Developmental Psychology
1987, Vol. 23, No. 1, 4-12

0012-1649/87/$00.75

Genetic Stability of Cognitive Development From Childhood to Adulthood

J. C. DeFries, Robert Plomin, and Michele C. LaBuda
Institute for Behavioral Genetics
University of Colorado, Boulder

Genetic stability from infancy to adulthood has been proposed to account for observed parent–child resemblance in cognitive test scores in the Colorado Adoption Project. Although genetic variance increases dramatically from infancy to adulthood, results of preliminary analyses suggested that the genetic influences on cognitive ability in infancy and in adulthood may be highly correlated. To assess genetic stability more rigorously, a path model of genetic and family environmental transmission was fitted to general cognitive ability data from adoptive and nonadoptive families in which children were tested at 1, 2, 3, and 4 years of age, as well as to published twin correlations. Longitudinal genetic correlations from infancy and early childhood to adulthood were modeled explicitly, as were the effects of phenotypic assortative mating on both parent–child and twin resemblance. In general, results of the present study suggest significant and substantial genetic stability from 2, 3, and 4 years of age to adulthood.

The longitudinal adoption study by Skodak and Skeels (1949) is one of the most frequently cited articles in developmental psychology. In this landmark study, 100 children adopted before 6 months of age were tested at least four times between early childhood and adolescence, and their IQ scores were compared to educational level and occupational status of their adoptive parents, to education and occupation of their biological parents, and to IQ scores of about two-thirds of the biological mothers. In general, the correlations between the IQ scores of biological mothers and those of their adopted-away children indicated increasing genetic influence from early childhood to adolescence, reaching a level of .44 at 13.5 years of age.

Despite the importance of this longitudinal adoption study, over 20 years elapsed before the adoption design was again used to study psychological development. Then, during the 1970s, a number of studies employing the adoption design were reported (see Plomin & DeFries, 1985, for a recent review); however, none assessed genetic stability from infancy and early childhood to adulthood.

In 1975, Plomin and DeFries (1985) initiated the Colorado Adoption Project (CAP). The CAP differs from previous adoption studies in a number of respects: Data are obtained from biological parents of adopted children, adoptive parents, adopted children, and members of nonadoptive families; biological fathers are tested when possible; the design is longitudinal (children are tested in their homes at 1, 2, 3, and 4 years of age and in a laboratory setting at 7 years of age; and follow-up studies during adolescence are in progress); the sample size is large (over 200 adoptive and 200 nonadoptive families are currently participating in the study); behavioral assessments are multivariate, and information concerning the home and family environment is collected; selective placement is minimal; and the design is prospective. These differences from previous adoption studies facilitate unique analyses pertaining to the etiology of stability from infancy and early childhood to adulthood.

One of the most exciting findings of the CAP to date has been the suggestion of genetic stability from infancy to adulthood for cognitive development (Plomin & DeFries, 1985). The power of an adoption design lies in its direct estimate of genetic influence from the phenotypic correlation between, for example, biological parents and their adopted-away offspring. However, when biological parents are tested as adults and their adopted-away offspring are tested as children, significant resemblance between them requires not only that the characteristic be heritable in adulthood and in childhood but also that it be influenced by some of the same genes at the two ages. The genetic correlation between cognitive scores in childhood and adulthood provides an index of the extent of such genetic stability (Plomin & DeFries, 1981). In the CAP, resemblance between general cognitive ability (IQ) of the biological parents and Bayley Mental De-

The Colorado Adoption Project (CAP) is supported in part by grants from the W. T. Grant Foundation, the National Institute of Child Health and Human Development (NICHD; HD-10333 and HD-18426), the National Institute of Mental Health (MH-16880), the National Science Foundation (BNS-7826204 and BNS-8200310), and the Spencer Foundation. This report was prepared while M. C. LaBuda was supported by NICHD Training Grant HD-07289.

We are grateful to the families participating in the CAP, who have so generously contributed their time and effort, and to the adoption agencies who make the study possible—Lutheran Social Services of Colorado and Denver Catholic Community Services. We also thank Rebecca G. Miles and J. Toni Ensley for expert editorial and secretarial assistance.

Correspondence concerning this article should be addressed to J. C. DeFries, Institute for Behavioral Genetics, Campus Box 447, University of Colorado, Boulder, Colorado 80309.

velopment Index (MDI) scores of the adopted infants suggests some genetic stability from infancy to adulthood.

Assuming random mating and no selective placement, the expected correlation between biological parents and their adopted-away children is as follows:

$$r_{pc} = .5h_c h_a r_G, \quad (1)$$

where r_{pc} is the parent–child correlation, h_c is the square root of heritability (the proportion of phenotypic variance due to additive genetic variance; see Plomin, DeFries, & McClearn, 1980) of MDI scores in children, h_a is the square root of heritability of general cognitive ability in adults, and r_G is the longitudinal genetic correlation. Thus, if no correlation is found between biological parents' IQ and their adopted-away infants' Bayley MDI scores, genetic effects on Bayley MDI scores in infancy are not correlated with genetic effects on adult IQ scores. If, on the other hand, the correlation between biological parents and their adopted-away children is significant, some developmental genetic stability must exist from infancy to adulthood. The magnitude of the genetic correlation depends on the parent–offspring correlation and the heritabilities in childhood and adulthood. The genetic correlation will be especially high when heritabilities are low at either or both ages; in this case, the modest genetic effects at each age must correlate highly between the ages in order to produce, as the product $.5h_c h_a r_G$, the observed phenotypic correlation between biological parents and their adopted-away offspring.

In the CAP, the observed correlation between biological parents' IQs and their adopted-away infants' Bayley MDI scores is about .10. Twin data suggest that the heritability of Bayley MDI scores in infancy is about .15 and the heritability of adult IQ scores is about .50. Thus, according to the simple model described in Equation 1, the genetic correlation between infant MDI scores and adult IQ is about .75 (Plomin & DeFries, 1985). This evidence for substantial genetic stability between infancy and adulthood suggests that, although heritability is relatively low during infancy, individual differences in Bayley MDI scores and adult IQ scores may be influenced by many of the same genes. In other words, even though genetic variance accounts for only a small proportion of the observed phenotypic variance in Bayley MDI scores, this genetic variance in infancy covaries highly with the greater genetic variance of adult IQ scores.

