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A Twin Study of Differentiation of Cognitive Abilities in Childhood

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The differentiation hypothesis in cognitive development states that cognitive abilities become progressively more independent as children grow older. Studies of phenotypic development in children have generally failed to produce convincing support for this hypothesis. The aim of the present study is to investigate the issue of differentiation at the genetic and environmental level. Six psychometric measures assessing verbal and nonverbal cognitive abilities were administered to 209 Dutch twin pairs at ages 5, 7, and 10 years. Longitudinal results provided little evidence for the differentiation hypothesis. Stability in subtest performance is due mainly to genetic influences. The shared environment contribution to phenotypic stability is small. The unique environment contributes to age-specific variance only.

KEY WORDS: Differentiation; cognitive abilities; heritability; longitudinal; childhood.

INTRODUCTION

The structure of individual differences in cognitive abilities during development has received considerable attention in cognitive developmental theory (Schaie, 1994; Vernon, 1976). One important hypothesis, dating back to Garrett (1946; see also Carroll, 1993; Reinert, 1970; Wohlwill, 1973), states that cognitive abilities become increasingly more differentiated during development.[4] In operational terms, this means that the intercorrelations among psychometric measures of ability decrease during normal cognitive development in children. So far, support for this hypothesis has been poor. In an early review of about 60 factor analytic studies, Reinert (1970) suggested that a trend toward increased differentiation was present. However, this conclusion was based on the selection of studies that actually reported a change. In a more recent review, Carroll (1993) failed to find clear evidence for the differentiation of abilities. More recent cross-sectional studies of the changes in abilities in both children and adults are consistent with Carroll's finding in that age-related differentiation of cognitive abilities was not observed (Bickley *et al.,* 1995; Deary *et al.,* 1996; Juan-Espinosa *et al.,* 2000; Werdelin and Stjernberg, 1995).

Although support for the differentiation is apparently weak, comparative factor analytic studies have two drawbacks. First, studies vary in the definition of differentiation within the factor model and in the criteria used to evaluate such change. A number of approaches have been suggested to assess factorial invariance. A comprehensive summary of theories of factorial invariance is given in Cunningham (1991; see also Horn, 1991). Factorial change and stability across time may be judged by criteria that vary in restrictiveness. A second drawback is the limitation of most studies to analyses at the phenotypic level. It has long been recognized in behavior genetics that the phenotypic covariance structure does not always reflect the latent covariance

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[4] As pointed out by a reviewer, another way to view differentiation is in terms of the divergence during development of phenotypic mean trends of, for instance, fluid and crystallized intelligence. Mean trends cannot be addressed with the present data as the IQ used is tailored to each age group. This gives rise to age-standardized test scores.

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structures due to genetic and(or) environmental factors (Plomin, 1983). The failure to establish phenotypic differentiation does not necessarily imply the absence of differentiation at the genetic and environmental level. Many papers have been devoted to the development of longitudinal behavior genetic models (Boomsma and Molenaar, 1987; DeFries and Fulker, 1986; Meyer *et al.,* 1999; Plomin and DeFries, 1981; McArdle, 1986; McArdle and Goldsmith, 1990). Several behavior genetic studies have addressed the issue of stability and change in cognitive development (Bishop *et al.,* 2003; Bartels *et al.,* 2002; Cardon and Fulker, 1993; Eaves *et al.,* 1986; Fulker *et al.,* 1993; Hay and O'Brien, 1983; Hewitt *et al.,* 1988; Plomin *et al.,* 1994; Reznick *et al.,* 1997; Wilson, 1983). Although these studies vary in the age range of the samples, psychometric instruments used to assess cognitive abilities, and statistical methods, the results show a considerable degree of agreement. Overall, genetic effects acting on specific cognitive abilities display both developmental stability (variance common to different ages) and change (age-specific variance). Genetic effects become increasingly more important in explaining individual differences as children grow older. In contrast to the differentiation hypothesis, the correlation between genetic factors that represent different domains of intelligence appear to increase with age (Casto *et al.,* 1995; Price *et al.,* 2000). The shared environmental effects contribute both to the correlation among psychometric subtests and to the correlation among these subtests over time. However, these effects diminish as children grow older (Patrick, 2000). Unshared environmental effects are important at every age but show little to no stability over time. Thus, with respect to the genetic and environmental contributions, no simple picture emerges with respect to the differentiation hypothesis. However, most research has involved univariate measures of cognitive abilities and has usually focused on the analyses of general intelligence (e.g., Bartels *et al.,* 2002; Bishop *et al.,* 2003). The number of behavior genetic studies incorporating a longitudinal design and multivariate measures is limited.

In the present study, we investigate the differentiation hypothesis using twin data. A multidimensional test of cognitive abilities was administered to 209 twin pairs at ages 5, 7, and 10 years. In part these are the same data as analyzed in Bartels *et al.,* (2002) and Boomsma and Van Baal (1998). However, rather than modeling a single repeated measure of general intelligence, here we model repeated measures of six specific cognitive abilities. We consider the least strict version of factorial invariance, namely configural invariance (Horn and McArdle, 1992; Schaie *et al.,* 1998). We present detailed results relating to the changes over time in genetic and environmental effects and to their contribution to the phenotypic stability. In these longitudinal analyses, we hope to establish the contributions, if any, of genetic and environmental effects to differentiation.

METHODS

Sample

This study is part of a longitudinal study of the development of intelligence and problem behavior. The sample was obtained from the Netherlands Twin Registry, which is maintained at the Vrije Universiteit in Amsterdam. The registry contains around 50% of all twins born in the Netherlands since 1986 (Boomsma, 1998). The recruitment of the initial sample of 209 twin pairs took place on the basis of age, zygosity, and city of residence. Mean ages at the three measurement occasions were 5.3 years (80% ranging from 5 years, 1 month to 5 years, 6 months), 6.8 years (80% ranging from 6 years, 6 months to 7 years, 1 month), and 10 years (80% ranging from 9 years, 11 months to 10 years, 1 month). All children had started elementary school at the beginning of the study. Zygosity of the same-sex twins in the initial sample was established by either blood group polymorphism (137 pairs) or DNA analyses (24 pairs), and in 9 twin pairs by physical resemblance as assessed by the test administrator. The sample comprised 47 monozygotic (MZ) female pairs, 37 dizygotic (DZ) female pairs, 42 MZ male pairs, 44 DZ male pairs, and 39 DZ opposite-sex pairs. At the three measurement occasions the number of participating pairs was 209 (age 5), 192 (age 7), and 197 (age 10). Five families did not participate at both age 7 and age 10. At the age of 5, one child failed one subtest due to difficulties during testing. One twin pair was assigned missing values on the verbal subtests measured at all three ages, because they suffered from hearing difficulties. A sample of 184 pairs provided complete data on all subtests at all three ages.

