A Longitudinal Genetic Analysis of Low Verbal and Nonverbal Cognitive Abilities in Early Childhood

Thomas S. Price[1], Philip S. Dale[2], and Robert Plomin[3]

[1] *Wellcome Trust Centre for Human Genetics, University of Oxford, UK*

[2] *University of Missouri–Columbia, MO, USA*

[3] *Social, Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, London, UK*

By middle childhood, the same genetic factors are largely responsible for individual differences in verbal and nonverbal abilities, suggesting a genetic basis for general cognitive ability ("g"). Our previous work on verbal and nonverbal abilities throughout the normal range of variation during infancy and early childhood suggests that genetic influences show domain-specific as well as domain-general effects, implying that the switch to nearly complete domain-general effects occurs later in development. Much less is known about the genetic structure of low cognitive performance, although our previous work has shown that a composite measure of low "g" is highly heritable at 2, 3 and 4 years of age. We report the first multivariate, longitudinal analyses of low verbal and nonverbal cognitive abilities (defined as the lowest 10% of the distribution) at 2, 3 and 4 years of age using data from 9026 pairs of UK twins assessed by their parents as part of the Twins Early Development Study (TEDS). Domain-general genetic influences increased significantly from 2 to 3 to 4 years. Although the phenotypic polychoric correlation between low verbal and low nonverbal ability was similar at 2, 3 and 4 years (.36, .43, .35), the genetic contribution to the phenotypic correlation increased dramatically (.37, .47, .76), with a corresponding decrease in the comorbid influence of shared environment (.61, .44, .35). We conclude that for low ability, as well as for normal variation in ability, genetic "g" emerges during early childhood but is not fully developed until middle childhood.

Much is known about the genetic and environmental origins of general cognitive ability ("g", often called intelligence or IQ). Twin studies totalling more than 10,000 pairs of twins, together with family and adoption studies, indicate that "g" is about 50% heritable, that heritability increases from about 20% in infancy to about 60% in middle age, and that shared environmental influences decrease during adolescence (Plomin, 1999a). Surprisingly little is known, however, about the low end of the distribution of "g". The first large twin study of mild mental impairment, defined as the lowest 5% or 10% of the "g" distribution, focused on twins assessed at 2, 3 and 4 years of age and found significantly greater heritability for low "g" (.49 for both 5% and 10% cut-offs) than for variation throughout the distribution (.24) (Spinath et al., 2004). Further findings from the same study also suggest that for preschool children, heritability estimates for low verbal ability (ranging .38–.81) (Viding et al., 2004a; Dale et al., 1998a; Eley et al., 2001) and low nonverbal ability (ranging .40–.60) (Viding et al., in 2004b; Eley et al., 1999, 2001) may exceed those for individual differences in ability.

One of the most surprising findings from genetic research on cognitive abilities comes from multivariate genetic analyses that dissect the covariance between abilities into genetic and environmental sources of covariance. Specific cognitive abilities — which are organized hierarchically into verbal and nonverbal abilities — are all moderately heritable, although not as heritable as "g" (Plomin & DeFries, 1998). Multivariate genetic analyses indicate that the same genes are largely responsible for the heritability of these diverse cognitive abilities (Petrill, 1997). Multivariate genetic analysis yields a statistic called the *genetic correlation*, which is the extent to which genetic effects on one trait correlate with genetic effects on another trait independent of the heritability of the two traits. That is, although cognitive abilities are moderately heritable, the genetic correlations between them could be anywhere from 0.0, indicating complete genetic independence, to 1.0, indicating that the same genes influence diverse cognitive abilities. Multivariate genetic analyses have consistently found that genetic correlations among specific cognitive abilities are very high, about 0.80 on average (Petrill, 1997). This

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Address for correspondence: Thomas Price, Wellcome Trust Centre for Human Genetics, Roosevelt Drive, Oxford OX3 7BN, UK. Email: tprice@well.ox.ac.uk

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research suggests that, from a genetic perspective, specific cognitive abilities are not specific. These results have major implications for theories of cognitive neuroscience that assume that the brain works in a modular fashion (Plomin & Spinath, 2002).

Most of this multivariate genetic research involves adults (e.g., Tambs et al., 1986; Pedersen et al., 1994), but it appears that genetic overlap among cognitive abilities emerges in childhood. Existing studies have tended to show that the genetic overlap between individual differences in verbal and nonverbal ability is less in early childhood than in middle childhood (Cardon & Fulker, 1993, 1994; Casto et al., 1995; Luo et al., 1994; Rietveld, et al., 2002). Furthermore, analyses of the present sample of twins at 2, 3 and 4 years found that domain-general genetic influences on cognitive ability — "genetic g", that is, genetic influences on "g" — increase developmentally during this age span (Price, 2003). Together these findings suggest a hypothesis that genetic influences on cognitive abilities in the normal range are relatively domain-specific early in development and become more domain-general during middle childhood. This hypothesis has not, however, been tested for the genetic structure of low abilities.