A substantial genetic correlation from infancy to adulthood implies that there is some phenotypic stability within individuals, but the phenotypic stability need not be substantial. The genetic correlation indicates the extent to which genetic effects overlap at two ages, regardless of their contribution to phenotypic variance. The genetic contribution to phenotypic stability, on the other hand, is $h_c h_a r_G$, the genetic correlation weighted by the square roots of the heritabilities at each age. Thus, the CAP data suggest that the genetic contribution to phenotypic stability from infant Bayley MDI scores to adult IQ is about .20. Environmental mediation of stability could add to this figure. Nonetheless, this prediction of phenotypic stability is within the limits of stability found in previous studies—for example, a review by McCall (1979) yielded a median correlation of .32 for three studies that compared test scores of 13- to 18-month-old infants to their IQ scores at 8 to 18 years of age.

Subsequent to our early work on genetic stability, LaBuda, DeFries, Plomin, and Fulker (1986) fitted a path model of genetic and shared family environmental transmission to general cognitive ability data from CAP adoptive and nonadoptive families to assess the etiology of longitudinal stability from infancy to early childhood. An inspection of genetic transmission parameters estimated from that study suggested substantial genetic stability from infancy and early childhood to adulthood.

Eaves, Long, and Heath (1986) independently formulated a path model of cognitive development in which genetic and environmental influences are either constant across ages or specific to each age. When this model was applied to cognitive data from infancy and childhood in the Louisville Twin Study, results suggested that genetic stability across ages is substantial.

Because of its important implications for developmental behavioral genetics, as well as the fact that genetic stability is counter to the prevailing perspective of developmental theory (Plomin, 1986), a more rigorous evaluation of the evidence is warranted. For example, one inadequacy of the rudimentary analysis by Plomin and DeFries (1985) was the assumption of random mating. Moderate assortative mating for general mental ability occurs in the CAP, and mate correlations contribute to the observed parent–offspring resemblance (see Plomin et al., 1980). Thus, solution of Equation 1 yields an overestimate of the longitudinal genetic correlation when the parent–child correlation is due in part to positive assortative mating. Moreover, when positive assortative mating occurs, heritability is underestimated by doubling the difference between identical and fraternal twin correlations. As may be seen from Equation 1, underestimates of heritability yield overestimates of the longitudinal genetic correlation. Although assortative mating was modeled in the LaBuda et al. (1986) analysis of CAP parent–offspring data, random mating was assumed when heritabilities were estimated from twin data. Furthermore, in neither of our previous analyses was the longitudinal genetic correlation explicitly modeled and tested for statistical significance.

Thus, the objective of the present study was threefold: (a) to explore genetic stability in more detail by formulating path diagrams that model the longitudinal genetic correlation explicitly and facilitate a test of its significance; (b) to model the effects of phenotypic assortative mating on both parent–offspring and twin resemblance; and (c) to analyze cognitive data from a larger sample of CAP adoptive and nonadoptive families than has previously been reported on.

Method

Sample

The CAP is a longitudinal, prospective study of behavioral development in which adopted children and matched nonadopted children are assessed during home visits at 1, 2, 3, and 4 years of age. Biological and adoptive parents of adopted children and parents of nonadopted children are administered a 3-hour battery of tests, and extensive questionnaire data are obtained. In the present study, separate analyses of family data for children tested at each age were undertaken. The number of adoptive families in each analysis ranges from 155 to 196, whereas the number of nonadoptive families ranges from 129 to 161.

As described in more detail by Plomin and DeFries (1985), the variance in the CAP sample with regard to socioeconomic status is repre-

sentative of the United States general population. However, the means for adoptive and nonadoptive fathers are about one standard deviation above the national average. For example, the mean (± standard deviation) revised Socioeconomic Index (SEI; Hauser & Featherman, 1977) values for adoptive and nonadoptive fathers are 63.4 ± 20.5 and 61.9 ± 18.2, respectively. As expected, biological fathers are considerably younger than adoptive or nonadoptive fathers (over 10 years on average), and their average SEI is lower (31.7 ± 22.4). However, the parents of these adults (i.e., the biological, adoptive, and nonadoptive grandparents) are highly similar with regard to both mean SEI and variance (see Plomin & DeFries, 1985), suggesting that the biological parents of CAP adopted children are not from a disadvantaged group. Selective placement has been found to be negligible for all measures examined to date, including socioeconomic status, education, and cognitive abilities.

In order to estimate longitudinal genetic correlations from the parent–offspring data, twin correlations were also incorporated into our analyses. Correlations of identical and fraternal twin pairs tested in the Louisville Twin Study (Wilson, 1983) at 1, 2, 3, and 4 years of age were employed for this purpose. These correlations were estimated from the following numbers of identical and fraternal twin pairs: 1 year of age, 89 and 92; 2 years, 88 and 115; 3 years, 104 and 125; and 4 years, 105 and 120. For older twins, corresponding correlations reported by Loehlin and Nichols (1976) were utilized. Although these correlations are based on data from young adults, the sample is large (1,300 identical and 864 fraternal twin pairs) and the obtained correlations are typical of other twin studies of adult IQ (cf. Bouchard & McGue, 1981).

Measures

A battery of 13 cognitive tests that assess verbal, spatial, perceptual speed, and memory factors is administered to adults. The tests, reliabilities, methods employed for sex and age adjustment, and the factor structure are described elsewhere (e.g., DeFries, Plomin, Vandenberg, & Kuse, 1981). The first unrotated principal component score is used as a measure of general cognitive ability.

During visits to the homes of the adopted and nonadopted infants at 12 and 24 months of age, the Bayley Scales of Infant Development (Bayley, 1969) are administered. The Stanford-Binet Intelligence Scale—Form L-M (Terman & Merrill, 1973) is administered when the children are 3 and 4 years old.

Path Model

The path model employed in the present analysis is an extension of a simplified version of the adoption model formulated by Fulker and DeFries (1983). The model facilitates tests of additive genetic and shared family environmental transmission, genetic continuity from childhood to adulthood, assortative mating, selective placement, and special environmental influences shared by members of twin pairs. Although elementary, the model appears to be plausible for mental development. Results of previous analyses of CAP cognitive data have provided little or no evidence for genotype–environment correlations or nonlinear interactions (e.g., Fulker & DeFries, 1983; Plomin & DeFries, 1985; Rice, Fulker, & DeFries, 1986).

