)</td>
<td>.06 (868)</td>
<td>.06 (575)</td>
</tr>
<tr>
<td>2</td>
<td>1.00 (1446)</td>
<td>.57 (1298)</td>
<td>.53 (1280)</td>
<td>.37 (1241)</td>
<td>.32 (953)</td>
<td>.29 (833)</td>
<td>.34 (557)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1.00 (1365)</td>
<td>.64 (1261)</td>
<td>.36 (1202)</td>
<td>.34 (920)</td>
<td>.30 (808)</td>
<td>.36 (544)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1.00 (1359)</td>
<td>.45 (1198)</td>
<td>.39 (913)</td>
<td>.35 (797)</td>
<td>.41 (535)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1.00 (1381)</td>
<td>.68 (1009)</td>
<td>.67 (865)</td>
<td>.58 (566)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>1.00 (1074)</td>
<td>.81 (881)</td>
<td>.62 (575)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.00 (928)</td>
<td>.66 (590)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>1.00 (647)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

* Sample size in parentheses.
in that MZ correlations are greater than DZ correlations and correlations for nonadoptive siblings exceed those for adoptive siblings. The twin data suggest increasing genetic influence but the adoption data do not. Shared environmental influence appears to be strongest at 3 years of age for both the twin data (the DZ correlation is much greater than half the MZ correlation) and the adoptive siblings. At 9 and 10 years, the twin and adoption data differ in their estimates of shared environmental influence. Because this is a longitudinal study, the twin and sibling cross correlations between occasions provide additional data about genetic and environmental influences on change and continuity during development that no cross-sectional study can yield. The cross-correlations across the eight ages for the four groups of siblings are not presented because each group would require a matrix the size of Table 1.

Because of the large number of possible models, we have followed Cherny et al. (1997) in considering each source of genetic and environmental influence in the following order: nonshared environment, special twin environment, shared environment, and then genetic influences. For each of these sources, we first considered the alternative models for continuity. We fit models that dropped only the common factor, only the transmission effects, or both of these together. We retained the model for continuity that best accounted for the data using the Akaike Information Criterion (AIC=$\chi^2 - 2*\text{degrees of freedom}$), taking into account both goodness of fit and parsimony. We then tested whether age specific effects could be dropped without a significant worsening of fit. Because of the very large total number of observations, we have also followed Cherny et al. (1997) in using a conservative .01 $\alpha$ level as a guide to the significance of parameter sets. Finally, we note that although the common factor and the transmission effects predict different patterns of correlations, when there are high correlations between occasions the two patterns become almost indistinguishable. In the limiting case of perfect correlations between occasions, the common factor and transmission models are interchangeable. As a consequence, common factor loadings and transmission parameters are not independent. Retaining both in a model can lead to negative dependencies between factor loadings and transmission parameters. This can result in negative parameter estimates that, while providing an optimal “mathematical” fit of a particular data set, are not

<table>
<thead>
<tr>
<th>Year</th>
<th>MZ n</th>
<th>DZ n</th>
<th>Nonadoptive Siblings n</th>
<th>Adoptive Siblings n</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.59</td>
<td>.40</td>
<td>.38 107</td>
<td>.07 87</td>
</tr>
<tr>
<td>2</td>
<td>.83</td>
<td>.64</td>
<td>.39 99</td>
<td>.04 88</td>
</tr>
<tr>
<td>3</td>
<td>.77</td>
<td>.51</td>
<td>.37 95</td>
<td>.26 84</td>
</tr>
<tr>
<td>4</td>
<td>.77</td>
<td>.51</td>
<td>.28 98</td>
<td>.06 87</td>
</tr>
<tr>
<td>5</td>
<td>.76</td>
<td>.40</td>
<td>.47 99</td>
<td>.04 88</td>
</tr>
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<td>6</td>
<td>.80</td>
<td>.21</td>
<td>.40 104</td>
<td>.24 83</td>
</tr>
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<td>7</td>
<td>.77</td>
<td>.12</td>
<td>.35 106</td>
<td>.25 85</td>
</tr>
<tr>
<td>8</td>
<td>n/a</td>
<td>n/a</td>
<td>.29 98</td>
<td>.12 88</td>
</tr>
</tbody>
</table>

* Significant correlations shown in bold ($P<.05$).
To avoid this, we have emphasized parsimony over statistical significance in arriving at the best model.