The present study is the first to have a genetically informative longitudinal sample of sufficient size to provide such a test. We aimed to investigate the genetic and environmental structure of low verbal ability and low nonverbal ability at 2, 3 and 4 years, in the context of a longitudinal genetic analysis of change and continuity. The etiological relationships among these categories of low ability were explored with confirmatory factor analysis, using multivariate, longitudinal liability-threshold models, an approach that allows complex dependencies between dichotomous variables to be specified and tested. In this way, we tested a series of hypotheses about the nature and development of genetic and environmental influences on low verbal and nonverbal ability in early childhood.

Methods

Sample

The sampling frame for the Twins' Early Development Study (TEDS; Trouton et al., 2002) consists of all twins born in England and Wales between 1994 and 1996. From 25,815 live twin births identified by the government organization responsible for birth records, 16,810 families (65% of the complete population) were contacted, of whom 13,588 (81%) agreed to participate. Each received a background booklet asking for details about the family; further booklets containing cognitive test materials followed at the time of the twins' second, third and fourth birthdays. Despite attrition in the sample, TEDS families are reasonably representative of parents with young children in the UK population in terms of ethnicity, education, and employment status (Price, 2003). Parents correlated $r = .55$ for educational achievement in the complete sample.

Families in which either twin suffered a severe perinatal problem (298 families) or medical condition (260 families) or for whom zygosity could not be determined (335 families) were excluded. Zygosity was diagnosed by parental ratings of physical similarity validated by the identity of 8–10 polymorphic DNA markers, a method achieving over 95% accuracy (Price et al., 2000). Test scores were invalidated if they were completed more than 6 months after the target birthday. Verbal scores were invalidated if the family spoke a language other than English in the home. The final sample, consisting of the 9026 eligible families who returned the background booklet and at least one other booklet, included 1428 pairs of MZ males (MZM), 1649 pairs of MZ females (MZF), 1499 pairs of DZ males (DZM), 1,499 pairs of DZ females (DZF), and 2,951 opposite-sex dizygotic (DZO) pairs.

Measures

Expressive vocabulary. Parents completed anglicized, age-appropriate adaptations of the MacArthur Communicative Development Inventory (MCDI) (Fenson et al., 1994) by identifying, from a checklist, words that their children have said. Uncompleted checklists were scored as missing, and were distinguished from true zeroes on the basis of the parents' other reports of the children's verbal abilities. The 2-year vocabulary measure consisted of 100 words that predict the full MCDI list with very high accuracy, $r = .98$ (Fenson et al., 1997). The 3-year measure was also a 100-item checklist taken from an upward extension of the MCDI with similar form and design principles (Dale et al., 1998b). The 4-year measure was similarly constructed and comprised 48 items.

Expressive grammar. Anglicized abbreviations of the MCDI grammar scales were employed at 2 and 3 years, each containing 13 items in which parents choose from a pair of word combinations the phrase that best characterizes what their child can say. The scores were rescaled into a 3-point composite at age 2 and a 5-point composite at age 3 (Dionne et al., 2003). At age 4, more global ratings of sentence complexity were sought in view of the increased difficulty faced by parents in reporting on specific aspects of their children's more advanced grammar, to derive a 3-point composite (Price 2003).

Nonverbal cognitive ability. Cognitive performance was assessed using subtests of the Parent Report of Children's Abilities (PARCA; Oliver et al., 2002; Saudino et al., 1998; Price 2003), in which parents administer an hour-long battery of cognitive tests to their children. Children's scores on these measures are based entirely on nonverbal responses. Some of these materials were adapted from standard tests while other items were developed specifically for use in TEDS.

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A separate validation study of 107 two-year-olds found a correlation between the parent-administered PARCA and the Bayley Mental Development Index of r = .41 (Saudino et al., 1998). In a further study of 85 three-year olds, the parent-administered PARCA correlated r = .32 with the McCarthy General Cognitive Index and r = .38 with a nonverbal McCarthy composite (Oliver et al., 2002). In both studies, the prediction of standard measures of cognitive ability by the PARCA was significantly improved by adding the vocabulary composite. Validation of the 4-year measures against standard assessments is underway.

At each age, a verbal composite was calculated by averaging the standardized vocabulary and grammar scores. A nonverbal composite was calculated as the total score on the parent-administered nonverbal items divided by the total possible score on these items. For the 3- and 4-year PARCA tests, the quotient was scaled so that if one of the subtests was entirely missing, the total possible score was taken as the maximum possible on the remaining subtests. Binary variables representing the lowest 10% of scores were derived from the verbal (V) and nonverbal (N) composites at each age. These categories carry a suffix indicating the age of measurement (V_2 indicates the lowest 10% category on the 2-year verbal composite, and so on).