The demographic characteristics of the employed twin mothers agree with those in the norm population (Central Bureau of Statistics, 2001). However, the occupational status of the employed twin fathers is not quite representative of the Dutch population. The twin sample is somewhat underrepresented in the lower end of the socioeconomic scale.

Procedure

When the twins were 5 and 7 years old, they participated in a combined study of the development of cognitive abilities and brain function (Van Baal *et al.,* 1996). At these occasions, the children and their parents visited the laboratory at the university. While one of the twins participated in the electro-physiological experiment, the co-twin completed the intelligence test. At the third measurement occasion (when the twins were about 10 years old), parents were invited by letter to make an appointment for testing in their own home or at the university. The majority of the families preferred testing at home (70%). No difference in full IQ score was observed between children tested at home and children tested at the university.

Intelligence Test

A shortened version of the Revised Amsterdam Children Intelligence Test, or the RAKIT, to use the Dutch abbreviation (Bleichrodt *et al.,* 1984) was administered at all three occasions. The RAKIT is a Dutch psychometric intelligence test for children, with subtests covering a broad spectrum of intellectual capabilities. The test is designed for children between about 4 and 11 years. The items in the subtests are organized by both age level and difficulty, and are arranged in overlapping sets. Each set is tailored to a specific age group. The overlap in items between the three measurement occasions decreases with the increasing interval between ages. The shortened version of the RAKIT includes six subtests measuring verbal and nonverbal abilities:

Exclusion measures understanding of figural classes or similarities. The child is presented with four figures, of which three figures have some common characteristic. After establishing the relationship between these figures, the child is asked to identify the odd one out. Total score is the number of correct decisions. Since children of older ages are shown more items, the raw total score is not comparable across ages.

Discs is a measure of spatial orientation and speed of spatial visualization. This test consists of a wooden board with pins fixed in a particular shape and of small wooden discs with holes in a particular shape. The child is required to fit the right discs onto the pegs at the right place and in the proper position as quickly as possible. The score is the total time required to place all the blocks. Children at age 5 were given only 12 discs out a set of 18 discs.

Hidden Figures requires visual analysis, pattern recognition, matching, and the ability to ignore irrelevant stimuli. A large drawing consisting of many lines and six smaller simplified drawings are shown to the child. The child has to find out which one of the smaller drawings is present in the more complex drawing. The number of correct judgments is the total score. Besides a difference in the number of items presented, children of older age are allowed less time to reach a decision.

Verbal Meaning assesses knowledge of concepts, and verbal conceptualization. In this vocabulary test, the child is shown four drawings representing familiar objects or acts. The tester reads the name of one object or act aloud and the child is asked to choose which of the drawings best suits the word. The number of correct items is the total score. Older children are presented with a large number of items than the younger children.

Learning Names is a verbal memory task, which is used to assess the ability to learn and recall names with pictures. The tester shows the child a book containing pictures of cats and butterflies and states the proper (fictional) name of each animal. At times, the tester explains why a given animal was given a particular name. The child has to recall the names of the animals within a certain time limit when shown the pictures. Five-year-old children are shown fewer pictures than the older children. The total score is the number of correct responses.

Idea Production is a measure of verbal fluency. Several simple questions are posed, like "what items can you put in your coat pocket?". The child has to respond by producing as many answers as possible within a certain time limit. The questions asked, the maximum time for answering, and the evaluation of the answers is the same for each age group. The number of acceptable answers forms the total score.

Age-corrected norms are based on an interval of four months. Raw subtest total scores are corrected for age and transformed into standardized scores with a mean of 15 and a standard deviation of 5. The reliabilities (internal consistency) of the subtests are stable across age, varying from .69 to .90. The concurrent validity with the WISC-R is .86 for total IQ (see also Bartels *et al.,* 2002).

Modeling of the Data

Standard genetic (co)variance modeling of twin data was used to estimate the size and the structure of the genetic and environmental effects (Neale and Cardon, 1992). Phenotypic variance is assumed to be due to shared environmental (C), unique environmental (E), and additive genetic (A) effects (Plomin *et al.,* 2001). Although meta-analyses suggest that nonadditive effects do contribute to individual differences in IQ test scores (15% of the variance according to Daniels, Devlin, and Roeder, 1997), we do not consider possible nonadditive genetic effects (dominance, epistasis) here. There is in fact little evidence of dominance effects in the present sample (Bartels *et al.,* 2002; Boomsma and Van Baal, 1998), while the correlations do suggest the presence of shared environmental effects (see Table I below). In addition, the present sample sizes do not confer sufficient power to detect relatively small nonadditive genetic effects (Eaves, 1972; Posthuma and Boomsma, 2000; Rietveld *et al.,* in press). In view of these considerations, and the fact that the present twin design does not provide sufficient information to fit a model including C and dominance effects simultaneously, we limit our attention to models including A, C, and E.

On the individual level an observed phenotype (P) can be represented as a function of a subject's additive genetic, common environmental, and unique environmental deviations:

$$P_{ij} = aA_{ij} + cC_{ij} + eE_{ij}$$

where $i = 1,2$ (members within a twin pair) and $j = 1, \ldots N$ (number of twin pairs). The coefficients *a, c,* and *e* are population invariant and can be considered as regression coefficients or factor loadings of P on the latent factors A, C, and E. If the latent factors are constrained to have unit variance (Neale and Cardon, 1992), the decomposition of the phenotypic variance is

$$V_p = a^2 + c^2 + e^2$$

The different degree of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs is used to estimate the contributions of the latent factors to the phenotypic variation in cognitive abilities. Similarity (covariances) between MZ twins can be due to additive genetic influences (a^2) and/or environmental influences that are shared by both twins (c^2). DZ covariances equal $1/2a^2$, in addition to the shared environmental influences (c^2). Environmental influences that make MZ and DZ twins different from one another are unique environmental influences (e^2). If multiple measures on the same twins are available, the association between measurements is also analyzed as a function of A, C and E (Boomsma and Molenaar, 1986; Martin and Eaves, 1977), where A, C, and E themselves may be uni- or multidimensional.