A path diagram of genetic and shared family environmental transmission in nonadoptive families is depicted in Figure 1. The basic model includes only two latent variables, additive genetic value (G) and environmental deviation (E), which completely determine P (the phenotypic value) with paths h and e, respectively. Residual causes have been omitted from the diagram because they do not contribute to expected correlations among the variables. Both mother (P_M) and father (P_F) contribute to the genetic (G_O) and environmental (E_O) determinants of a child's mental performance (P_O). In order to assess genetic stability from childhood to adulthood, the model includes genetic values of parents both as adults (G_M and G_F) and as children (G_{Mc} and G_{Fc}). Thus, the longitudinal genetic correlation (r_G) is modeled explicitly as a path coefficient between the parental genetic values as adults and as children. Genetic transmission from parent to offspring is modeled via G_{Mc} and G_{Fc} with path coefficients of 1/2; transmission involving shared family environment via the maternal and paternal phenotypes is modeled with paths m and f, respectively.

Genotype–environment correlation is assumed to be zero and thus is not modeled explicitly. Because of this assumption it is possible to standardize G, E, and P to zero mean and unit variance for both adopted and nonadopted children and their parents. However, it may be noted that in Figure 1 a correlation between genetic and environmental influences on the child will be induced if genetic and environmental transmission both occur. Thus, the significance of the genetic and environmental transmission parameters included in the model will provide a test of the validity of this assumption.

Phenotypic assortative mating, that is, the tendency for individuals to select mates on the basis of observed phenotypes, is assumed. This process results in a phenotypic correlation between spouses (p) and induces correlations between their genotypic values (both as adults and as chil-

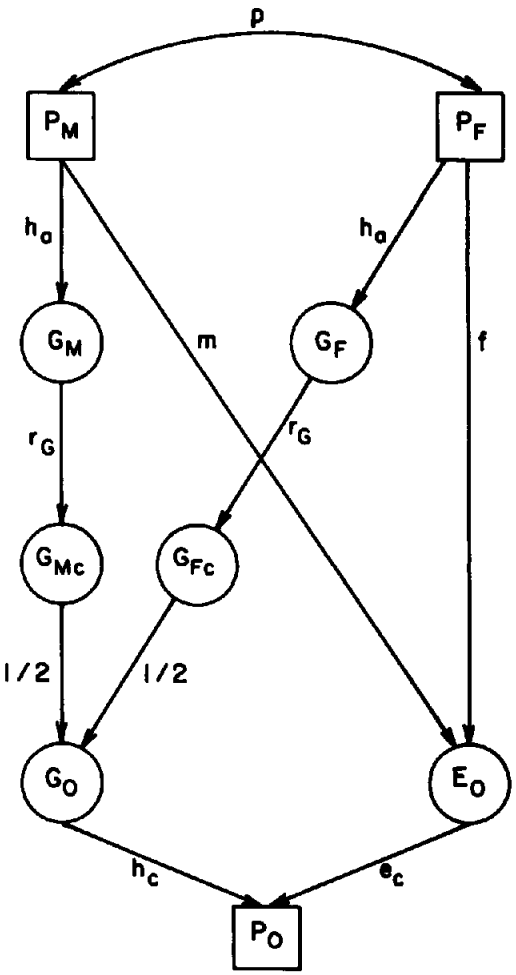

Figure 1. Path diagram representing genetic and shared family environmental transmission in nonadoptive families (G = additive genetic value, E = environmental deviation, P = phenotypic value, M = mother, F = father, O = offspring, a = adult, c = child).

Table 1
Expected Phenotypic Correlations

Variable	Expected correlation
Nonadoptive families	
P_M, P_F	p
P_M, P_O	$.5h_a h_c r_G(1+p) + e_c(m+pf)$
P_F, P_O	$.5h_a h_c r_G(1+p) + e_c(f+pm)$
Adoptive families	
P_{BM}, P_{BF}	q
P_{AM}, P_{AF}	p
P_{BM}, P_{AF}	x_1
P_{BM}, P_{AM}	x_2
P_{BF}, P_{AF}	x_3
P_{BF}, P_{AM}	x_4
P_{BM}, P_{AO}	$.5h_a h_c r_G(1+q) + e_c(x_1 f + x_2 m)$
P_{BF}, P_{AO}	$.5h_a h_c r_G(1+q) + e_c(x_3 f + x_4 m)$
P_{AF}, P_{AO}	$e_c(f+pm) + .5h_a h_c r_G(x_1 + x_3)$
P_{AM}, P_{AO}	$e_c(m+pf) + .5h_a h_c r_G(x_2 + x_4)$
Adult twins	
P_{T1}, P_{T2} (identical)	$h_a^2 + e_a^2(m^2 + f^2 + 2mfp + t_a^2)$
P_{T1}, P_{T2} (fraternal)	$.5h_a^2(1 + h_a^2 p) + (e_a^2(m^2 + f^2 + 2mfp + t_a^2)$
Child twins	
P_{T1}, P_{T2} (identical)	$h_c^2 + e_c^2(m^2 + f^2 + 2mfp + t_c^2)$
P_{T1}, P_{T2} (fraternal)	$.5h_c^2(1 + h_a^2 r_G^2 p) + e_c^2(m^2 + f^2 + 2mfp + t_c^2)$

Note. M, F, O, BM, BF, AM, AF, AO, T1, and T2 symbolize nonadoptive mother, nonadoptive father, nonadopted child, biological mother, biological father, adoptive mother, adoptive father, adopted child, twin, and co-twin, respectively. See Figures 1–4 for corresponding path diagrams.

dren). To model this complexity, the method of reverse path analysis (see Fulker & DeFries, 1983) was employed.

Figure 1 facilitates the derivation of three expected correlations among the three manifest variables following the rules of path analysis (Li, 1975). These expectations for nonadoptive families, expressed in terms of the model parameters, are presented at the top of Table 1.

Our model of transmission in adoptive families is depicted in Figure 2. Adoptive parents contribute only environmental influences (m and f), whereas biological parents contribute only genetically to the adopted child's phenotype. Although little or no evidence of selective placement has been found to date in the CAP, for completeness it is modeled in Figure 2 by correlations between the phenotypes of biological and adoptive parents (x_1 through x_4).