Analysis

The etiological relationships among these categories of low ability were explored by confirmatory factor analysis, using multivariate liability-threshold models (Neale & Cardon, 1992) implemented in Mx (Neale, 1997). Previous genetic analyses of low ability in this sample — including univariate analyses of low verbal ability, low nonverbal ability, and low "g" (Dale et al., 1998a; Eley et al., 1999; Viding et al., 2004a; Spinath et al., 2004), longitudinal analyses of low verbal and nonverbal ability (Eley et al., 2001; Bishop et al., 2003), and bivariate analyses of low verbal and nonverbal ability at 2 years (Purcell et al., 2002) and 4 years (Viding et al., in press-b) — have used DF extremes analysis (DeFries & Fulker, 1988; Purcell & Sham, 2003) to analyze the mean difference between low ability probands and quantitative ability scores in co-twins. In contrast, the liability-threshold model used in the present analyses defines low ability as a category, manifested when a continuously-distributed liability exceeds a certain threshold. The major advantage of the liability-threshold approach is that it facilitates multivariate statistical modeling, allowing complex hypotheses to be tested.

Latent structures modeled the influences of genetics, shared environment, and nonshared environment on the liability to low verbal and nonverbal ability. Common factors loaded on the low verbal and nonverbal ability measures at each age, with specific verbal and nonverbal latent factors accounting for domain-specific liability. For each common factor, the loadings on the verbal and nonverbal measures were constrained to be equal to ensure model identification. The common factor loadings represented shared influences on the liability to low verbal and nonverbal abilities, whereas the residual factor loadings represented domain-specific influences on the liability to low cognitive ability.

Longitudinal modeling enabled the testing of hypotheses about the developmental relationships among these factors. Although various time-series models are appropriate to describe the development of cognitive abilities at these ages (e.g., McArdle, 1986; Molenaar & Boomsma, 1987), the current analyses tested extensions of a genetic simplex model whose applicability to modeling continuity and change in cognitive abilities has been demonstrated (Eaves et al., 1986; Hewitt et al., 1988; Phillips & Fulker, 1989). The simplex model usefully interprets longitudinal data by providing direct estimates of age-to-age stability in latent variables (Anderson, 1960; Guttman, 1954).

Successive models of increasing generality were fit to tables of raw ordinal data using maximum likelihood estimation, until a significant decrease in fit was observed using the Likelihood-Ratio test (Neale & Cardon, 1992). Exhaustive model-fitting procedures were not followed due to the extremely complex and time-consuming nature of the analyses, which also precluded the calculation of confidence intervals on the parameter estimates.

Results

Table 1 shows the longitudinal concordances, cross-concordances and odds ratios for the low 10% nonverbal and verbal ability categories. Longitudinal concordances were calculated as the proportion of probands at a certain age whose co-twin was a proband on the *equivalent* measure at a later age. The longitudinal cross-concordances were calculated as the proportion of probands at a certain age whose co-twin was a proband on the *other* measure at a later age. Odds ratios were calculated using the logistic regression analyses implemented in SAS (SAS Institute Inc., 1996), entering the earlier measure as the predictor, with the co-twin's status on the later category as the dependent variable. The smallest cell frequency for any of the concordances or cross-concordances reported in Table 1 was 22, which was the number of MZ males concordant for low nonverbal ability at age 2 and low verbal ability at age 4.

For both male and female pairs, the MZ concordances exceeded the DZ concordances. The pattern of concordances was reflected in the results of the logistic regression analyses, with odds ratios for MZ pairs generally exceeding those for DZ pairs. These results suggest that genetic factors contributed to the continuity of low verbal and nonverbal cognitive abilities. The concordances and risk ratios tended to be higher for verbal than nonverbal measures, suggesting greater familial contributions to the stability

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Table 1

Longitudinal Concordances, Cross-concordances, and Odds Ratios for Low 10% Verbal and Nonverbal Ability Categories

	Age	Probandwise concordance (%)					Odds Ratio				
Measure	Span	MZM	DZM	MZF	DZF	DZO	MZM	DZM	MZF	DZF	DZO
Verbal	2→3	36.9	28.9	31.5	27.5	13.4	9.3**	5.3**	12.7**	13.9**	3.5**
	3→4	36.5	30.5	40.4	23.8	20.6	13.9**	9.3**	15.4**	4.2**	4.7**
	2→4	26.4	20.2	25.3	20.9	16.7	7.0**	4.8**	6.9**	6.0**	4.4**
Nonverbal	2→3	25.4	17.6	16.7	9.0	15.2	5.8**	2.5*	2.5*	2.3*	3.2**
	3→4	33.2	19.9	13.8	13.2	12.1	8.1**	4.9**	5.5**	6.0**	2.5*
	2→4	20.3	11.8	10.5	6.9	9.2	3.0**	2.5*	4.8**	2.5	2.9**
	Age	Probandwise cross-concordance (%)					Odds Ratio				
Measure	Span	MZM	DZM	MZF	DZF	DZO	MZM	DZM	MZF	DZF	DZO
Verbal→	2→3	21.4	18.0	14.8	15.4	14.1	3.6**	2.5*	3.9**	2.0	1.9*
Nonverbal	3→4	22.7	16.7	11.8	8.6	6.3	3.8**	2.8**	1.9	2.2	1.6
	2→4	16.6	15.4	13.0	5.5	8.5	1.3	1.2	2.6*	1.9	1.5
Nonverbal	2→3	26.3	16.2	16.7	6.9	8.1	4.3**	2.4*	3.0	6.3**	2.8**
→Verbal	3→4	24.2	15.2	14.3	15.5	12.1	4.9**	2.8**	3.7**	2.8	1.4
	2→4	12.7	9.8	14.8	8.3	7.9	2.7**	2.7*	4.7**	2.2	2.0*