Model Fitting

All models were fit using the program Mx (Neale *et al.,* 1999). Parameter estimates were obtained by maximizing the raw data likelihood. Given the presence of missing data, albeit limited in number in the present study (8% at age 7; 6% at age 10), this method is preferable to the analysis of covariance matrices (e.g., Wothke, 2000). To test various hypotheses concerning the genetic and environmental covariance structures, we fitted a number of models to the longitudinal, multivariate data. Some of these models were nested, in the sense that one model was derivable from a second, less restrictive model by fixing selected parameters in the second model to equal certain values or by imposing equality constraints on selected parameters in the second model. Given this nesting, minus twice the difference in log-likelihood between the models may be used to evaluate the imposed restrictions. This statistic is asymptotically chi-square distributed if the restrictions are tenable. The number of degrees of freedom of this test statistic equals the difference in the number of estimated parameters between the two models. If the chi-square test is not significant, we consider the restrictions tenable (e.g., Loehlin, 1992).

Two series of model fitting took place; analyses of the cross-sectional data followed by analyses of the longitudinal data. The aim of the cross-sectional analyses was to provide an initial insight into the multivariate data, to test for sex differences in covariance structure, and to obtain a starting point for the longitudinal analyses. The cross-sectional data were initially modeled as a Cholesky decomposition. Sex differences were evaluated by constraining the covariance structures to be equal for boys and girls. Cross-sectional analyses proceeded with the specification of a more parsimonious and hypothesis-driven factor model, derived from model-fitting results obtained in our previous study of cognitive abilities at age 5 (Rietveld *et al.,* 2000). The genetic covariance structure was specified as a correlated two-factor structure with subtest-specific factors. The shared environmental covariance structure was modeled as one general factor, without subtest-specific factors. The unique environmental covariance structure was modeled

as subtest-specific factors only. This model served as a reference for the final three analyses, in which the importance of the latent factors was evaluated by constraining the relevant factor loadings at zero.

To establish that the same trait is being measured at different ages, we imposed the minimal condition of configural invariance. Configural invariance implies that the factor pattern is invariant over time but that the factor loadings may vary in size. Below we do consider the more restrictive hypothesis that factor loadings are in fact equal over time. Configural invariance does not imply stability, in that similar factor patterns at each age do not imply high correlations over time between the genetic and environmental factors. The issue of stability was addressed by model fitting of the longitudinal dataset. These analyses were initiated with the specification of an autoregressive, or simplex model (Boomsma and Molenaar, 1987; Guttman, 1954). This particular model was chosen because it provides a straightforward account of the stability of individual differences over time. Figure 1 represents a path diagram of the simplex model that was applied to the genetic covariance matrix. The model supposes that genetic variance at any age is partly a function of genetic variance at the previous age, via paths *t*, and partly determined by other genetic factors, via paths *i*. The former paths represent transmission effects and the latter represent innovation effects. The Nonverbal and Verbal innovation factors are correlated within age 7 and 10 (paths $r(I_{nv}, I_v)$). To accommodate shared variance between the genetic Nonverbal and Verbal factor at age 5, we added an interfactor correlation, path $r(A_{nv}, A_v)$.

Figure 2 illustrates the environmental parts of the model. Since the shared environmental structure was originally modeled as a single common factor, only two transmission parameters were added to the model to account for the variance transmitted from age 5 to 7 and from age 7 to 10 (paths *t*). Innovation effects or unshared variance at age 7 and age 10 are represented by paths *i*. The unique environmental contributions were found specific in origin. As with the genetic specifics, each subtest-specific factor was allowed to correlate over time in the longitudinal model.

Having established that this model was acceptable by comparison with a Cholesky decomposition, longitudinal analyses proceeded by testing various hypotheses within this model. First, the correlations that were specified between the genetic specific factors and the unique environmental specific factors were constrained at zero. In so doing we investigated whether the stability in subtest scores is accounted for completely by the common factors. Second, the importance of the genetic and familial environmental group factors was evaluated by constraining the relevant factor loadings at zero. Third, it was explored whether the influences of the genetic group factors and the shared environmental group factors were of equal magnitude at different ages.

RESULTS

Phenotypic Analyses

Evaluation of skewness and kurtosis of each subtest measured at each age indicated no serious departure from normality. Visual inspection revealed no outliers, so we included all data in subsequent analyses. Mean differences due to gender, zygosity, and birth order of the twins were found to be absent. Nonparticipation at ages 7 ($N = 17$) and 10 ($N = 12$) was not related to the twins' full-score IQ at age 5. By confirmatory age-specific factor analyses using structural equation modeling, the covariance matrix of the six subtest scores was best described by an oblique two-factor model with subtest specifics (age 5, $\chi^2 = 8.09$ (8), $p = .43$; age 7, $\chi^2 = 2.74$ (8), $p = .95$; age 10, $\chi^2 = 11.35$ (8), $p = .18$). The subtests Verbal Meaning, Learning Names, and Idea Production loaded on one common factor. Exclusion, Discs, and Hidden Figures loaded on the other common factor. The common factors thus represented verbal and nonverbal (spatial) abilities. The consistency of these results across measurement occasions suggests that configural invariance at the phenotypic level is tenable.

Cross-Sectional Genetic Analyses

A first impression of the relative magnitude of genetic and environmental influences is obtained by the inspection of twin correlations. Because correlations of male, female, and opposite twin pairs did not differ by formal testing, they were pooled. Correlations for each subtest at the three ages are provided in Table I.