By comparing Figures 1 and 2, it may be seen that spouse resemblance is assumed to be similar for adoptive and nonadoptive parents. However, assortative mating may differ for biological (unwed) parents of adopted children. In order to test this hypothesis, a different assortative mating parameter for biological parents of adopted children (q) is included in the model.

The five manifest variables included in Figure 2 yield a total of 10 expected correlations. These expectations are expressed in terms of the model parameters in the middle portion of Table 1.

The path diagrams in Figures 3 and 4 illustrate how genetic and environmental influences contribute to observed twin resemblance when the twin pairs are tested either as adults or as children. In addition to the transmission parameters that cause parent–child resemblance, a latent variable (E_t) with path coefficient t is included in the twin diagrams. This parameter is included to account for any unique resemblance between members of twin pairs that is not due to parent–child transmission (e.g., special shared environmental influences due to twin contemporaneity, etc.). In other words, t refers to correlations between the environmental deviations of twins. The contribution of this parameter to the twins' phenotypic correlation, usually called shared or common environment, is e^2t^2, where e^2 is the proportion of observed variance due to all environmental influences. Twins have been found to share environmental influences to a greater extent than do other family members such as nontwin siblings (e.g., Plomin & DeFries, 1980), but the effective environments apparently are highly similar for the two types of twins (Loehlin & Nichols, 1976; Scarr & Carter-Saltzman, 1979).

Although data from parents of twins were not included in our analysis, it is assumed that spouse resemblance for parents of twins is similar to that for adoptive and nonadoptive parents of singletons (p). In addition, it is assumed that environmental transmission is similar in families of twins and singletons. Thus, it is possible to express expected identical and fraternal twin correlations in terms of t and the parent–child model parameters (see bottom of Table 1).

Analysis

For families of singletons, the summary statistics to which the model was fitted are variances and covariances. In the case of nonadoptive families, which include mothers, fathers, and children tested at a given age, these statistics form a 3×3 matrix. The numbers of nonadoptive families on which these matrices are based are as follows: child tested at 1 year of age, 161; 2 years, 153; 3 years, 146; and 4 years, 129. In the case of adoptive families, complete data on biological mothers, biological fathers, adoptive mothers, adoptive fathers, and adopted children yield a 5×5 covariance matrix for each age at which the children were tested.

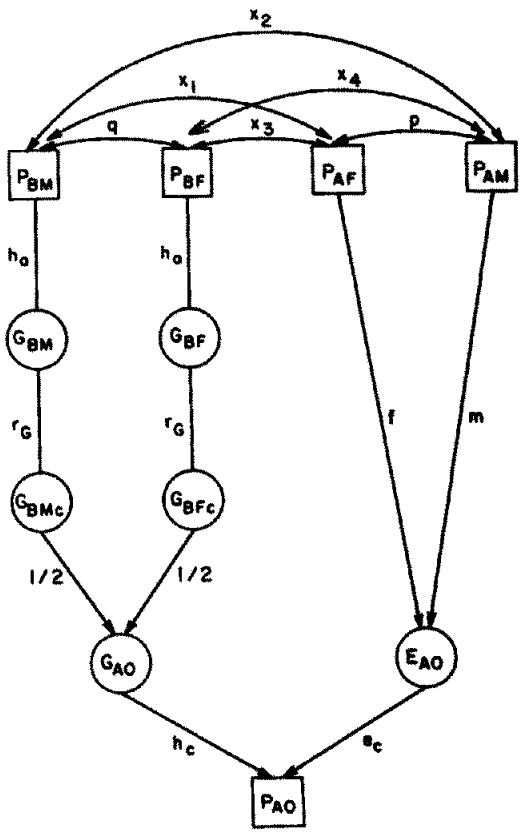

Figure 2. Path diagram of genetic and shared family environmental transmission in adoptive families (G = additive genetic value, E = environmental deviation, P = phenotype value, BM = biological mother, BF = biological father, AM = adoptive mother, AF = adoptive father, AO = adopted offspring, a = adult, c = child).

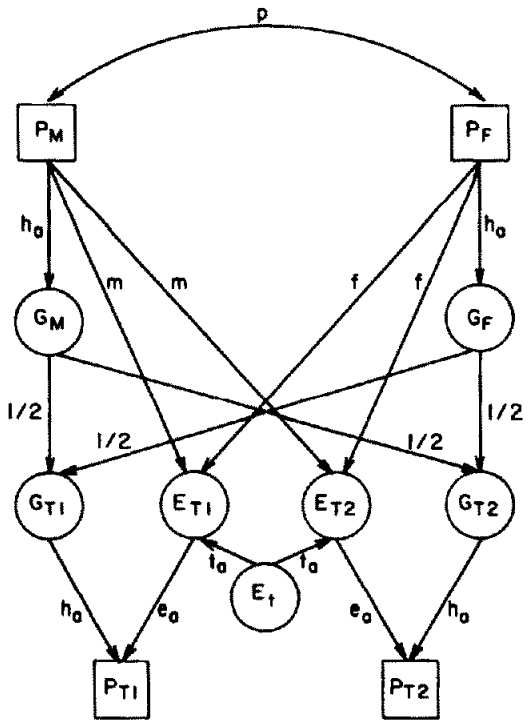

Figure 3. Path diagram of genetic and shared family environmental transmission in families of twins tested as adults (G = additive genetic value, E = environmental deviation, P = phenotypic value, M = mother, F = father, T1 = twin, T2 = co-twin, a = adult).

Although considerable effort was made to test biological fathers of adopted children, the 5 × 5 matrices that include data from biological fathers are based only on the following numbers of families: children tested at 1 year of age, 42; 2 years, 39; 3 years, 40; and 4 years, 34. However, the sample of adoptive families lacking information from biological fathers is larger (1 year, 154; 2 years, 140; 3 years, 136; and 4 years, 121) and was used to calculate independent 4 × 4 matrices. In the case of twins, 2 × 2 correlation matrices (identical and fraternal twin correlations for children tested at each age and for adults) were analyzed, using the data of Wilson (1983) and Loehlin and Nichols (1976).