Note: $^{*}p < .05$, $^{**}p < .001$.

of low verbal abilities than low nonverbal abilities. The cross-domain analyses also showed a tendency toward greater MZ than DZ concordances and risk ratios, suggesting that shared (domain-general) genetic factors contribute to the stability of low abilities. Familial concordances tended to be higher for males than females, presumably due to the greater prevalence of low abilities in males since the patterns of sex differences in the odds ratios were much less obvious.

To further explore these data, liability-threshold models were applied to the familial contingencies of the low ability categories. The most general model imposed a hierarchy of general (A_G) and specific (A_V, A_N) genetic factors at each age. To ensure an identified model, equality constraints were imposed on the general factor loadings within each time point. Longitudinal correlations were freely estimated between the general and specific genetic factors. The longitudinal relationships between general and specific factors of shared environment (C_G, C_V, C_N) were modeled by simplex paths, with residual pathways on the observed variables representing measure-specific shared environmental effects. In order to identify the model, these residual paths on the verbal measures were fixed to be equal at each time point, as were the residual paths on the nonverbal measures. Nonshared environmental influences were modeled by six latent factors which were allowed to vary without any restriction, a structure equivalent to a saturated cholesky model. Path diagrams representing these genetic and shared environmental structures for this model are shown in Figure 1. The different structures for genetic and shared environmental influences were dictated by the practicalities of estimating such a complex model: this baseline model was the most general longitudinal model that could be estimated that retained a hierarchical structure of general and specific genetic and shared environmental influences.

The fit statistics for the multivariate liability-threshold analyses are presented in Table 2. A fully sex-limited baseline model (1) allowed sex differences in all the parameters and allowed the coefficient of genetic relatedness between twins in opposite-sex pairs to vary between 0 and .5. Sex differences in the ACE parameter estimates were tested by comparing the fit of the baseline model with more parsimonious alternatives defined by fixing the genetic relatedness to .5 and equating all male and female parameters, other than the threshold parameters (2). Model (2) provided a better fit, indicating no sex differences in the genetic and environmental influences. A nested submodel of (2) constraining the nonshared environment structure to that provided by a single common factor and six residual factors did not fit significantly worse (3), and it did provide superior fit to a model allowing only age- and domain-specific nonshared environmental influences (4). Model (3) was constrained such that the longitudinal relationships among the general and specific genetic factors fit a simplex structure, without significant loss of fit (5). The domain-general shared environmental influences could be modeled by a single common factor without significant reduction in fit (6). Further model-fitting aimed to test the statistical significance of the domain-specific genetic and shared environmental sources of continuity. Constraining the simplex parameters between the nonverbal genetic factors to zero, so that only the general factors contributed to genetic continuity among measures of low nonverbal ability, did not result in a significant loss of fit (7). The domain-specific genetic influences on the continuity in low verbal ability, however, could not be dropped from the model without significant deterioration in

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Table 2

Model Fit Statistics

Model	Description	*−2LL*	*df*	*Δ-2LL*	*Δdf*	*p*	Comparison
(1)	Sex-limited baseline model	36238.1	68857				
(2)	(1) allowing sex differences in thresholds only	36249.1	68913	11.0	56	1.00	(1)
(3)	(2) constraining NE to a single common factor plus specific factors	36253.5	68922	4.4	9	0.88	(2)
(4)	(3) constraining NE to uncorrelated residual factors	36277.7	68928	24.2	6	0.00*	(3)
(5)	(3) constraining continuity in general and specific G to simplex structure	36253.6	68925	0.1	3	1.00	(3)
(6)	(5) constraining domain-general SE to a single common factor	36253.7	68927	0.1	2	0.96	(5)
(7)	(6) constraining nonverbal-specific G to age-specific factors	36254.0	68929	0.3	2	0.85	(6)
(8)	(7) constraining verbal-specific G to age-specific factors	36273.5	68931	19.5	2	0.00*	(7)
(9)	(7) constraining nonverbal-specific SE to age-specific factors	36288.9	68932	32.9	3	0.00*	(7)
(10)	(7) constraining verbal-specific SE to age-specific factors	36300.6	68932	44.6	3	0.00*	(7)
(11)	(7) constraining domain-general G to a single common factor	36267.9	68931	12.0	2	0.00*	(7)

Note: G = genetics, NE = nonshared environment, SE = shared environment. *$p < .05$.