To test which developmental processes may be operating for general cognitive ability from ages 1 through 12 and to arrive at the most parsimonious model that could explain these data, we performed the series of model comparisons described above, beginning with tests of the nonshared environmental processes (Table 4). The first test was whether the common factor could be dropped from the model without a significant decrement in fit. We found that, indeed, a nonshared environmental common factor was unnecessary in explaining these data (Model 2). Next, the transmission parameters were tested and also found unnecessary (Model 3). Then the common factor and transmission parameters were tested as a set and, unlike the results reported by Cherny et al. (1997), were found to be necessary as a set (Model 4). Examination of the various transmission paths and common factor loadings led to a model (Model 5) that dropped all common factor paths and all transmission paths except those between 7 and 9 and between 9 and 10, which were the three ages that showed the highest longitudinal correlations (Table 1). This model adequately fit the data and was retained for further comparisons. Occasion-specific nonshared environmental influences could not be dropped (Model 6).

Table 4
Tests of nonshared environment development patterns

<table>
<thead>
<tr>
<th>Model</th>
<th>Description</th>
<th>−2LL</th>
<th>NPAR</th>
<th>χ²</th>
<th>df</th>
<th>P</th>
<th>AICc</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Full model</td>
<td>17,794.811</td>
<td>89</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Model 1, drop common factor</td>
<td>17,804.160</td>
<td>81</td>
<td>9.349</td>
<td>8</td>
<td>&gt;.30</td>
<td>−6.651</td>
</tr>
<tr>
<td>3</td>
<td>Model 1, drop transmission</td>
<td>17,795.946</td>
<td>82</td>
<td>1.135</td>
<td>7</td>
<td>&gt;.99</td>
<td>−12.865</td>
</tr>
<tr>
<td>4</td>
<td>Model 1, drop transmission/common factor</td>
<td>17,841.443</td>
<td>74</td>
<td>46.632</td>
<td>15</td>
<td>&lt;.001</td>
<td>16.632</td>
</tr>
<tr>
<td>5</td>
<td>Model 1, drop transmission except between 7–9 and 9–10 and common factor</td>
<td>17,806.849</td>
<td>76</td>
<td>12.038</td>
<td>13</td>
<td>&gt;.50</td>
<td>−13.962</td>
</tr>
<tr>
<td>6</td>
<td>Model 5, specifics</td>
<td></td>
<td>d</td>
<td>68</td>
<td>8</td>
<td>&lt;.001</td>
<td>d</td>
</tr>
</tbody>
</table>

a Log-likelihood function.
b Number of free parameters.
c Akaike’s information criterion.
d Cannot be estimated.

Robust. To avoid this, we have emphasized parsimony over statistical significance in arriving at the best model.

To test which developmental processes may be operating for general cognitive ability from ages 1 through 12 and to arrive at the most parsimonious model that could explain these data, we performed the series of model comparisons described above, beginning with tests of the nonshared environmental processes (Table 4). The first test was whether the common factor could be dropped from the model without a significant decrement in fit. We found that, indeed, a nonshared environmental common factor was unnecessary in explaining these data (Model 2). Next, the transmission parameters were tested and also found unnecessary (Model 3). Then the common factor and transmission parameters were tested as a set and, unlike the results reported by Cherny et al. (1997), were found to be necessary as a set (Model 4). Examination of the various transmission paths and common factor loadings led to a model (Model 5) that dropped all common factor paths and all transmission paths except those between 7 and 9 and between 9 and 10, which were the three ages that showed the highest longitudinal correlations (Table 1). This model adequately fit the data and was retained for further comparisons. Occasion-specific nonshared environmental influences could not be dropped (Model 6).

Table 5
Tests of twin environment development patterns

<table>
<thead>
<tr>
<th>Model</th>
<th>Description</th>
<th>−2LL</th>
<th>NPAR</th>
<th>χ²</th>
<th>df</th>
<th>P</th>
<th>AICc</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Model 5</td>
<td>17,806.849</td>
<td>76</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Model 5, drop common factor</td>
<td>17,810.663</td>
<td>69</td>
<td>3.814</td>
<td>7</td>
<td>&gt;.80</td>
<td>−10.186</td>
</tr>
<tr>
<td>9</td>
<td>Model 5, drop transmission</td>
<td>17,810.750</td>
<td>70</td>
<td>3.901</td>
<td>6</td>
<td>&gt;.90</td>
<td>−12.099</td>
</tr>
<tr>
<td>10</td>
<td>Model 9, drop specifics</td>
<td>17,834.567</td>
<td>56</td>
<td>15.682</td>
<td>7</td>
<td>&gt;.02</td>
<td>1.682</td>
</tr>
</tbody>
</table>

a Log-likelihood function.
b Number of free parameters.
c Akaike’s information criterion.
Table 5 summarizes tests of developmental patterns for the special twin shared environment. A test of the twin environment common factor (Table 4) indicated that it was not necessary for an adequate model fit (Model 7), nor were the simplex transmissions parameters (Model 8). As a set, the common factor and transmission parameters could be dropped in this case (Model 9). Lastly, the age-specific variances were dropped from the model (Model 10), yielding a base model with no environmental influences unique to twins and no nonshared environmental influences contributing to continuity before age 7. That is, environmental influences shared by twins, over and above those influences shared by ordinary siblings, do not appear to contribute importantly to individual differences in general cognitive ability, at least through age 10 years.