Although there is considerable variation in the observed correlations, the MZ twin correlations are consistently larger than the DZ twin correlations. With the possible exception of scores on the Learning Names and Idea Production subtests at age 10, nonadditive genetic effects appear to be absent. The mean correlations show that MZ twins increase and DZ twins decrease in similarity over time. This suggests that genetic effects gain importance in explaining phenotypic individual differences. Shared environmental effects appear to decrease over time. Because the MZ correlations are well

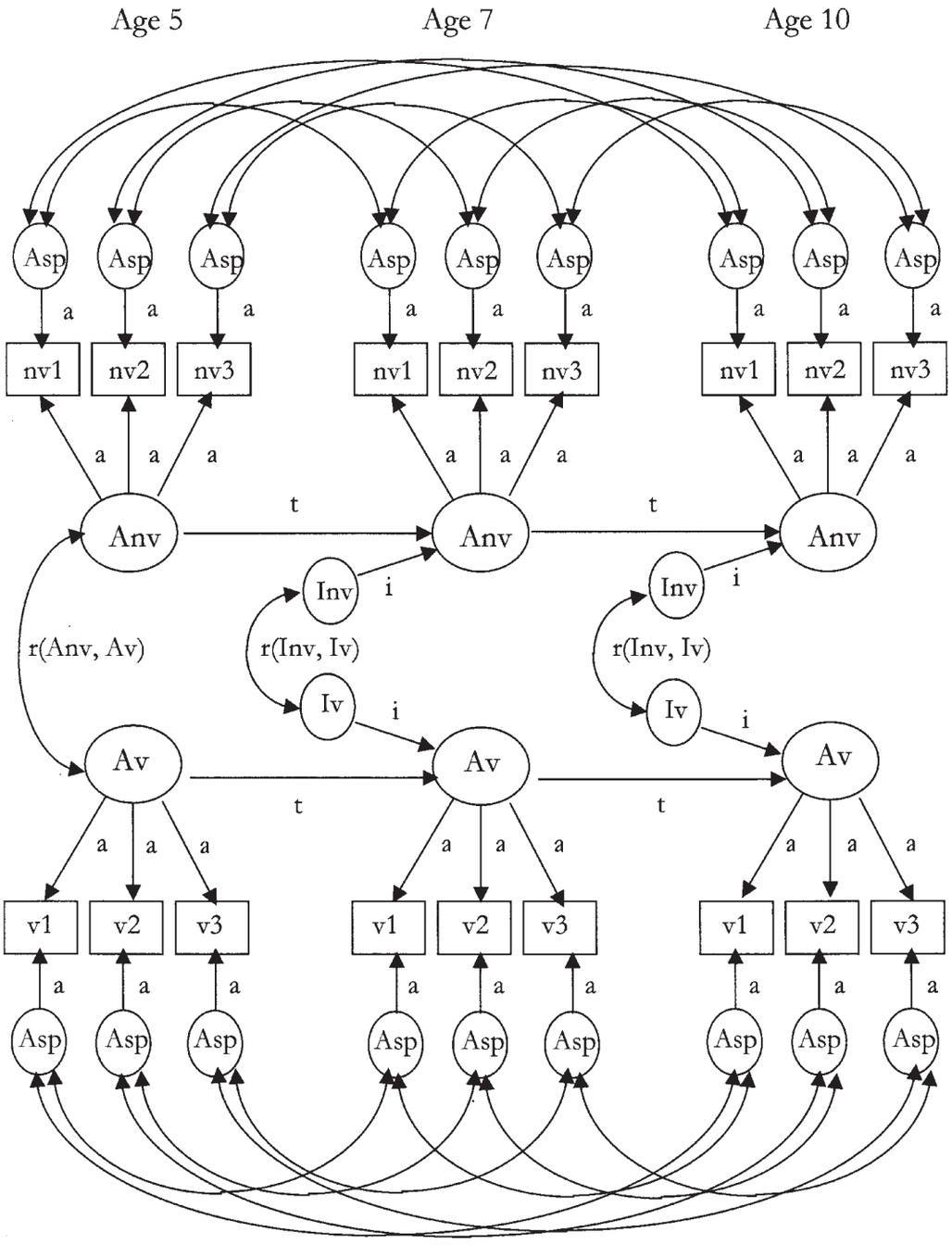

Fig. 1. Path diagram depicting the genetic part of the model. The simplex structure with correlated specifics (A_{sp}) suggests that both genetic common factors and subtest-specific residuals are needed to explain variance in subtest performance. The correlation between the Nonverbal (A_{nv}) and Verbal (A_v) factor at age 5, and the correlation between innovation factors (I) at age 7 and age 10 accommodate the covariation between nonverbal and verbal subtests. The variance that is shared across age is accounted for by autoregressive parameters (*t*), as specified between the common factors, and by the correlations as specified between the subtest-specific residuals. Each individual parameter (*a, t, i,* and *r*) is freely estimated. Exclusion, nv_1; Discs, nv_2; Hidden Figures, nv_3; Verbal Meaning, v_1; Learning Names, v_2; Idea Production, v_3.