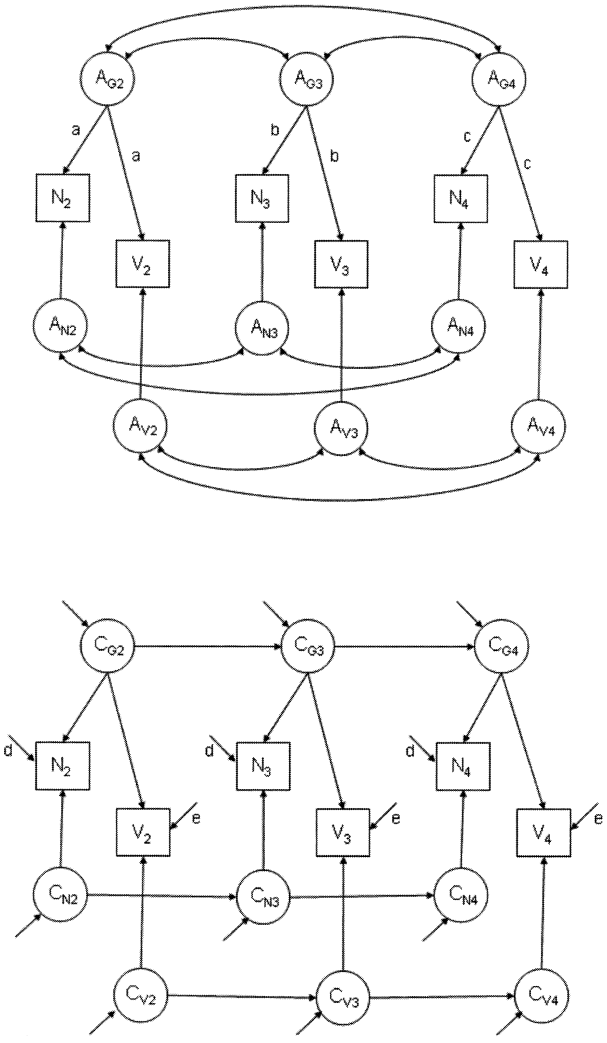

Figure 1

Path diagram of the liability-threshold model, showing the structures of genetic influences (upper diagram) and shared environmental influences (lower diagram) on low abilities. Equated paths marked by a lower case letter.

Table 3

Polychoric Correlations of the Low Ability Measures Estimated from the Best-Fitting Model

	N_2	V_2	N_3	V_3	N_4
V_2	.36				
N_3	.41	.35			
V_3	.35	.65	.43		
N_4	.31	.28	.47	.34	
V_4	.29	.55	.37	.73	.35

fit (8). The domain-specific shared environmental influences on continuity were statistically significant for both low nonverbal (9) and verbal (10) ability. Finally, dropping the innovations on the general factors of the genetic structure, such that a single general genetic factor influenced measures at all three ages, also resulted in a significantly worse fit (11). The best overall fit was provided by model (7), for which the results are reported as follows.

The expected polychoric correlations among the low ability categories are shown in Table 3. Contemporaneous verbal and nonverbal ability categories correlate to a similar extent at ages 2, 3, and 4 (r = .35–43), suggesting no strong developmental trends in the overlap (comorbidity) between low verbal and nonverbal abilities. The expected correlations are higher among the low verbal ability measures (r = .55–.73) than among the low nonverbal ability measures (r = .31–.47), suggesting greater continuity of low verbal ability than low nonverbal ability.

Table 4 shows the expected proportions of variance on the liabilities for low ability. The liabilities for all the low ability categories are moderately heritable with strong shared environmental influences, with estimates of heritability (h^2) ranging .21–.50 and estimates of shared environment (c^2) ranging .27–.67. The average h^2 for verbal and nonverbal ability

