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Academic Achievement and IQ: A Longitudinal Genetic Analysis

by

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B.S., University of Colorado, Boulder, 1992

M.A., University of Colorado, Boulder, 1997

A thesis submitted to the

Faculty of the Graduate School of the

University of Colorado in partial fulfillment

of the requirement for the degree of

Doctor of Philosophy

Department of Psychology

1999

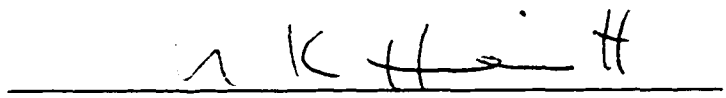
This thesis entitled:

Academic Achievement and IQ: A Longitudinal Genetic Analysis

written by Marcie Chambers

has been approved for the Department of Psychology

Gregory Carey - Committee Chair



John Hewitt

The final copy of this thesis has been examined by the signators, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

Chambers, Marcie L. (Ph.D., Psychology)

IQ and Academic Achievement in Twins: A Longitudinal Study

Thesis directed by Associate Professor Gregory Carey

A Cholesky decomposition model was used to analyze IQ and academic achievement in twin pairs. IQ was measured by the Bayley Scales of Infant Development (Bayley, 1969) at ages 14 months (N=398 pairs), 20 months (N=323 pairs) and 24 months (N=371 pairs), Stanford-Binet Intelligence Scale (Terman & Merrill, 1973) at ages 3 (N=346 pairs)and 4 (N=359 pairs), and the Wechsler Intelligence Scale for Children - Revised (Wechsler, 1974) at age 7 (N=290 pairs). Academic achievement was assessed by the Child Behavior checklist (Achenbach & Edelbrock, 1986) at ages 7 (N=203 pairs) and 9 (N=105 pairs). An analysis was carried out to estimate the additive genetic, common environmental, and individual environmental effects as well as to examine the associations between the variables. The results from the most parsimonious model indicate that there is an additive genetic component present at 14 months that affects IQ up to age 7. A second genetic component is present at age 3 and affects IQ and academic achievement through age 9. A third genetic component comes in at age

7 affecting IQ and academic achievement through age 9. Heritability estimates range from 38% to 61%. The common environment accounts for 17% to 50% of the variance. Genetic correlations between IQ and academic achievement range from 70% to 89% at age 7.

Phenotypic causality models were examined and were found not to fit the data.

I dedicate this dissertation
and my unconditional love
to my children
Preston and Brittney.

They were my
motivation and inspiration
in attaining this goal.

ACKNOWLEDGEMENTS

I would like to thank my advisor, Professor David W. Fulker, without whom I may not be writing this. He encouraged me to apply to this graduate program, set goals for me in the beginning that taught me this journey wasn't going to be an easy one, and was there when I needed him. I am ever so grateful to him for giving me a chance, believing in me, and being my friend. His untimely death has saddened me deeply.

I am very grateful to Professor Greg Carey who was gracious enough to step in as my advisor after David passed away. I couldn't have asked for a more kind, generous, giving person to work with than Greg.

I would like to thank Professor John Hewitt for his help during David's illness and for his great stress relieving parties. I would also like to thank Professor Jim Wilson and Dr. Toni Smolen for their help and kindness and Dr. Andy Smolen for freely giving his time and energy to teach me DNA analysis as well as for being supportive, helpful and my friend.

Thanks also goes to the professor who informed me that I wasn't cut out for this program and would never make it. It made me more determined then ever.

I am greatful for the many wonderful friends I have made here who have made this journey one that, in a way, I am sad to see end. A special thank you to Susan Young

and Chayna Davis.

On a personal level, I would like to thank my husband Art Ruiz for his love and support. I would like to thank my children Preston and Brittney who have had to deal with having a mother that was also a student for the past 11 years. During the stressful times, when there weren't enough hours in the day, they have learned how to prepare dinner for themselves, do their own laundry, and grow up a little faster than some children. They were my motivation to make a better life for us, but they had to make sacrifices and have done so without too much complaining. For that, I am eternally grateful. I have been truly blessed with two wonderful children.

I would like to thank my mother Lorma Marcotte who taught me perseverance and determination. Her, along with my sister Brenda Noble, brother Robert Schmitz, and sister-in-law Betha Schmitz have been very supportive.

Finally, a special thank you to my best friend Karen Mayne, who listened to me for hours on end, gave me good advise and believed in me. I will treasure her friendship always.