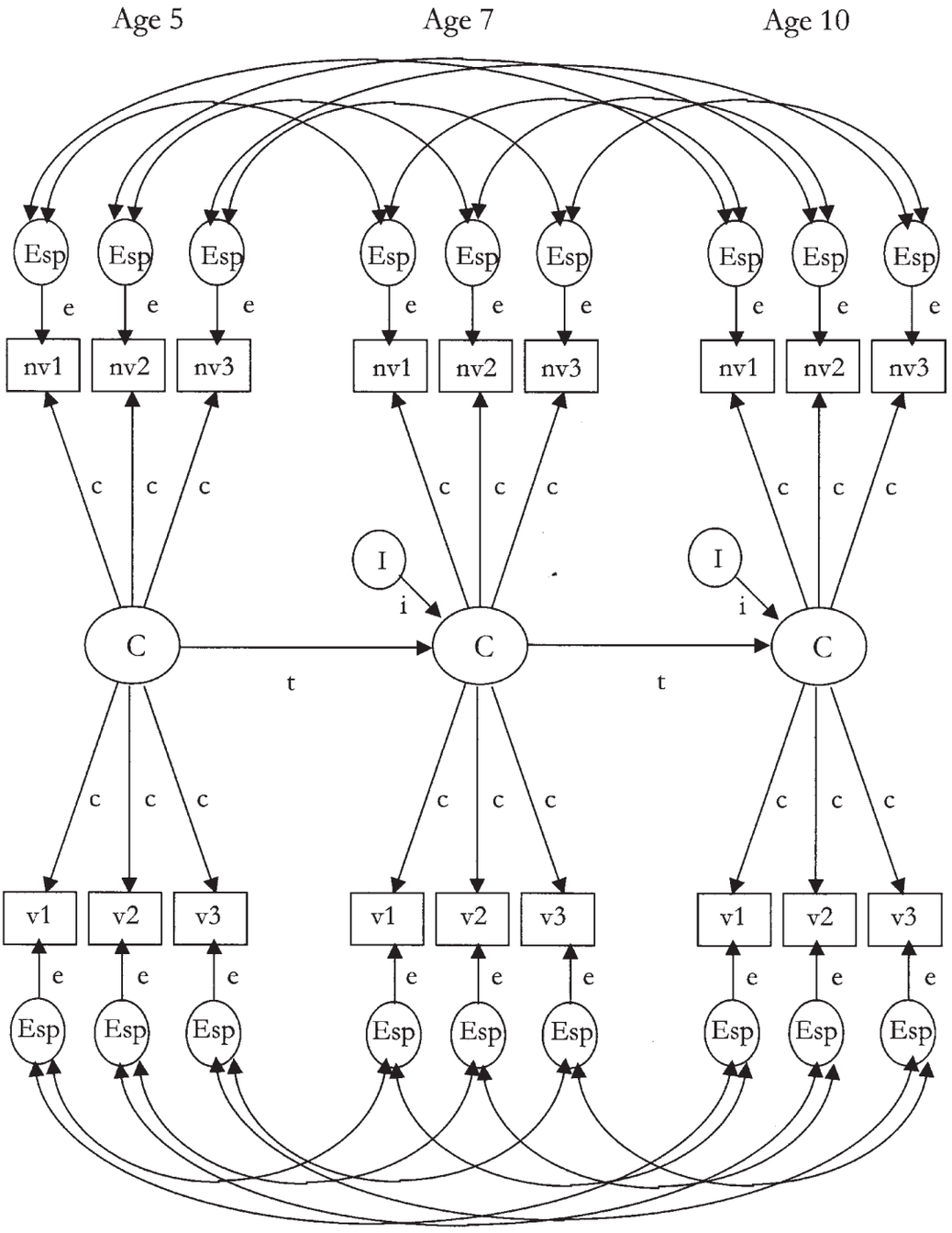

Fig. 2. Path diagram depicting the environmental part of the model. The simplex structure with three shared environmental common factors (C) suggests that the verbal and nonverbal subtests share all their age-specific variance. The variance that is shared among verbal and nonverbal subtests across age is accounted for by the autoregressive parameters (*t*) specified between the general C factors. Innovation factors (I) suggest time-specific variance for C. The subtest-specific unique environmental factors (E_{sp}) explain all variance in subtest performance at a specific age. The correlations as specified between the subtests-specific E factors account for variance shared by the same subtest across age. Each individual parameter (*c, e, t,* and *i*) is freely estimated. Exclusion, nv_1; Discs, nv_2; Hidden Figures, nv_3; Verbal Meaning, v_1; Learning Names, v_2; Idea Production, v_3.

Table I. Pearson Correlations for MZ and DZ for 6 Subtests, Measured at Age 5, 7 and 10

	Age 5		Age 7		Age 10	
	MZ $N = 89$	DZ $N = 120$	MZ $N = 79$	DZ $N = 113$	MZ $N = 82$	DZ $N = 115$
Exclusion	.60	.38	.42	.29	.65	.40
Discs	.44	.23	.61	.30	.39	$.18^{ns}$
Hidden Figures	.63	.43	.53	.32	.62	.31
Verbal Meaning	$.61^{a}$	$.46^{a}$	$.32^{a}$	.22	$.76^{a}$	.48
Learning Names	$.73^{a}$	.44	$.69^{a}$	.50	$.81^{a}$	.30
Idea Production	$.63^{a}$	.38	$.52^{a}$	.44	$.68^{a}$	$.17^{ns}$
Mean	.61	.39	.52	.35	.65	.31

Note: MZ = monozygotic twins; DZ = dizygotic twins; N = number of twin pairs.
[a] Based on $N - 1$ pairs; *ns* = nonsignificant at $p < .05$.

below unity, moderate to large unique environmental effects are expected to be present at each age (Plomin *et al.,* 2001). Systematic differences in results between the verbal and nonverbal subtests are absent. In Table II a summary of the cross-sectional analyses is given.

The nonsignificant difference between Model 1 (Cholesky decomposition with sex differences) and Model 2 (Cholesky decomposition without sex differences) suggests that sex differences are absent at all three ages. This result is consistent with other studies on general IQ in which the same twins were included (Bartels *et al.,* 2002; Boomsma and Van Baal, 1998). Model 3 (different factor structure for A, C, and E) gives an acceptable description of the data at all ages. Indicated by the significant detoriation in goodness of fit, we found that both the genetic common factors (Model 4, drop A correlated factors) and the genetic specific factors (Model 5, drop A correlated specific factors) are important at every age. The one-factor shared environmental structure accounts for a significant part of the total variation at ages 5 and 7, but not at age 10 (Model 6, drop C). On the basis of the agreement in model fits across age, we note that configural invariance appears to be tenable, with the exception of the apparent absence

Table II. Cross-Sectional Analyses

	Age 5		Age 7		Age 10	
Model	-2 LL (df)	χ^2 (df)	-2 LL (df)	χ^2 (df)	-2 LL (df)	χ^2 (df)
1. Cholesky decomposition ACE with sex differences	13785.9 (2315)		13192.1 (2112)		13307.2 (2172)	
2. Cholesky decomposition ACE, no sex differences	13840.5 (2378)	54.5 (63)	13263.1 (2175)	71.0 (63)	13361.0 (2235)	53. (63)
3. A: correlated two-factor structure + specifics C: one-factor structure E: specifics only	13868.9 (2416)	28.4 (38)	13304.1 (2213)	40.9 (38)	13395.1 (2273)	34.1 (38)
4. Drop A correlated factors, keep C and E	13929.0 (2423)	60.0 (7)*	13392.4 (2220)	88.3 (7)*	13523.4 (2280)	128.2 (7)*
5. Drop A specifics, keep C and E	13958.1 (2422)	89.1 (6)*	13325.8 (2219)	21.7 (6)*	13444.3 (2279)	49.1 (6)*
6. Drop C, keep A and E	13901.2 (2422)	32.2 (6)*	13317.2 (2219)	13.1 (6)*	13402.6 (2279)	7.44 (6)