To estimate the model parameters for each age at which the children were tested, the seven observed matrices were simultaneously equated to the expected matrices using a maximum-likelihood estimation procedure (see Fulker & DeFries, 1983). The following function was minimized using the generalized numerical optimization package MINUIT (CERN, 1977):

$$F = \sum_{k=1}^{m} N_k\{\ln|E(S_k)| - \ln|S_k| + \mathrm{tr}[S_k E(S_k)^{-1}] - p_k\}, \qquad (2)$$

where the k observed and expected matrices are symbolized S_k and $E(S_k)$, respectively. N_k is the degrees of freedom of the kth matrix, and p_k is the order of the matrix. In the present analysis, p_k varies from five to two and we sum over seven matrices. F, the log-likelihood ratio statistic, is distributed asymptotically as chi-square with the following degrees of freedom:

$$df = \sum_{k=1}^{m} [p_k(p_k + 1)/2] - n, \qquad (3)$$

where n is the number of parameters estimated.

The full model was first fitted to the data (separately as a function of child's age) to estimate the model parameters. Then a series of reduced models was fitted to the data to test the significance of these parameter estimates. Although standard errors of the parameter estimates were computed, they are only approximate because of the numerical methods employed to derive them. In contrast, comparing the goodness of fit of alternative models using chi-square provides significance tests that are more robust.

Results

Observed correlations in nonadoptive families, pooled estimates of observed correlations in adoptive families, and the twin correlations analyzed each year the children were tested are presented in Table 2. Inspection of the observed correlations involving adoptive and biological parents indicates that selective placement is negligible in the CAP and that assortative mating is moderate. Moreover, the correlations between adopted children and their biological and adoptive parents suggest increasing genetic and shared environmental influences as a function of child's age.

Although inspection of the observed correlations in Table 2

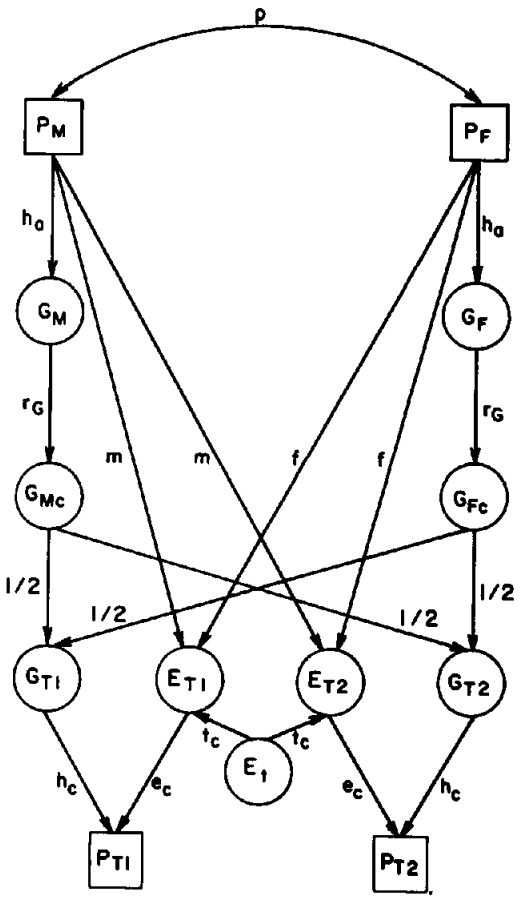

Figure 4. Path diagram of genetic and shared family environmental transmission in families of twins tested as children (G = additive genetic value, E = environmental deviation, P = phenotypic value, M = mother, F = father, T1 = twin, T2 = co-twin, a = adult, c = child).

Table 2
Observed Correlations

Variable	Phenotypic correlation			
	Year 1	Year 2	Year 3	Year 4
P_M, P_F	.13	.10	.12	.13
P_M, P_O	.07	.20	.16	.21
P_F, P_O	.10	.17	.09	.08
Adoptive families (pooled)				
P_{BM}, P_{BF}	.28	.28	.35	.37
P_{AM}, P_{AF}	.28	.28	.29	.31
P_{BM}, P_{AF}	−.01	−.03	−.03	.07
P_{BM}, P_{AM}	.02	.06	.07	.14
P_{BF}, P_{AF}	−.13	−.08	−.07	.03
P_{BF}, P_{AM}	.04	.11	.10	.19
P_{BM}, P_{AO}	.10	.07	.18	.24
P_{BF}, P_{AO}	.26	.34	.27	.48
P_{AF}, P_{AO}	.09	.08	.15	.11
P_{AM}, P_{AO}	.12	.05	.14	.17
Adult twins				
P_{T1}, P_{T2} (identical)				
P_{T1}, P_{T2}	.86	.86	.86	.86
(fraternal)	.62	.62	.62	.62
Child twins				
P_{T1}, P_{T2} (identical)				
P_{T1}, P_{T2}	.68	.81	.88	.83
(fraternal)	.63	.73	.79	.71

Note. Adult and child twin correlations are from Loehlin and Nichols (1976) and Wilson (1983), respectively. M, F, O, BM, BF, AM, AF, AO T1, and T2 symbolize nonadoptive mother, nonadoptive father, nonadopted child, biological mother, biological father, adoptive mother, adoptive father, adopted child, twin, and co-twin, respectively.

is instructive, parameter estimates resulting from the simultaneous analysis of all of the data are more informative. Parameter estimates resulting from the fit of the full model (Model 1) to the data are presented in Table 3 for each year the children were tested. As expected, the assortative mating parameters (p and q), selective placement (x_1 through x_4), environmental transmission (f and m), the special twin environments (t_a and t_c), and the square root of heritability for adults (h_a) are fairly constant across the four ages despite differences in sample size. In general, assortative mating is moderate both for biological parents (q) and for adoptive and nonadoptive parents (p); selective placement parameters (x_1 through x_4) are near zero; and shared environmental transmission (f and m) is low. The finding that shared environmental influence between parents and children is low is not unusual: For example, in a study of 94 adoptive families, the IQ correlation between adoptive mothers and their adopted children at 4 years of age was .07 (Fisch, Bilek, Deinard, & Chang, 1976). Studies of older children also have found little resemblance between adoptive parents and adopted children (Plomin, 1986).

In contrast, estimates of the square root of heritability for adults (h_a) and the special twin environments (t_a and t_c) are large. It should be noted that the estimate of the heritability of adult IQ, $h_a^2 = .55$, is somewhat higher than the value that we used in our previous analyses (viz., .50), presumably due to the fact that assortative mating was incorporated into the present analysis. As expected, the heritability of the infant and childhood measures increases as a function of age (.10 to .26), and these estimates are very similar to those used in our previous analysis of a somewhat smaller data set (LaBuda et al., 1986).