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Chapter I

Introduction and Literature Review

Introduction

The amount of variation in intelligence that can be accounted for by genetic affects has been of interest for many years. Bouchard and McGue (1981) published a comprehensive review of the literature on IQ correlations between relatives. In the 111 studies that met explicit selection criteria, they found that in general, as the number of genes the family members had in common increased, the average correlation between IQ increased as well. They stated that it is indisputable that IQ is partially determined by genes. The strength of the affect appears to increase from infancy (h^2=.09) to middle childhood (h^2=.36), and continues to increase through out the lifespan (Fulker, DeFries, & Plomin, 1988; Plomin & Petrill, 1997).

The etiology of academic achievement has also been examined. Loehlin and Nichols (1976) examined data from over 2,100 twin pairs in one of the best known studies of achievement. They examined data in twins who were administered the National Merit Scholarship Qualifying Test. They found that genetic components accounted for about 40% of the variation across different subjects.

These heritabilities were somewhat smaller than reported by Martin (1975) and Gill et al. (1985). The genetic correlations among the tests were substantial.

Most of the previous behavioral genetic studies on academic achievement and IQ have used standardized tests as a measure of academic achievement. This has been the case for twin studies (Martin, 1975; Gill et al., 1985; Thompson et al., 1991; Brooks, DeFries, & Fulker, 1990; Gillis, DeFries, & Fulker, 1992) as well as adoption studies (Wadsworth, 1994, Wadsworth et al., 1995; Cardon et al., 1990).

Genetic research on intelligence is now beyond the nature vs nurture question and is ready to make more important discoveries about intelligence (Plomin & Petrill, 1997).

Here we first review the results of previous behavioral genetic studies of academic achievement and its relationship to IQ, and then describe a current twin study of IQ and teachers' ratings of academic achievement.

The Intermediate Examination of the Public Examinations Board in South Australia (Martin, 1975) was used to assess the inheritance of scholastic abilities in 149 twin pairs. The subjects in this sample were 15 year olds who were generally expected to have spent three years at secondary school preparing for examination in

about seven subjects. Heritability estimates ranged from .47 (history) to .89 (chemistry). It was concluded that educational achievements, at least at the level of large scale impersonal testing programs, have the same heritabilities as IQ.

The Tertiary Admission Examination has also been used as a measure of scholastic achievement. In this study the Australian Scholastic Aptitude Test measured IQ in 264 pairs twins (Gill et al., 1985). The exams were administered during the students' final year in high school. They found that heritability accounted for 60 to 70% of the variation in both measures and shared environment accounted for around 20%. They concluded that a distinction between IQ tests and tests of scholastic achievement was not justified.

Scholastic achievement was examined in 6 to 12 year old twin pairs (n=278 pairs) using the Metropolitan Achievement Test (MAT) (Prescott, Barlow, Hogan, & Farr, 1986, Thompson et al., 1991). The extent to which phenotypic relationships among specific cognitive abilities and scholastic achievement were mediated by genetic factors was explored. Cognitive ability was assessed by eight subtests selected from a battery developed in the Colorado Adoption Project (CAP) (DeFries & Plomin, 1985). They found that heritability for cognitive measures ranged from .45 (memory) to .76

(spatial) and that the common environment had no influence. For achievement, heritability ranged from .19 (math) to .29 (reading). Here the common environmental effect ranged from .62 to .71. They also found that the phenotypic correlations between achievement and ability were almost entirely due to genetic mediation.

Again using a twin sample (N=146 pairs), Brooks, Fulker & DeFries (1990) tested the hypothesis that the phenotypic covariance among three reading measures was due to IQ. They also examined the genetic and environmental etiologies of the phenotypic relationships. The three reading measures used were subtests of the Peabody Individual Achievement Test (PIAT) (Dunn & Markwardt, 1970) and consisted of Reading Recognition (REC), Reading Comprehension (COMP) and Spelling (SPELL). The Wechsler Intelligence Scale for Children - Revised (WISC-R) and the Wechsler Adult Intelligence Scale-Revised (WAIS-R) (Wechsler, 1974) were used to measure general cognitive ability. The correlations obtained between IQ and the three reading variables were .38 (REC), .47 (COMP), and .13 (SPELL). They found that genetic factors account for most of the observed covariation between IQ and reading achievement.