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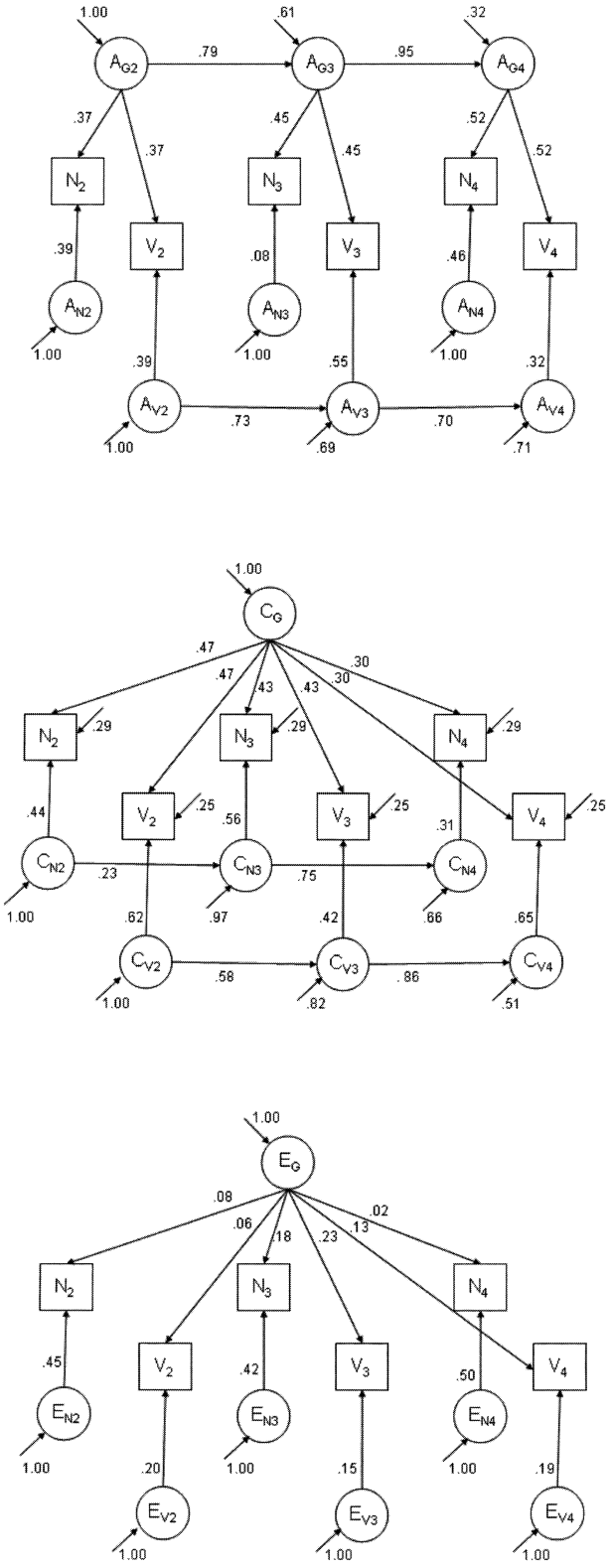

Figure 2

Path diagram of the best-fitting liability-threshold model, showing the structures and parameter estimates for the genetic (top diagram), shared environmental (middle diagram), and nonshared environmental (bottom diagram) influences on low abilities.

Table 4

Proportions of Variance in Liability to Low Ability

	h^2	c^2	e^2
N_2	.29	.50	.21
V_2	.28	.67	.04
N_3	.21	.59	.21
V_3	.50	.43	.08
N_4	.48	.27	.25
V_4	.37	.58	.05

increased across the three years (.29, .36, .43) and c^2 showed a corresponding decrease (.59, .51, .43).

Figure 2 shows the standardized parameter estimates for the best-fitting model. The orthogonal structures of genetic, shared environmental, and nonshared environmental influences are presented in separate diagrams.

The horizontal lines on Figure 2 denote simplex paths, modeling the continuity in latent factors from age to age. These indicated very strong genetic continuity at the domain-general level, with simplex coefficients of .79 between age 2 and age 3, and .95 between age 3 and age 4. The genetic influences specific to verbal ability also showed substantial stability from age to age: .73 between age 2 and age 3, and .70 between age 3 and age 4. In contrast, the model-fitting analyses showed that continuity in the genetic influences specific to low nonverbal ability were nonsignificant; that is, all of the stable genetic influence on nonverbal ability was absorbed by the domain-general factor.

Both general and specific genetic influences contributed to liability for low verbal ability at each age. Particularly noteworthy was the pattern of increasing domain-general genetic influences evident from the developmental increases in the loadings on the general genetic factors, from .37 at age 2 to .45 at age 3 and .52 at age 4.

For shared environment, both general and specific influences contributed to liability for both verbal and nonverbal ability. In contrast to increasing domain-general genetic influence from 2 to 4 years, domain-general shared environmental influence decreased from a factor loading of .47 at 2 years to loadings of .43 at 3 years and .30 at 4 years. Domain-general shared environmental influence was completely stable between the ages of 2 and 4, as indicated by the single general factor of shared environment. There was also substantial continuity between the domain-specific factors of shared environmental influence for both verbal and nonverbal abilities. The loadings on the single general factor of nonshared environment were small, indicating that nonshared environmental influences were mainly age- and domain-specific.

Table 5 presents the estimates of genetic and environmental correlations among the liabilities for low

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ability. The genetic correlations between contemporaneous low verbal and nonverbal abilities increased from r_G = .42 at age 2 to r_G = .62 at age 3 and r_G = .63 at age 4, giving some indication of a developmental trend toward increasing genetic correlations. The corresponding shared environmental correlations were .40, .44 and .29. Nonshared environmental influences were largely uncorrelated.