As mentioned earlier, the contribution of t to the phenotypic correlation of twins, the shared environmental component of variance, can be estimated as e^2t^2. At 4 years of age, $t_c = .87$ and $h_c = .51$. The proportion of the variance due to all sources of environmental influence (e^2) is estimated by assuming that $h^2 + e^2 = 1$, that is, $e^2 = .74$. Thus, shared environmental influences contribute about .56 to the observed twin correlation. As in the usual estimate of shared twin environments (given identical and fraternal twin correlations of .83 and .71, respectively, at 4 years of age in the Louisville Twin Study), the t parameter in the present analysis suggests that twins share environmental influences salient to cognitive development (e.g., age-linked maturational events with coincidental environmental effects due to contemporaneity) to a substantial degree.

Table 3
Parameter Estimates (± SE) for the Full Model (Model 1) and the Most Parsimonious Model (Model 3) When Children Are Tested at 1, 2, 3, and 4 Years of Age

Parameter	Model 1				Model 3			
	1 year	2 years	3 years	4 years	1 year	2 years	3 years	4 years
p	.22 ± .05	.20 ± .05	.22 ± .05	.23 ± .06	.23 ± .05	.21 ± .05	.23 ± .05	.24 ± .05
q	.32 ± .13	.26 ± .15	.34 ± .13	.34 ± .13	—	—	—	—
x_1	−.01 ± .07	−.02 ± .07	−.03 ± .07	.06 ± .08	—	—	—	—
x_2	.03 ± .07	.08 ± .07	.09 ± .08	.15 ± .08	—	—	—	—
x_3	−.08 ± .14	−.10 ± .15	−.05 ± .14	−.02 ± .15	—	—	—	—
x_4	−.07 ± .16	.04 ± .15	.10 ± .16	.17 ± .16	—	—	—	—
f	.06 ± .06	.07 ± .06	.07 ± .06	−.01 ± .07	—	—	—	—
m	.06 ± .07	.07 ± .07	.10 ± .06	.11 ± .08	—	—	—	—
t_a	.83 ± .02	.83 ± .02	.82 ± .03	.82 ± .03	.83 ± .02	.83 ± .02	.83 ± .02	.83 ± .02
t_c	.80 ± .03	.87 ± .02	.91 ± .01	.87 ± .02	.80 ± .03	.88 ± .02	.92 ± .01	.88 ± .02
h_a	.74 ± .04	.73 ± .04	.74 ± .04	.74 ± .04	.74 ± .04	.74 ± .04	.74 ± .04	.74 ± .04
h_c	.32 ± .25	.41 ± .11	.43 ± .07	.51 ± .09	.33 ± .22	.42 ± .11	.44 ± .07	.52 ± .09
r_G	.42 ± .50	.61 ± .33	.56 ± .28	.75 ± .27	.67 ± .52	.85 ± .31	.79 ± .25	.90 ± .23

Table 4
Fit of Alternative Models

Model	Age of child	Model statistics		Change statistics		
		χ^2	*df*	χ^2	*df*	*p*
1 Full	1	19.45	20	—	—	—
	2	20.35	20	—	—	—
	3	18.79	20	—	—	—
	4	19.73	20	—	—	—
2 $p = q; x_1 = x_2 = x_3 = x_4 = 0$	1	20.67	25	1.22	5	.95
	2	22.27	25	1.92	5	.90
	3	21.44	25	2.65	5	.80
	4	24.62	25	4.89	5	.50
3 $p = q; x_1 = x_2 = x_3 = x_4 = 0; m = f = 0$	1	22.54	27	1.87	2	.50
	2	24.30	27	2.03	2	.50
	3	25.12	27	3.68	2	.20
	4	27.15	27	2.53	2	.30
4 $p = q = 0; x_1 = x_2 = x_3 = x_4 = 0$	1	41.83	26	21.16	1	.001
	2	38.40	26	16.13	1	.001
	3	41.34	26	19.90	1	.001
	4	43.46	26	18.84	1	.001
5 $p = q; x_1 = x_2 = x_3 = x_4 = 0; h_a = 0$	1	292.37	26	271.70	1	.001
	2	296.93	26	274.66	1	.001
	3	295.59	26	274.15	1	.001
	4	303.59	26	278.97	1	.001
6 $p = q; x_1 = x_2 = x_3 = x_4 = 0; h_c = 0$	1	22.55	26	1.88	1	.20
	2	29.66	26	7.39	1	.01
	3	34.23	26	12.79	1	.001
	4	41.14	26	16.52	1	.001
7 $p = q; x_1 = x_2 = x_3 = x_4 = 0; r_G = 0$	1	22.05	26	1.38	1	.30
	2	26.62	26	4.35	1	.05
	3	25.61	26	4.17	1	.05
	4	33.28	26	8.66	1	.01

Note. Model 2 is tested against Model 1, whereas all others are tested against Model 2.

Of major interest to the present study is the childhood-to-adulthood genetic stability parameter (r_G) estimated at each age. It may be seen from Table 2 that this parameter is moderate at 1 year (.42) but increases to .75 by 4 years of age. Although these estimates are somewhat lower than those we have previously reported (LaBuda et al., 1986; Plomin & DeFries, 1985), they nonetheless indicate substantial genetic continuity from infancy and early childhood to adulthood. As is indicated at the top of Table 4, results of goodness-of-fit tests indicate that the full model provides an adequate representation of the data ($p \simeq .50$ at each age).

To test the significance of the parameter estimates and explore the data further, a series of reduced models was fitted to the data. The difference in goodness of fit between two models is itself distributed as chi-square and thus may be used to evaluate various constraints imposed on the reduced model. Because the selective placement parameters were near zero, they were dropped from the model first, and assortative mating was assumed to be comparable for biological and adoptive parents of adopted children; that is, x_1, x_2, x_3, and x_4 were each constrained to be zero in Model 2, and the assortative mating parameters (p and q) were equated. As shown in Table 4, the changes in chi-square from Model 1 to Model 2 indicate the acceptability of these constraints for each age at which the children were tested.