The covariation between performance in reading and mathematics in 264 reading disabled twin pairs and 182 control twin pairs was analyzed (Gillis, DeFries and

Fulker, 1992). Reading was assessed by three subtests of the PIAT. Math was assessed by the PIAT Mathematics subtest, the Arithmetic subtest of the WISC-R, and the Spatial Relations subtest of the Primary Mental Abilities Test (Thurstone, 1962). They reported that the heritability for reading was .78 for the reading disabled children and .74 for the control group. The respective math heritabilities were .51 and .60. Common environment contributed significantly to the math scores (.44 and .37) but for reading was only .04 and .09 (reading disabled and control group respectively). Individual differences in both measures appeared to be caused by many of the same genetic influences.

Along with twin studies, adoption studies have also been used to estimate heritability and examine the covariation between IQ and academic achievement.

Wadsworth (1994) assessed the etiology of academic achievement and general cognitive ability of children at age 7 in the Colorado Adoption Project (CAP). The analysis included 199 adopted and 216 nonadopted children. Academic achievement was assessed by scores on the Reading Recognition (REC) subtest of the Peabody Individual Achievement Test (PIAT) and a composite mathematics measure (MATH) from the Numeration, Addition, and Subtraction subtests of the Key Math Diagnostic Arithmetic Test (Connolly, Nachtman, & Pritchett, 1976).

General cognitive ability was assessed by the Wechsler Intelligence Scale for Children - Revised (WISC-R) Verbal and Performance IQ tests. A substantial amount of heritability was found for Verbal IQ (.40), Performance IQ (.89), and Reading (.36), but not for MATH (.12). Shared environment accounted for between 24% of the variance (MATH) to 4% (Performance IQ).

Among the same CAP sample, Wadsworth et al. (1995) examined 190 pairs of siblings at age 7. The same variables as before were used except Perceptual Organization (PERCEP) replaced Performance (both from the WISC-R) as a measure of IQ. A phenotypic analysis showed that approximately 40% of the observed correlation between the achievement variables was due to influences shared with cognitive ability. The genetic analysis indicated that heritabilities were 21% for MATH, 26% for VERB, 30% for REC, and 60% for PERCEP. Shared environmental influences were negligible for the ability measures, and accounted for no more than 20% of the variance in the achievement measure.

Cardon et al. (1990) investigated the genetic and environmental etiologies of IQ and school achievement in 119 adoptive and 120 nonadoptive families participating in CAP. The children's IQ was measured with the WISC-R (Wechsler, 1974) and the PIAT Reading Recognition subtest (Dunn & Markwardt, 1970) was used to measure school

achievement. The children were tested after completion of their first year in elementary school. They found the phenotypic correlation between IQ and Reading to be .45. However, the genetic correlation between the two measures was unity and the environmental correlation was .04. Heritability estimates for Reading and IQ were .36 and .38, respectively, suggesting that school achievement measures are just as heritable as IQ.

Many of these studies indicate that most of the observed correlation between IQ and academic achievement can be accounted for by genetic factors. The twin studies showed that heritability accounted for most of the variation in both measures. In the adoption studies, where more specific abilities were analyzed, heritability was more diverse. The influence of the common environment was variable from study to study, but on average explained less of the correlation between IQ and academic achievement than did genetics.

While previous studies have used standardized tests to assess academic achievement, performance on these tests does not necessarily translate into school performance. The purpose of the present research is to examine the relationship between IQ and academic achievement as measured by teachers ratings instead of standardized tests. It is hypothesized that the variance in IQ and academic achievement as assessed by teacher

report will not be accounted for equally by genes and the environment as IQ is a more global measure than classroom performance. It is known that there are children who are "bright" and score high on IQ tests, but who did not apply themselves in school for one reason or another. While it can be justifiably argued that a high IQ would lead to superior academic achievement, it is not necessarily so and the factors influencing the two may be somewhat different.

An alternative index of achievement that may be related more closely to performance in the classroom, is provided by teachers' ratings of achievement. Using this measure, teachers rate the children in math and reading by comparing them to the other students in the classroom. This measure is much more similar to the grades the students earn in school as grades are also based on comparing children to the other children in the class room (C's being average, B's above average, etc.). IQ measures and traditional measures of academic achievement are both standardized tests. At times academic achievement has been assessed by subsets of the WISC-R, which is traditionally thought of as an IQ measure. When a student applies to college, two of the primary considerations admissions boards examine are results of standardized testing (e.g. SAT, GRE) and grade point average. While standardized tests are important and the

genetic influence on these tests have been examined, it is also important to analyze a measure of how well students preform in the classroom compared to their cohorts. A student with a high grade point average has other benefits such as discounts on auto insurance and obtaining a work permit. Also, many colleges require athletes to maintain a certain grade point average in order to participate in sports. Hence, determining what accounts for the variation grades is an important issue.