Note: LL = log-likelihood, χ^2 = chi-square, difference in -2LL between nested models. Model 2 is nested with Model 2; Model 3 is nested with Model 2; Model 4, 5, and 6 are nested with Model 3. * = significant at $p < .05$.

of the shared environmental factor at age 10. The genetic structure displays a close resemblance to the phenotypic structure in that the subtests cluster around two correlated genetic factors, which are interpretable as the verbal and nonverbal abilities. We limit the detailed discussion of results in terms of the actual parameters to the longitudinal analyses.

Longitudinal Genetic Analyses

Although the shared environmental factor appeared to be absent at the age of 10, it was decided to retain this factor in the initial stage of the longitudinal analyses. Model fit indices are given in Table III.

The log-likelihood difference between Model 1 (Cholesky decomposition) and Model 2 (simplex structure) suggested that the longitudinal model (see Figs. 1 and 2) provided an acceptable point of departure. Three series of analyses were performed. First, the correlations between the subtest-specific factors were evaluated. The correlations obtained after fitting the factor model to the longitudinal data ranged from −1.00 to 1.00 for the genetic specifics and from −.13 to .23 for the unique environmental specifics. The worsening in goodness of fit of Models 3 (drop A correlations between specifics at ages 5 and 7), Model 4 (drop A correlations between specifics at ages 5 and 10), and Model 5 (drop A correlations between specifics at ages 7 and 10) suggested that the three sets of genetic correlations were significantly different from zero. Tests of the three sets of unique environmental correlations indicated that the correlations between ages 5 and 10 (Model 7) and between ages 7 and 10 (Model 8) were insignificant and that the correlations between ages 5 and 7 were marginally significant (Model 6). In view of this marginal effect, we dropped the correlations between subtest-specific unique environmental residuals. This implies that unique environmental effects do not contribute to the stability in subtest performance between ages (Model 9; E correlations between specifics at ages 5, 7, and 10 are dropped). This model served as the new reference model for subsequent analyses. The second series of analyses evaluated the importance of latent group factors. It was decided not to evaluate the genetic effects because their significant contribution to the observed (co)variance was already established by the age-specific analyses. Evaluation of the shared environmental effects confirmed the earlier obtained age-specific result, that is, the significant contribution at ages 5 and 7 (Model 10, drop C at age 5; Model 11, drop C at age 7). Here, as opposed to the cross-sectional analyses, the shared environment explained a significant part of the (co)variance at age 10 (Model 12, drop C at age 10). Presumably this was due to the fact that the shared environmental common factor served both to explain the covariance between the subtest scores at age 10 and part of the covariance between ages 7 and 10. The third and final analyses were carried out to explore the varying magnitude of the genetic and shared environmental influences at the three occasions. The log-likelihood

Table III. Longitudinal Analyses

Model	−2 LL (*df*)	Comparison	χ^2 (*df*)
1. Cholesky decomposition ACE, no sex differences	39463.0 (6608)		
2. A: simplex structure with 3 verbal and 3 nonverbal factors, 18 correlated specifics C: simplex structure with 3 general factors E: 18 correlated specifics	39801.5 (7025)	Model 1	338.5 (417), *ns*
3. Drop A correlations specifics between ages 5–7	39835.6 (7031)	Model 2	34.1 (6), $p < .05$
4. Drop A correlations specifics between ages 5–10	39818.3 (7031)	Model 2	16.8 (6), $p < .05$
5. Drop A correlations specifics between ages 7–10	39843.6 (7031)	Model 2	42.1 (6), $p < .05$
6. Drop E correlations specifics between ages 5–7	39814.1 (7031)	Model 2	12.7 (6), $p = .05$
7. Drop E correlations specifics between ages 5–10	39807.2 (7031)	Model 2	5.7 (6), *ns*
8. Drop E correlations specifics between ages 7–10	39812.4 (7031)	Model 2	10.9 (6), *ns*
9. Drop E correlations specifics between every age-interval	39828.3 (7043)	Model 2	26.8 (18), *ns*
10. Drop C age 5	39860.5 (7032)	Model 9	32.2 (8), $p < .05$
11. Drop C age 7	39860.6 (7032)	Model 9	32.3 (8), $p < .05$
12. Drop C age 10	39850.7 (7032)	Model 9	22.4 (8), $p < .05$
13. A factor loadings age 5 = age 7	39846.9 (7049)	Model 9	18.7 (6), $p < .05$
14. A factor loadings age 7 = age 10	39847.8 (7049)	Model 9	19.6 (6), $p < .05$
15. C factor loadings age 5 = age 7	39843.9 (7049)	Model 9	15.6 (6), $p < .05$
16. C factor loadings age 7 = age 10	39847.0 (7049)	Model 9	18.7 (6), $p < .05$

Table IV. Percentages of Total Variance Explained by Additive Genetic and Environmental Factors