Test of the developmental processes present at the level of the shared sibling environment (which twins also share to some extent) appear in Table 6. When age-to-age transmission is retained in the model, the common factor is not significant (Model 11). When the common factor is dropped, age-to-age transmission is not significant (Model 12). However, both of these sources of continuity cannot be dropped (Model 13). Inspection of the parameter estimates shows that when the common factor is dropped, the transmission parameters are close to unity and the age-specific innovations are close to or at zero, mimicking the common

Table 6
Tests of shared-environment development patterns

<table>
<thead>
<tr>
<th>Model</th>
<th>Description</th>
<th>$-2\text{LL}$</th>
<th>NPAR</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$P$</th>
<th>AICc</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Model 10, drop common factor</td>
<td>17,846.476</td>
<td>48</td>
<td>11.909</td>
<td>8</td>
<td>&gt;.10</td>
<td>−4.091</td>
</tr>
<tr>
<td>12</td>
<td>Model 10, drop transmission</td>
<td>17,845.698</td>
<td>49</td>
<td>11.131</td>
<td>7</td>
<td>&gt;.10</td>
<td>−2.869</td>
</tr>
<tr>
<td>13</td>
<td>Model 10, drop transmission/common factor</td>
<td>17,866.642</td>
<td>41</td>
<td>32.075</td>
<td>15</td>
<td>&lt;.01</td>
<td>2.075</td>
</tr>
<tr>
<td>14</td>
<td>Model 12, drop specifics</td>
<td>17,851.559</td>
<td>41</td>
<td>5.861</td>
<td>8</td>
<td>&gt;.50</td>
<td>−10.139</td>
</tr>
</tbody>
</table>

$^a$ Log-likelihood function.
$^b$ Number of free parameters.
$^c$ Akaike’s information criterion.

Table 7
Tests of genetic development patterns

<table>
<thead>
<tr>
<th>Model</th>
<th>Description</th>
<th>$-2\text{LL}$</th>
<th>NPAR</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$P$</th>
<th>AICc</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>Model 14, drop common factor</td>
<td>17,851.559</td>
<td>41</td>
<td>20.867</td>
<td>8</td>
<td>&lt;.01</td>
<td>4.867</td>
</tr>
<tr>
<td>16</td>
<td>Model 14, drop transmission</td>
<td>17,980.973</td>
<td>34</td>
<td>129.414</td>
<td>7</td>
<td>&lt;.001</td>
<td>115.414</td>
</tr>
<tr>
<td>17</td>
<td>Model 14, drop transmission/common factor</td>
<td>18,567.233</td>
<td>26</td>
<td>715.674</td>
<td>15</td>
<td>&lt;.001</td>
<td>685.674</td>
</tr>
<tr>
<td>18</td>
<td>Model 15, drop specifics</td>
<td>18,832.059</td>
<td>25</td>
<td>40.367</td>
<td>8</td>
<td>&lt;.01</td>
<td>24.367</td>
</tr>
<tr>
<td>19</td>
<td>Model 14, drop common factor and psi-g at 10 and 12</td>
<td>17,873.983</td>
<td>31</td>
<td>22.424</td>
<td>10</td>
<td>&gt;.01</td>
<td>2.424</td>
</tr>
</tbody>
</table>

$^a$ Log-likelihood function.
$^b$ Number of free parameters.
$^c$ Akaike’s information criterion.
factor pattern. This together with the AIC values leads us to prefer Model 12 over Model 11 for our account of developmental continuity. Furthermore, age-specific shared environmental variance can be dropped without significant worsening of the fit (Model 14). Thus, it is the model with a single common factor at the shared environmental level and nonshared transmission from 7 to 9 and 9 to 10 years of age that is now the base model for subsequent tests of the genetic developmental processes.