Chapter II

IQ and Academic Achievement at Age 7

Method

Subjects

The subjects in this study are participants in the Longitudinal Twin Study (LTS), an ongoing study at the Institute for Behavioral Genetics, University of Colorado at Boulder designed to obtain information on child development. Ascertainment began with twins born in the state of Colorado from 1984 to 1990. The parents of the twins were contacted through the Colorado Department of Health which sent out letters describing the study and requesting participation. To be eligible the children had to weigh at least 1,750 grams at birth and the gestation period had to be at least 34 weeks. There are currently 482 families participating in LTS. All of the subjects are tested at least once a year on numerous measures. The mean ages of the mothers and fathers at the time the twins were born were respectively 30 (s.d. 4.5) and 32 (s.d. 5.2). The mean educational level of the parents is 14.4 years with a range from 11 to 18 years.

The parents of the twins were assigned NORC occupational ratings of prestige (Hauser & Featherman,

1977). The NORC used the 1970 census classification of occupations to rank occupational titles according to prestige. For the U.S. white labor force the mean prestige rating is 41.7 (SD 13.9). For this sample, the mean NORC rating for the mothers is 38.7 (SD 16.5) and for the fathers is 48.2 (SD 13.5).

This sample is somewhat better educated and slightly older than average parents in this area (Plomin et al, 1990).

For the present analysis WISC-R scores were examined for 162 MZ twin pairs and 138 DZ twin pairs. Data on reading achievement were available on 117 MZ twin pairs and 96 DZ twin pairs; for math achievement, data were available on 117 MZ twin paris and 94 DZ twin pairs. Grades are dependant on teacher report, hence the smaller sample of grades compared to WISC-R scores. Sample sizes along with descriptive statistics are given in Table 1.

Measures

The twins were administered a full scale Wechsler Intelligence Scale for Children-Revised (WISC-R) (Wechsler, 1974) during the summer after they completed the first grade to assess IQ. The average reliability coefficient for this test is .96. Academic achievement was determined by teacher report. The teachers completed the Child Behavior Checklist (Achenbach & Edelbrock,

1986) in which they were asked to rate the child's performance in reading and math on five point scale ranging from far below grade level (1) to far above grade level (5).

It is common for the twins to be assigned to different classrooms so that different teachers evaluate the members of the twin pairs. If the twins are in the same classroom it is requested a second teacher or teacher's assistant complete the form to avoid the same teacher rating both twins. Unfortunatly, I do not currently have the information available regarding what percentage of the twin pairs are assigned to different teachers. The teachers are asked to rate the children in relation to the other children in the class and there is no reason to expect group differences from one class to the next.

Analysis

Genetic Analysis

Twin data can give us information about the heritability of a measure, for example, the proportion of the phenotypic variance that can be accounted for by the difference in genes among individuals (Plomin & Petrill, 1997). MZ twins, coming from a single zygote or

fertilized egg, are genetically identical. DZ twins share one half of their genes on average. If genes do not influence a trait, the DZ correlation for that trait should be about the same as the MZ correlation. If, however, the MZ correlation is greater than that of the DZ correlation, then genes do influence a trait.

Traditional twin models partition variance into additive genetic (A), shared or common environment (C), and non-shared environmental (E) components. Shared or common environment includes all those environmental influences that twins share and that make twins similar on the trait. Potential examples include aspects of the home, school, and social environment. Non-shared, or individual environment is the environment that is unique to the twin, such as the classroom if the twins were assigned to different teachers.

The path diagram for the model is illustrated in Figure 1.