Model 3 retains the constraints of Model 2 but also assumes the absence of transmission due to shared family environment. The changes in chi-square shown in Table 4 indicate that this model is also acceptable at each age. As noted in a previous section, this finding substantiates the absence of genotype–environment correlations for CAP cognitive data. Model 3 is the most parsimonious of the models for which adequate fits to the data were obtained; thus, parameter estimates resulting from the fit of this model to the data for each age are also presented in Table 3. It may be seen that the parameter estimates obtained from the fit of Model 1 to the data are highly similar to those estimated from Model 3, with the exception of r_G, the genetic continuity parameter. The estimates for this parameter are somewhat higher when estimated from Model 3, presumably because all parent–child resemblance is assumed to be due to genetic influences in that model. Although the environmental transmission parameters m and f are small and nonsignificant, they are predominantly positive. Thus, when Model 3 is fitted to the data, estimates of the genetic correlations are somewhat inflated.

Models 4–7 test other parameter estimates. For example, Model 4 retains the constraints of Model 2 as well as assuming that assortative mating is absent. As may be seen from the changes in chi-square listed in Table 4, this hypothesis may be rejected with considerable confidence. A comparison of Model 2 with Model 5 provides a test of the null hypothesis that the heritability of IQ in adulthood is zero. As indicated by the very

large changes in chi-square shown in Table 4, adult heritability is highly significant. Correspondingly, Model 6 tests the hypothesis that the heritabilities of the measure during infancy and childhood are zero. This hypothesis cannot be rejected for children tested at 1 year of age. However, there is evidence for significant heritability at each of the later three ages. Of special interest is Model 7, which constrains the longitudinal genetic correlation to be zero. As may be seen in Table 4, this hypothesis cannot be rejected for infants tested at 1 year of age; however, the longitudinal genetic correlations are significantly different from zero at the later ages. This finding of substantial and significant genetic stability from infancy and early childhood to adulthood confirms and extends our previous findings.

Discussion

The primary objective of the present analysis was to explore childhood-to-adulthood genetic stability in more detail (a) by incorporating the longitudinal genetic correlation into our adoption model of genetic and environmental transmission, (b) by modeling the effects of phenotypic assortative mating both for parent–child and for twin resemblance, and then (c) by applying the model in a simultaneous analysis of published twin correlations and data from a larger sample of CAP adoptive and nonadoptive families than was previously reported on. The model was found to be tractable and was fitted to data from families whose children were tested at 1, 2, 3, and 4 years of age and to correlations for twin pairs tested at similar ages.

By inspection of the parameter estimates, as well as tests of specific hypotheses resulting from comparisons of goodness of fits of alternative models, several general findings emerged: First, it was found that assortative mating is moderate (about .25) and similar for biological and adoptive parents of adopted children and for nonadoptive parents. Second, selective placement is near zero, and environmental transmission parameters are small and nonsignificant. Third, the correlation between twin pairs due to shared environmental influences (t^2) is large and highly significant, indicating that special factors such as contemporaneity add importantly to twin resemblance. Fourth, the heritability of IQ in adults is large (about .55) and highly significant. Fifth, heritability of IQ in children is lower, but increases from .10 at 1 year of age to .26 at 4 years of age. And sixth, estimates of the longitudinal genetic correlation increase from a nonsignificant .42 at 1 year of age to a highly significant .75 at 4 years of age. Although somewhat smaller than our previously reported estimates of genetic correlations, the results of the present study suggest that genetic continuity from infancy and early childhood to adulthood is substantial.

Substantial longitudinal genetic correlations suggest that genetic factors that cause individual differences in infancy and childhood covary highly with genetic causes of individual differences in adulthood. This finding is all the more remarkable because infant scores are measures of developmental rate, not final level—all human beings eventually are able to stick the yellow pegs in the pegboard of the Bayley test. Genetic stability does not imply that the physiological or psychological processes that underlie scores on cognitive tests are the same in childhood and adulthood. For example, the same genes would not necessarily be transcribed at the two ages even if the age-to-age genetic correlation were 1.0: The relevant genes in childhood might no longer be actively transcribed, but their structural legacy (e.g., differences in neural networks) could produce the genetic correlation between childhood and adulthood. Similarly, the psychological processes underlying the measures in childhood and adulthood need not be isomorphic. For instance, "childhood genes" might affect rate of language acquisition, whereas "adult genes" might affect symbolic reasoning. However, it is also possible that similar cognitive processes are involved at both ages, as has been argued in the case of infant novelty preference and its relation to later IQ (Fagan, 1982, 1985):

> To predict later intelligence, the task is to tap processes during infancy which are similar in kind to processes known to be related to later intelligence. On later intelligence tests, children are asked, for example, to discriminate among stimuli, to retain new information, to identify similarities, and to categorize. Over the last decade, methodological advances and empirical studies in the field of infant visual perception and recognition memory have made it possible to ask an infant to exhibit discrimination, retention, identification, and categorization. (Fagan, 1982, p. 22)

One important direction for future research is to identify the processes in childhood and adulthood through which genetic stability occurs.

One question that arises from finding such substantial genetic stability is: How can genetic stability be so high when genetic variance increases so dramatically from about .20 in early childhood to over .50 in adulthood? For example, if the genetic correlation were 1.0, any increase in heritability from childhood to adulthood would present a paradox. Although the magnitude of the genetic correlation in the present study is not necessarily discrepant with the increase in heritability, it is interesting heuristically to consider the possibility that the effects of genes that cause individual differences early in life become amplified during development. In other words, although genetic variation accounts for a smaller proportion of the observed variance of cognitive ability in infancy and early childhood than in adulthood, the effects of many of the same genes may be manifested increasingly as development proceeds.

How could the effects of the same genes be amplified during cognitive development? Consider the genetic control of neurological development as a hypothetical example. Let us suppose that genetic differences among infants are responsible for differences in the complexity of dendritic spines as they develop during the first few years of life and that information-processing capability is a function of the complexity of dendritic spines. Such structural differences might result in only minor functional differences among infants due to their limited information-processing capability. However, functional differences could be manifested to a greater extent during childhood as more information is processed. In this manner, genetic differences would contribute only negligibly to variance in Bayley MDI scores but would increase in their relative contribution as development proceeds. Although the genetic variance is less during early development, the genetic correlations from infancy and early childhood to adulthood could be substantial because the effects of many of the same genes are being expressed at each stage of development.