		% Variance accounted for by genetic and environmental effects with 95% confidence intervals								
Subtest	Age	A_{nv}	A_v	A_{sp}	h^2	95%	c^2	95%	e^2	95%
Exclusion	5	28		10	38	.23–.53	19	.09–.31	43	.32–.55
	7	37		3	40	.28–.53	5	.01–.13	55	.42–.66
	10	55		3	58	.46–.69	7	.01–.17	35	.26–.45
Discs	5	24	—	22	46	.35–.57	3	.00–.09	51	.41–.62
	7	30	—	28	58	.45–.68	5	.01–.16	37	.28–.47
	10	29	—	15	44	.34–.54	4	.00–.11	52	.42–.61
Hidden	5	8	—	40	48	.33–.60	11	.03–.23	41	.31–.54
Figures	7	29	—	8	37	.20–.50	14	.06–.29	49	.38–.62
	10	36	—	12	48	.34–.61	8	.02–.19	44	.32–.56
Verbal	5	—	11	10	21	.08–.37	39	.24–.53	40	.31–.49
Meaning	7	—	9	10	19	.06–.33	17	.06–.29	64	.53–.75
	10	—	25	17	42	.23–.60	32	.16–.48	26	.18–.36
Learning	5	—	49	4	53	.38–.66	17	.06–.31	30	.23–.39
Names	7	—	23	16	39	.23–.60	32	.12–.48	29	.22–.39
	10	—	58	18	74	.65–.83	4	.00–.13	22	.15–.29
Idea	5	—	8	50	58	.45–.68	5	.01–.14	37	.28–.42
Production	7	—	2	56	58	.43–.68	4	.00–.14	38	.29–.51
	10	—	11	49	60	.45–.71	4	.00–.12	36	.26–.50

Note: A_{nv} = nonverbal genetic factor, A_v = verbal genetic factor, A_{sp} = specific genetic factors. h^2 = proportion of total variance explained by genetic factors ($A_{nv} + A_v + A_{sp}$), c^2 = proportion of total variance explained by the shared environmental general factor, e^2 = proportion of total variance explained by unique environmental specific factors. $h^2 + c^2 + e^2 = 100\%$.

difference tests suggested that the equality constraints on the factor loadings were tenable neither in the genetic part of the model (Model 13, A factor loadings age 5 equal to age 7; Model 14, A factor loadings age 7 equal to age 10) nor in the shared environmental part of the model (Model 15, C factor loadings age 5 equal to age 7; Model 16, C factor loadings age 7 equal to age 10). Strictly speaking, changes in factor loading indicate qualitative changes in the traits that are measured by the RAKIT. However, the interpretation of these results is complicated by the fact that the item content of the RAKIT is tailored to each age group. The overlap in items is large but not complete. The observed results may therefore be due to a true developmental effect and/or changes in item content.

Based on the best-fitting longitudinal model (Model 9), we calculated the decomposition of phenotypic variance of each subtest at each occasion. The results are shown in Table IV.

The contribution of the shared environment is either small at all three occasions (Discs, Idea Production), or decreases with time (Exclusion, Hidden Figures, Learning Names). The subtest Verbal Meaning forms an exception in that the contribution is variable, but consistently large. Generally, the subtest variance explained by the common genetic factors increases over time. Judging by the explained variance in the subtest Idea Production, it appears that this subtest is not well represented by the verbal common factor. With respect to the genetic subtest specifics, the results relating to the nonverbal and verbal abilities differ. As opposed to the verbal subtests, the nonverbal subtests display a decrease in the variance of these genetic residuals. Unique environmental influences are large, accounting for around one third to one half of the total variance for each measurement at all ages.

The longitudinal structure of the common genetic and shared environmental factors were modeled using (first-order) autoregressions.

The regressions were used to calculated the correlations among these factors over time. The correlations and confidence intervals are shown in Table V. [5]

[5] In the earlier study (Rietveld *et al.,* 2000) the 95% confidence interval of the nonsignificant genetic correlation was reported incorrectly (−.26 to .10). The correct confidence interval is −.26 to +.43. The confidence intervals between the present and earlier study overlap greatly. As opposed to the age-specific study, the genetic correlation between A-verbal and A-nonverbal at age 5 differs significantly from zero in the present study.

Table V. Correlations for Genetic Common Factors (top) and Shared Environmental Common Factors (bottom). The boundaries of 95% confidence intervals are shown in parentheses.

	Genetic correlations					
	Age 5 Nonverbal	Age 5 Verbal	Age 7 Nonverbal	Age 7 Verbal	Age 10 Nonverbal	Age 10 Verbal
Age 5 Nonverbal	1.00					
Age 5 Verbal	.25 (.07–.41)	1.00				
Age 7 Nonverbal	.92 (.80–1.00)	.23 (.07–.39)	1.00			
Age 7 Verbal	.25 (.07–.41)	.99 (.88–1.00)	.28 (.09–.43)	1.00		
Age 10 Nonverbal	.87 (.73–1.00)	.22 (.06–.37)	.94 (.86–1.00)	.26 (.08–.41)	1.00	
Age 10 Verbal	.24 (.07–.40)	.94 (.79–1.00)	.26 (.08–.42)	.94 (.80–1.00)	.30 (.13–.45)	1.00

	Shared environmental correlations		
	Age 5	Age 7	Age 10
Age 5	1.00		
Age 7	.82 (.67–.92)	1.00	
Age 10	.79 (.60–.92)	.97 (.80–1.00)	1.00

Judging by the correlations between the genetic common factors over occasions, the stability of genetic individual differences on these factors is large (correlations between .87 and .99). The common shared environmental factor likewise contributes greatly to the stability of individual differences over time (correlations equal .82, .97, and .79).

Table VI lists the contributions of genetic and environmental effects to the between-occasion subtest correlations.

Most of the expected phenotypic correlations are estimated between about .40 and .60. The correlations between ages 7 and 10 are larger than those between ages 5 and 7 in four subtests (Exclusion, Discs, Hidden Figures, Idea Production). The decomposition of the correlations reveals that genetic effects are an important source of stability. This is due to both the relative magnitude of the genetic effects at each occasion and to the large genetic correlations over time. On average, genetic effects explain around 74% of the observed stability in subtest performance from age 5 to age 7 and over 80% from age 7 to age 10.

As already suggested by the results in Table IV, specific genetic factors contribute substantially to the expected correlations in the subtests Discs and Idea Production. With the exception of the subtest Verbal Meaning, the contribution of shared environment to the stability is relatively small. Thus, although the correlations between the shared environmental factors are large (Table V), the relatively minor and, over time, diminishing contributions of the shared environment at each occasion (Table IV) render the contribution to the phenotypic stability of subtest performance.