Table 5 also shows the proportions of the expected polychoric correlations mediated by genetics, shared environment, and nonshared environment. There was a notable developmental trend toward increasing genetic mediation and decreasing shared environmental mediation of polychoric correlations. The proportion of the polychoric correlation between contemporaneous low verbal and nonverbal ability that was mediated by genetics increased substantially, from .37 at age 2, to .47 at age 3, and .76 at age 4. There were corresponding falls with increasing age in the proportions of the polychoric correlation mediated by shared environment (.61, .44, .25).

Genetic correlations (r_G) among the verbal measures were very substantial, ranging .64–.80, indicating that the genetic influences on low verbal ability contributed mostly to stability. Genetic continuity within the nonverbal domain was somewhat weaker (r_G ranged .34–.69). Shared environment correlations within both the verbal and nonverbal domains were substantial (r_C ranged .51–.85), suggesting that the family environment contributed mainly to stability rather than change in the liability to low cognitive abilities. In relation to the proportions of the expected polychoric correlations explained by genetic and environmental factors, continuity in low nonverbal ability (i.e., the polychoric correlations among N_2, N_3 and N_4) was mediated mostly by shared environment (estimates were in the range .54–.64) with moderate genetic influences on continuity (estimates ranged .32–.47). Similarly, continuity in low verbal ability was mediated more by shared environment (.49–.61) than by genetics (.37–.59).

Further model-fitting analyses placed constraints on the model in order to test these developmental trends. Constraining the genetic correlations to be equal at each age showed that the increase in genetic correlation between ages 2 and 3 was not statistically significant (Δ-$2LL$ = 0.402, Δdf = 2, p = .82). However, the domain-general genetic influence at each age — genetic *g*, equivalent to the squared factor loading from the general genetic factor — did increase developmentally (see Figure 3). The increases in domain-general genetic influence from age to age were well-modeled by a linear trend (Δ-$2LL$ = 0.155, Δdf = 1, p = .69), the slope of which was significantly greater than zero (Δ-$2LL$ = 10.892, Δdf = 1, p = .001). The increasing domain-general genetic influences contributed to a significant increase in the genetic mediation of the polychoric correlation (bivariate heritability) between ages 2 and 4 (Δ-$2LL$ = 10.441, Δdf = 2, p = .005).

Table 5

Genetic and Environmental Correlations (Below Diagonal) and Proportions of Expected Polychoric Correlation (Above Diagonal) Expected from the Liability-threshold Model

Genetics

	N_2	V_2	N_3	V_3	N_4	V_4
N_2		.37	.32	.37	.47	.49
V_2	.42		.38	.44	.51	.37
N_3	.47	.54		.47	.46	.59
V_3	.30	.75	.62		.64	.47
N_4	.34	.39	.69	.45		.76
V_4	.39	.64	.79	.80	.63	

Shared environment

	N_2	V_2	N_3	V_3	N_4	V_4
N_2		.61	.64	.58	.54	.48
V_2	.40		.59	.54	.50	.61
N_3	.51	.37		.44	.55	.35
V_3	.47	.75	.44		.38	.49
N_4	.53	.42	.85	.50		.25
V_4	.27	.61	.25	.84	.29	

Nonshared environment

	N_2	V_2	N_3	V_3	N_4	V_4
N_2		.01	.04	.05	-.01	.04
V_2	.05		.03	.02	.00	.01
N_3	.07	.11		.10	-.01	.06
V_3	.15	.23	.33		-.01	.04
N_4	-.01	-.01	-.02	-.04		-.01
V_4	.10	.16	.22	.47	-.02	

Discussion

This first multivariate longitudinal analysis of low verbal and nonverbal abilities indicates that genetic "g" emerges during early childhood. That is, domain-general genetic influences increase significantly from 2 to 3 to 4 years. The genetic correlation between low verbal and low nonverbal ability increased from .42 at 2 years to .63 at 4 years. Although the phenotypic relationship between low verbal and low nonverbal ability does not increase during early childhood, the genetic contribution to the phenotypic correlation increases dramatically from .37 at 2 years to .47 at 3 years and to .76 at 4 years. Domain-general shared environmental influences show a corresponding decrease from .61 at 2 years to .44 at 3 years and to .35 at 4 years.