Another implication of the genetic stability model is that it

predicts some phenotypic stability from early childhood to adulthood. As mentioned in the introduction, the genetic contribution to phenotypic stability is a function of the longitudinal genetic correlation between the two ages and the square roots of the heritabilities at each age. If either of the heritabilities is low, the genetic contribution to phenotypic stability (i.e., $h_a h_c r_G$) will also be low. Results of the present study suggest that the genetic contribution to phenotypic stability between 3 years of age and adulthood is .18. Between 4 years and adulthood, the genetic contribution to observed cognitive stability is .28. Longitudinal studies of IQ have yielded stability correlations of about .40 and .50, respectively, from 3 and 4 years of age to adulthood (Bayley, 1954; Honzik, MacFarlane, & Allen, 1948). Thus, the results of the present study suggest that about half of the phenotypic stability between childhood and adulthood is mediated genetically.

The genetic contribution to phenotypic correlations between childhood and adulthood (about .20 to .30) is not large enough to be of practical utility in predicting adult IQ, at least with our current measures. However, the present findings support an emerging theory of the origins of individual differences in cognitive development. Genetic differences among children account for only 10% to 15% of the variance assessed by the Bayley MDI at 1 and 2 years and only 15% to 25% of the variance assessed by the Stanford-Binet at 3 and 4 years. Nonetheless, the surprising aspect of these results is that most of this genetic variance covaries highly with adult IQ. As mentioned earlier, an important direction for future research is to identify those genetically influenced psychological and physiological processes that correlate so highly between childhood and adulthood. Finally, although the magnitude of the genetic correlations from childhood to adulthood is surprisingly high, they are less than 1.0. Thus, these data point to both genetic change and genetic continuity during cognitive development. A little bit of genetic change might go a long way toward altering the course of development. For this reason, developmental behavioral genetics must continue to focus on genetic change as well as continuity.

References

Bayley, N. (1954). Some increasing parent–child similarities during the growth of children. *Journal of Educational Psychology, 45,* 1–21.

Bayley, N. (1969). *Manual for the Bayley Scales of Infant Development.* New York: Psychological Corporation.

Bouchard, T. J., & McGue, M. (1981). Familial studies of intelligence: A review. *Science, 212,* 1055–1059.

CERN (1977). *MINUIT: A system for function minimization and analysis of the parameter errors and correlations.* Geneva: Author.

DeFries, J. C., Plomin, R., Vandenberg, S. G., & Kuse, A. R. (1981). Parent–offspring resemblance for cognitive abilities in the Colorado Adoption Project: Biological, adoptive, and control parents and one-year-old children. *Intelligence, 5,* 245–277.

Eaves, L. J., Long, J., & Heath, A. C. (1986). A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behavior Genetics, 16,* 143–162.

Fagan, J. F. III (1982). A visual recognition test of infant intelligence. *Infant Behavior and Development, 5,* 22. (Abstract)

Fagan, J. F. III (1985). A new look at infant intelligence. In D. K. Detterman (Ed.), *Current topics in human intelligence: Vol. 1. Research methodology* (pp. 223–246). Norwood, NJ: Ablex.

Fisch, R. O., Bilek, M. K., Deinard, A. .C., & Chang, P. N. (1976). Growth, behavioral, and psychologic measurements of adopted children: The influences of genetic and socioeconomic factors in a prospective study. *Behavioral Pediatrics, 89,* 494–500.

Fulker, D. W., & DeFries, J. C. (1983). Genetic and environmental transmission in the Colorado Adoption Project: Path analysis. *British Journal of Mathematical and Statistical Psychology, 36,* 175–188.

Hauser, R. M., & Featherman, D. L. (1977). *The process of stratification: Trends and analysis.* New York: Academic Press.

Honzik, M. P., MacFarlane, J. W., & Allen, L. (1948). Stability of mental test performance between 2 and 18 years. *Journal of Experimental Education, 17,* 309–322.

LaBuda, M. C., DeFries, J. C., Plomin, R., & Fulker, D. W. (1986). Longitudinal stability of cognitive ability from infancy to early childhood: Genetic and environmental etiologies. *Child Development, 57,* 1142–1150.

Li, C. C. (1975). *Path analysis: A primer.* Pacific Grove, CA: Boxwood Press.

Loehlin, J. C., & Nichols, R. C. (1976). *Heredity, environment, and personality.* Austin: University of Texas Press.

McCall, R. B. (1979). The development of intellectual functioning in infancy and the prediction of later IQ. In J. D. Osofsky (Ed.), *Handbook of infant development* (pp. 707–741). New York: Wiley-Interscience.

Plomin, R. (1986). *Development, genetics, and psychology.* Hillsdale, NJ: Erlbaum.

Plomin, R., & DeFries, J. C. (1980). Genetics and intelligence: Recent data. *Intelligence, 4,* 15–24.

Plomin, R., & DeFries, J. C. (1981). Multivariate behavioral genetics and development: Twin studies. In L. Gedda, P. Parisi, & W. E. Nance (Eds.), *Progress in clinical and biological research, Vol. 69B, Twin research 3: Intelligence, personality, and development* (pp. 25–33). New York: Liss.

Plomin, R., & DeFries, J. C. (1985). *Origins of individual differences in infancy: The Colorado Adoption Project.* Orlando, FL: Academic Press.

Plomin, R., DeFries, J. C., & McClearn, G. E. (1980). *Behavioral genetics: A primer.* San Francisco: Freeman.

Rice, T., Fulker, D. W., & DeFries, J. C. (1986). Multivariate path analysis of specific cognitive abilities in the Colorado Adoption Project. *Behavior Genetics, 16,* 107–125.

Scarr, S., & Carter-Saltzman, L. (1979). Twin method: Defense of a critical assumption. *Behavior Genetics, 9,* 527–542.

Skodak, M., & Skeels, H. M. (1949). A final follow-up on one hundred adopted children. *Journal of Genetic Psychology, 75,* 85–125.

Terman, L. M., & Merrill, M. A. (1973). *Stanford-Binet Intelligence Scale: 1972 norms edition.* Boston: Houghton Mifflin.

Wilson, R. C. (1983). The Louisville Twin Study: Developmental synchronies in behavior. *Child Development, 54,* 298–316.

Received December 9, 1985
Revision received April 3, 1986 ■