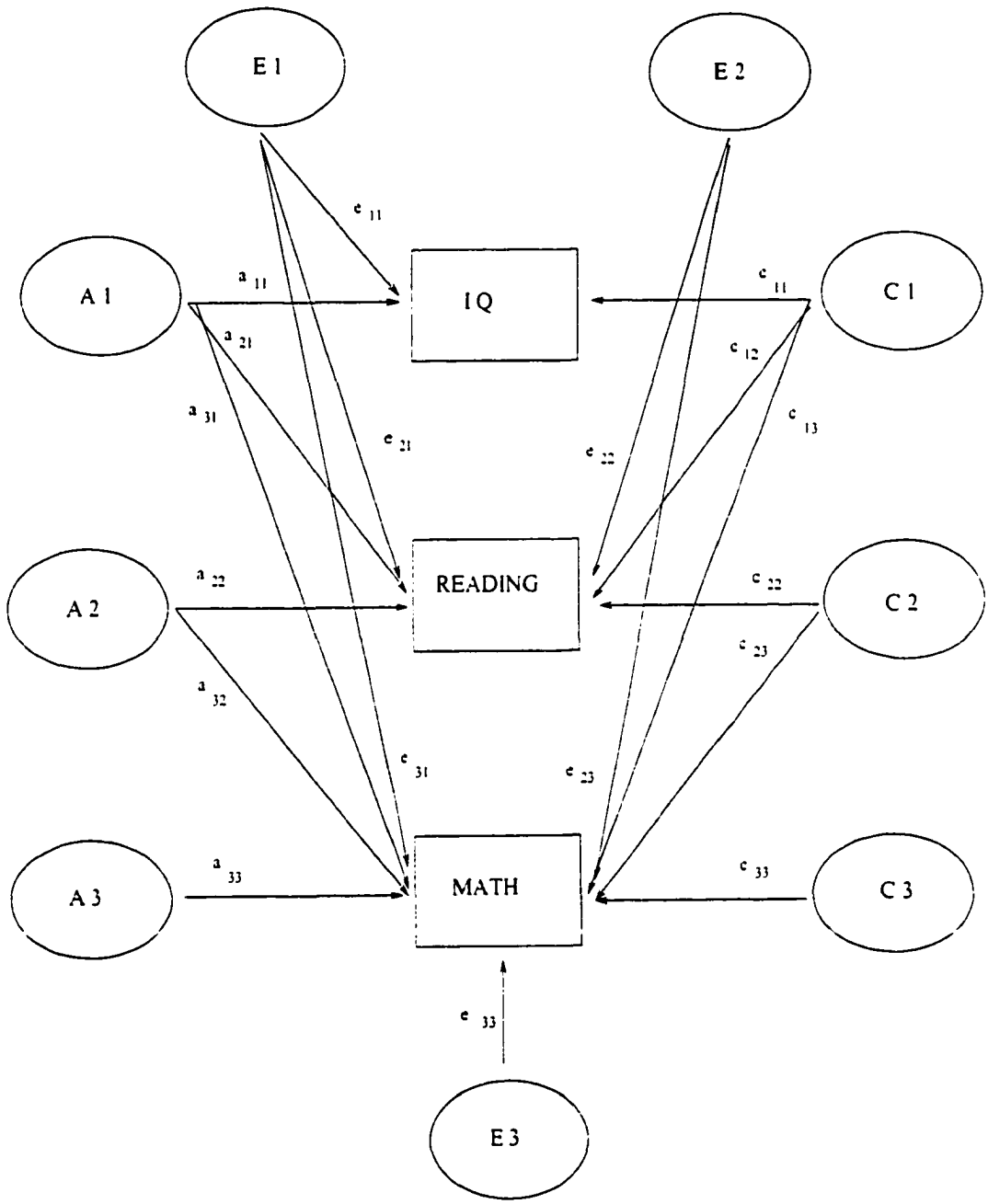


Figure 1

Path diagram illustrating the Cholesky decomposition model.

The first additive genetic component (A1) loads on, or influences all three measures. The second component (A2) loads on the last two measures, and so on. The path coefficients (factor loadings) are represented by lower case letters, "a" for additive genetics effects, "c" for common environmental effects and "e" for non-shared environmental effects. The first subscript of the lower case letters indicates the variable the path is leading to (endogenous variable), while the second corresponds to where the path begins (exogenous variable). The endogenous variable is directly caused or influenced and the exogenous variable acts as a cause (Hayduk, 1987). The full model and more restricted models (with some of the paths set to zero) were fit to determine which was the most parsimonious model that adequately fit the data.