A very comparable pattern of results has been reported from a multivariate analysis of individual differences throughout the distribution in verbal and nonverbal abilities (Price, 2003). A developmental trend toward increasing domain-general genetic influences on verbal and nonverbal abilities mirrors the current finding of a statistically significant developmental trend toward increasing domain-general genetic influences on

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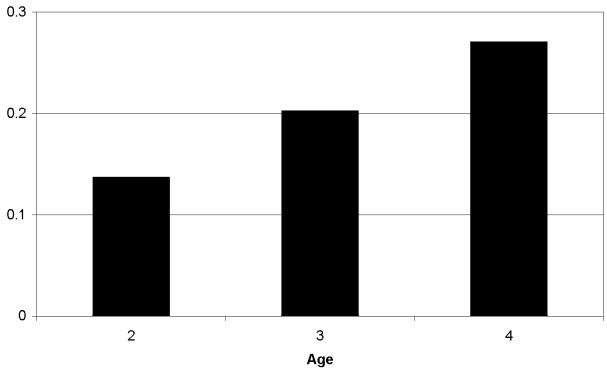

Figure 3

Genetic "g" at ages 2, 3, and 4, estimated from the best-fitting model (7).

low cognitive abilities. The complexity of the current analyses did not permit confidence intervals to be calculated around the parameter estimates, limiting the extent to which conclusions can be drawn from this comparison. Even so, the similarities between these sets of results are consistent with the hypothesis that liability for the low extremes of verbal and nonverbal ability in early childhood are influenced by the same genetic and environmental factors that operate over the whole range of abilities (Plomin, 1999b). This hypothesis suggests a continuum of cognitive ability rather than an etiologically distinct category of disability. The implication for clinicians is that not only children meeting clinical diagnostic criteria, but also children with subthreshold cognitive difficulties, may be disadvantaged.

These multivariate conclusions about the genetic links between low verbal and nonverbal ability at 2, 3 and 4 years of age are drawn from a simultaneous longitudinal analysis. The phenotypic stability from year to year is modest, and the majority of this stability can be attributed to shared environmental influences, which were generally stable. Nevertheless, the extent of *genetic* stability is surprising. For low "g" — as measured by the domain-general latent variable — genetic stability from 2 to 3 years was .79 and from 3 to 4 years was .95. Low verbal ability showed domain-specific as well as domain-general genetic influence, and genetic influence on the domain-specific verbal ability was also highly stable from 2 to 3 years (.73) and from 3 to 4 years (.70). These findings suggest that in spite of the transient nature of many early cognitive delays, children with low verbal abilities in early life are at considerable additional risk of continuing language difficulties if there is a family history of language problems, whether or not these problems are associated with low cognitive ability generally. A previous analysis of this dataset has arrived at a similar conclusion using a very different methodology (Bishop et al., 2003). In contrast, all of the genetic continuity in low nonverbal ability was absorbed by the stable genetic "g".

The design of this study necessitated definitions of low ability that may map imperfectly onto clinical conceptions of impairment. For example, we employed arbitrary cut-offs based on the distribution of scores for same-aged peers, although this approach is consistent with previous research on cognitive, language and reading problems. Also, our measure of verbal ability incorporated both expressive grammar and lexical ability, on the basis that children suffering from developmental language problems present with heterogeneous impairments. This approach has empirical justification in studies of normal language acquisition, which consistently show strong correlations between early lexical and grammatical development (Dale et al., 2000; Fenson et al., 1994). Twin studies show little evidence for genetic influences specific to these subcomponents of language (Dale et al., 2000; Dionne et al., 2003; Hohnen & Stevenson, 1999; Plomin & Dale, 2000). Nevertheless, we acknowledge that other measures of linguistic and nonverbal development, such as tests of receptive rather than expressive language or measures administered by testers rather than parents, might yield different results.

The main features of the results reported here suggest themselves from the patterns of concordance and cross-concordance shown in Table 1. We aimed to go beyond these simple summaries of the data by employing statistical model-fitting. Multivariate, longitudinal, liability-threshold analyses provide a sophisticated framework for hypothesis-testing, but rely for their power on a number of modeling assumptions. The standard assumptions of the classical twin model are made, as is the assumption that a normally-distributed continuum of liability underlies each category of low ability — one that has generally held up to scrutiny in research on the etiology of developmental disorders (van den Oord et al., 2003). The maximum likelihood estimates generated by these multivariate models may not reach their asymptotic values if fit to tables of multivariate ordinal data in which many of the cell frequencies are small or zero.

Future work using the TEDS sample will focus on the cognitive abilities and disabilities and educational achievements of these children as they are followed up into middle childhood at 7 and 9 years of age. Findings from twin and adoption studies of specific cognitive abilities in the normal range suggest that genetic correlations do not reach a peak until middle childhood, but that heritabilities increase throughout childhood and adolescence: as a consequence, genetic "g" continues to rise developmentally (for a review, see Price, 2003). We hypothesise that similar patterns will become evident for low abilities, and predict that genetic "g" will be stronger in middle childhood than we find in early childhood, both for abilities and disabilities. We also predict that high heritabilities and genetic correlations will extend beyond psychometric measures of ability to scholastic skills such as reading and mathematics. Ultimately, molecular genetics will

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provide a definitive test of these hypotheses. That is, we predict that genes associated with cognitive disabilities will also be associated with cognitive abilities, that genes associated with verbal abilities will also be associated with nonverbal abilities — more so in middle childhood than in early childhood — and that genes associated with "g" will also be associated with reading and mathematics abilities and disabilities.

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