Table 7 summarizes the tests of the genetic components in the developmental model. Although the common factor was statistically significant (Model 15), it was only just so in contrast to the simplex transmission parameters which represent the largest contribution to

---

Table 8
Path coefficients for the reduced model of cognitive development

<table>
<thead>
<tr>
<th>Path</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 7</th>
<th>Year 9</th>
<th>Year 10</th>
<th>Year 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Psi_G$</td>
<td>0.56</td>
<td>0.44</td>
<td>0.20</td>
<td>0.09</td>
<td>0.43</td>
<td>0.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\beta_G$</td>
<td>0.58</td>
<td>0.60</td>
<td>1.00</td>
<td>0.76</td>
<td>0.89</td>
<td>1.03</td>
<td>0.81</td>
<td></td>
</tr>
<tr>
<td>$\Gamma_C$</td>
<td>0.17</td>
<td>0.42</td>
<td>0.55</td>
<td>0.47</td>
<td>0.18</td>
<td>0.17</td>
<td>0.17</td>
<td>0.34</td>
</tr>
<tr>
<td>$\Psi_E$</td>
<td>0.41</td>
<td>0.20</td>
<td>0.28</td>
<td>0.27</td>
<td>0.25</td>
<td>0.27</td>
<td>0.21</td>
<td>0.40</td>
</tr>
<tr>
<td>$\beta_E$</td>
<td>0.12</td>
<td>0.29</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
developmental continuity. Age-specific genetic influences were substantial at earlier ages and highly significant. A model, which drops the common factor and the age-specific innovations at the later ages (10 onwards), fits the data well enough for us to prefer it over retaining both common factor and transmission parameters that become confounded because of the very high genetic correlations from age to age from 9 years onwards.

The final reduced model is, therefore, Model 19, shown in Fig. 3, with parameter estimates shown in Table 8.

This final developmental model gives an account of the data not significantly worse than the initial full model ($\chi^2=79.172$ for 58 df, $P > .025$). Table 9 gives the estimates of heritability ($h^2$), shared environmental variation ($c^2$), and nonshared environmental variation ($e^2$) under this model. Heritability is substantial at all years, with an increasing trend except for year 12 where the sample does not yet include twins. Shared environment is modest after year 4. Also shown in this table is the percentage of each source of variation that is newly arising at each age of assessment. Substantial new genetic variation emerges up through year 7. At 9 years, new genetic variation is modest and, at 10 and 12 years, no new genetic variation emerges, suggesting that genetic influence contributes largely to continuity during the transition to adolescence. Although shared environmental influence is modest, it contributes entirely to continuity. Nonshared environment contributes to change with the possible exception of middle childhood where it also contributes to continuity.

Tables 10 and 11 show the genetic ($R_g$) and nonshared environment ($R_e$) correlations, estimated from our final model. Shared environmental correlations ($R_c$) would be unity since our final model includes only a single common factor. As suggested by the fit of the final model (see Fig. 2), genetic correlations are generally moderate, suggesting genetic change as
well as continuity. One exception is from 3 to 4 years where the genetic correlation is .91 as compared to .64 from 4 to 7 years, which may reflect genetic change during the transition from early to middle childhood. The other exception is that genetic correlations approach unity at 7, 9, 10, and 12 years, suggesting again that genetic factors contribute largely to continuity during the transition to adolescence. The nonshared environment correlations in Table 11 confirm that nonshared environment contributes almost entirely to change with the exception of 7, 9, and 10 years where there is some contribution to continuity.

4. Discussion

The final model from 1 to 12 years largely confirms hypotheses drawn from longitudinal genetic analyses in infancy and childhood, especially the combined twin and sibling analyses reported by Cherny et al. (1997). Heritability, which is substantial at all ages, tends to increase. Shared environmental influence is negligible after 4 years. Nonetheless, to the extent that they have an effect, shared environmental influences shared by virtue of being reared in the same household appear to be of a global nature, represented by a single common factor with relatively constant influence from infancy through early adolescence. Socioeconomic status (SES) is a likely candidate. Although the association between parental SES and children’s IQ is substantially mediated by genetic factors, some shared environmental mediation remains when genetics is controlled (Plomin, 1994) and would seem likely to be stable.

Nonshared environment largely contributes to change, although these analyses suggest that during middle childhood (from 7 to 10 years), some continuity emerges for nonshared environment. In other words, at these ages there may be environmental influences that affect general cognitive ability in a cumulative way. Although this cumulative effect of nonshared environment might be limited to the early school years, it is possible that such effects continue. The effect could not yet be confirmed to continue through age 12, but this may be because MZ twin data—the most powerful for unambiguously detecting nonshared environmental influence—are not yet available for analysis at age 12 years. It is also possible that

<table>
<thead>
<tr>
<th>Age in years</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>7</th>
<th>9</th>
<th>10</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.00</td>
<td>.00</td>
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<td>.31</td>
<td>.00</td>
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<td>.00</td>
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<td>.00</td>
</tr>
<tr>
<td>9</td>
<td>1.00</td>
<td>.31</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
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<tr>
<td>12</td>
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<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
</tr>
</tbody>
</table>
this nonshared environmental influence is detected only for the component scores analyzed here for ages 7 through 10, rather than the individual test scores at the other ages. Clearly, it will be important to extend this analysis into the adolescent years with the full adoption and twin longitudinal data set to determine which of these possibilities is correct.