DISCUSSION

The aim of the present study is to investigate the differentiation hypothesis at the level of the genetic and environmental covariance structure. To this end, multivariate data were analyzed of 209 twin pairs, who were tested at ages 5, 7, and 10 years. As a point of departure of comparison of factor structures over time, we considered configural invariance (Horn and McArdle, 1992). Configural invariance implies that the same observed variables load on the same factors across measurement occasions. Configural invariance was established in phenotypic factor analyses of the data observed at each occasion. An oblique two-common-factor model fit the data

Table VI. Within-Trait, Across-Age Expected Correlations, Partitioned into Genetic and Environmental Contributions

	Age interval	E(r_p)	A group	A specifics	C
Exclusion	Age 5 to 7	.41	.30	.03	.08
	Age 7 to 10	.50	.43	.01	.06
Discs	Age 5 to 7	.47	.25	.19	.03
	Age 7 to 10	.53	.28	.20	.05
Hidden Figures	Age 5 to 7	.27	.14	.02	.11
	Age 7 to 10	.47	.31	.05	.11
Verbal Meaning	Age 5 to 7	.41	.10	.10	.21
	Age 7 to 10	.43	.14	.06	.23
Learning Names	Age 5 to 7	.61	.34	.08	.19
	Age 7 to 10	.58	.35	.13	.10
Idea Production	Age 5 to 7	.39	.04	.31	.04
	Age 7 to 10	.47	.04	.39	.04

Note: E(r_p) = expected phenotypic correlation based on final model, being the sum of $a_i * r_a * a_k$ (calculated separately for A group and A specifics), and $c_i * r_c * c_k$ (C). Parameters a and c represent the unstandardized path loadings at a specific age, i and k represent the initial and subsequent test occasion, r represents the correlation between factors. The unique environment does not contribute to stability of subtest performance.

relatively well. The common factors represented verbal (Verbal Meaning, Learning Names, Idea Production) and nonverbal cognitive abilities (Exclusion, Discs, Hidden Figures). Configural invariance was also found to be tenable in the genetic and environmental factor models, established in analyses of the cross-sectional data. The unique environment did not contribute to the covariance between the subtests. The genetic factor structure comprised two correlated, verbal and nonverbal common factors and six subtest-specific factors. A single common factor without residuals accounted for the covariance structure of the shared environmental effects. The comparison of the phenotypic results with the genetic and environmental results indicates that the phenotypic two-factor solution is due mainly to the genetic structure.

The analyses of the longitudinal dataset produced various interesting results. With respect to the structure and importance of the genetic and environmental effects, the results presented here are in agreement with those presented by other twin studies (Cardon and Fulker, 1993; McCartney *et al.,* 1990; McGue *et al.,* 1993; Patrick, 2000). When outcomes from two large infant studies (Price *et al.,* 2000; Reznick *et al.,* 1997) are combined with the outcomes obtained here, it emerges that between the age of 2 and the age of 5, and up to the age of 10, genes become increasingly important in explaining variation in verbal and nonverbal intellectual abilities. Genetic effects not only increase in importance as a source of individual differences at specific ages but also as a source of stability over ages.

Shared environmental effects are highly stable from infancy to middle and late childhood, but diminish in importance during this time period. At age 10, the relative influence of the familial environment is estimated at less than 10% of the total variance of five subtests. In our previous paper (Rietveld *et al.,* 2000) we reported a correlation of .46 between parents for highest attained education. Given the association with IQ, marital assortment for educational level may have contributed to the detected shared environmental effects. However, we do not believe that assortment can account for the presence of shared environmental effects in these data. First, on the assumption that assortment is phenotypic, the induced genetic correlation will be lower than .46. Second, the phenotypic correlation between educational attainment and general intelligence is .5 (median value; e.g., Kline, 1991). Taken together, these findings suggest that the genetic correlation for IQ tests induced by assortment for educational attainment will be low. In addition, we find that shared environmental effects decrease over time. This decrease is known to continue beyond 10 years. This is inconsistent with the possible consequences of phenotypic assortment at the genetic and environmental level.

The unique environmental influences are specific to each age and to each subtest. Although the unique environmental effects lack stability, they do remain the most important environmental influence in explaining individual differences in cognitive abilities. Specific factors account for one third to half of the total variance for each subtest at each age. Numerous studies of developmental intelligence have pointed at the importance of the environment that make children in the same family different from one another (for discussion see Plomin and Daniels, 1987; Plomin *et al.,* 1996; Turkheimer and Waldron, 2000). The large estimates for unique environmental effects at each age cannot be explained by the degree of unreliability of the administered RAKIT subtests (Bleichrodt *et al.,* 1984). From the reported internal consistencies that vary between about .70 and .90, it is suggested that a substantial part of the variance unique to the individual must result from influences different from test unreliability. The unique environmental effect may include an effect due to the interaction between genetic and environmental deviations (Boomsma and Martin, 2002; Plomin *et al.,* 1977). This interaction implies that variations in the environment affect individuals differently depending on their genotypes. If such an interaction is between genetic effects and unique environmental effects, the unique environmental effects are overestimated.

The essence of the differentiation hypothesis, as discussed in the Introduction, concerns the correlations among the subtest scores. The phenotypic correlations are expected to decrease during normal cognitive development. In investigating this hypothesis, we must consider genetic and environmental sources of individual differences to determine whether differentiation has taken place. First, as noted above, we find that the correlations between the common genetic factors at each occasion display little support for this hypothesis at the genetic level (.25, .28 and .30; see Table V). Second, we find that the shared environmental effects are either constant or decrease over time. The shared environmental common factor at each occasion is certainly a source of correlation among the subtest scores. Any decline in these effects over time may thus be viewed as a contribution to differentiation by decreasing the phenotypic correlations among subtests. Clear decrease in factor loadings is limited to the subtests Exclusion, Hidden Figures, and Learning Names. However, the shared environment in general explains relatively little of the variance, and thus the differentiation due to the decrease in shared environmental effects is thus weak at best. The results concerning the unique environmental effects are straightforward. These effects are specific to the subtests and they do not contribute to the stability of individual differences over time. An increase in the unique environmental effect over time will result in differentiation at the phenotypic level, by lowering the phenotypic intersubtest correlations. The results show that there is certainly no increase in the relative contributions to the variance. We emphasize that these results are limited to the age range from 5 to 10 years; we cannot extrapolate beyond the age of 10. However, within this age range, we find little support for the differentiation hypothesis at the genetic or the environmental level.

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