The most interesting finding is that by 9 years of age genetic factors contribute only to continuity, which disconfirms the hypothesis from earlier childhood. This increased genetic stability in early adolescence may be the genetic underpinning for the increased long-term phenotypic stability that occurs by early adolescence (Deary et al., 2000). These results replicate those reported by Cherny et al. (1997) through age 7 years, but differ from their earlier results at age 9 and 10 years now that twin data at those ages have become available as well as CAP sibling data at age 12.

As in the analysis by Cherny et al. (1997), there was no evidence of significant special twin environment effects that make twins more similar than nontwin siblings. This result is especially noteworthy because the combined twin and sibling design is biased in favor of finding such effects in the sense that the twins are tested at the same age on the same occasion, whereas siblings are tested at the same age but on different occasions at least 2 years apart. Our results seem to contrast with the hypothesis and Bayesian meta-analysis of previously published correlations reported by Devlin et al. (1997). Those authors found that the data they reviewed were best accounted for by a model that assigned 20% of the variance to twins’ shared prenatal environment (while only 5% for siblings). They also reported that an age effects hypothesis (increasing heritability, decreasing shared environment) fit quite well, though not as well ‘by Bayes factor’ as their maternal effects model. Unfortunately, meta-analyses of published correlations from methodologically diverse studies cannot be definitive and we are impressed by our overall failure to substantiate the twin maternal (shared womb) effect in our study using the same assessment procedures at the same ages for all family types. Nonetheless, the story might be more complicated developmentally. The twin and sibling correlations in Table 3 suggest special twin shared effects from 1 to 4 years when the DZ correlations average .52 and the nonadoptive sibling correlations average .35. However, at 9 and 10 years, the twin and sibling correlations suggest less shared environmental influence (or more contrast effects) for twins in that the average DZ correlation is .17 and the average nonadoptive sibling correlation is .38. The overall result that there are no special twin effects may be due to these counterbalanced developmental trends.

We further tested the possible heterogeneity of result for the twin and sibling adoption designs into two ways. First, we fit the final model separately to the twins and siblings. The fit of the model for twins (−2LL=10,465.277 for 28 parameters) differed little from the fit of the model for siblings (−2LL=7349.379 for 31 parameters). These sum to an overall −2LL for the heterogeneous model of 17,814.656 for 59 parameters. Testing this against the final model in the paper (−2LL=17,823.564 for 31 parameters) yields a difference chi-square test of heterogeneity of 8.908, \(df=28\), \(P>.99\). In other words, using this test of heterogeneity, the twins and sibs yield essentially the same parameters. However, this test may be weak because the model in the individual samples may be wrong (because we arrived at it by doing the series of tests with twin and sib parameters already equated), thus, inflating the individual
-2LL’s. Comparing their sum with the combined sample, -2LL would yield a weak test of heterogeneity.

For this reason, rather than using the final model, we also compared full models (common factor plus transmissions plus specifics, for each of G, C, and E) for twins and siblings. For the full model, -2LL was 7310.516 for 60 parameters for twins and 10,428.086 for 69 parameters for siblings. These -2LL sum to 17,738.602 for 129 parameters. The full model for the combined twin and sibling analyses yielded -2LL of 17,820.056 for 69 parameters. The difference in -2LL is 81.454, which yields P > .03 with 60 df. This test of heterogeneity does not reach the level of significance (P < .01) adopted in this paper but warrants future exploration of a possible difference between the twin and sibling adoption designs.

These analyses confirm that the most important source of individual differences in general cognitive ability is the genetic inheritance of the individual child. Nonshared environmental variation is substantial, although it must be kept in mind that this includes measurement unreliability and any other sources of “error”. Environmental influences shared by the family are significant and especially important in infancy, prior to attendance at school. The most interesting aspect of these results is the pattern of genetic influences during development. During infancy and early childhood, genes contribute in novel ways at each age of assessment, strongly suggesting that the biological underpinnings of general cognitive ability are not fixed at birth but continue to change along with the still developing brain. Later, by age 10 or 11 years, we witness the end of this developmental process and the beginning of the stabilization of the heritable influences on general cognitive ability.

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References