For each of the three components of the model- A, C, and E- a matrix of the coefficients between phenotype and genotypes may be constructed. Here that coefficient matrix will be denoted as lambda (Λ) with a subscript to denote the appropriate component. For example, the coefficient matrix for A would be,

$$\Lambda_A = \begin{matrix} a_{11} & 0 & 0 \\ a_{21} & a_{22} & 0 \\ a_{31} & a_{32} & a_{33} \end{matrix}$$

Let ΣP denote the within individual covariance matrix. In terms of the model,

$$\Sigma_p = \Lambda_A \Lambda_A' + \Lambda_C \Lambda_C' + \Lambda_E \Lambda_E'$$

Let Σ_t denote the cross-twin covariance matrix. Then

$$\Sigma_t = \Upsilon\Lambda_A \Lambda_A' + \Lambda_C \Lambda_C'$$

where Υ is the correlation between the genetic values of the twins. For MZ pairs, $\Upsilon=1$ and for DZ pairs, $\Upsilon=½$. The actual covariance matrix, denoted here as Σ, contains both the within individual covariances and the cross twin covariances.

$$\Sigma = \begin{matrix} \Sigma_p & \Sigma_t \\ \Sigma_t & \Sigma_p \end{matrix} .$$

In order to use each observation in the analysis, a pedigree or raw data analysis was employed using the MX statistical package (Neale, 1995). A log-likelihood for each case is calculated separately as:

$$L_i = -1/2 \log [\Sigma_i] - 1/2 (x-\mu)' \Sigma i (x-\mu)$$

where the expected covariance matrix for the ith pedigree is Σ, x is the observed data vector within that pedigree, and the expected means is a μ vector (Neale & Cardon, 1992).

To test the significance of each component, sub-models were evaluated by constraining the components to be tested to zero. For example, to test the significance of genetic effects, the additive genetic parameters were

set to zero, thus excluding Λ_A from the model. The resulting model would then be a CE model. For model comparisons, -2 times the log-likelihood from the first model (ACE) is subtracted from that of the constrained (CE) and the df from the first model is subtracted from that of the constrained model. This statistic is asymptotically distributed as chi-square. This chi-square with its df indicate whether the additional constraints have resulted in a worse fitting model. If the new "p" value is >.05, the model fit is more parsimonious with the additional constraints (Hayduk, 1987).

After testing the significance of each component, the nature of each component was further explored in terms of significant parameters within the component. For example, as shown in Figure 2, only one common genetic factor is allowed to influence the three variables. Again, model comparisons determine which model best represents the data.

Finally, models were tested to see if dominance was important in explaining the variance of the measures.

Results

Descriptive Statistics

As shown in Table 1, the mean WISC-R score for this

sample was 106.57 with a standard deviation of 13.48. This mean is above the population expectation of 100, probably due to some combination of the educated nature of the sample and cohort effects from the standardized sample. Hoverer, the standard deviation is very close to its population value of 15. The mean reading and math scores were 3.28 (s.d. 1.0) and 3.39 (s.d. .82) respectively.

Table 1.

Descriptive Statistics for 7 Year Data.

Measure	MZ pairs	DZ pairs	Mean	Std Dev
IQ	162	138	106.57	13.48
Reading	117	96	3.28	1.00
Math	117	94	3.39	.82

Table 2-a.

Within Individual and Cross Twin Correlations for 7 Year. Data

Within Individuals	
WISC-R - G-Read	.45
WISC-R - G-Math	.51
G-Read - G-Math	.72

Cross Twins			
Measure		MZ twins	DZ twins
Twin 1	Twin 2		
WISC-R -	WISC-R	.81	.58
G-Read -	G-Read	.79	.32
G-Math -	G-Math	.62	.27
WISC-R -	G-Read	.62	.24
WISC-R -	G-Math	.54	.28
G-Read -	G-Math	.66	.35

Note. All correlations are significant at $p<.001$

Table 2-b.

Correlations Between Standardized Tests and Teacher. Report

Between Measures	
PIAT Read - G-Read	.69
PIAT Read - G-Math	.49
Key Math - G-Read	.30
Key Math - G-Math	.34

As shown in Table 2, the correlations between measures as well as the correlations between MZ and DZ twins were computed. The correlations are as follows: WISC-R and Math=.45, WISC-R and Reading = .51, Reading and Math = .72. The correlation between the academic achievement measures is greater than the correlations between achievement and IQ. MZ twins have greater correlations than DZ twins indicating that genes influence these measures.

Correlations were also calculated for teacher ratings on math and reading and two standardized tests used to measure math and reading. The first test is the PIAT reading recognition test and the second one is the KEY Math test. The correlations range from .30 to .69.

ACE Model Comparisons

The results of the Cholesky Decomposition and the model comparisons are shown in Table 3.

Model 1 shows the results of the full model with -2 times the log likelihood of the data being 6260.70 with 1417 df. Constraining the common environment (model 2) and additive genetic effects (model 3) to zero results in a worse fitting models ($p<.05$ and $p<.001$ respectively). Model 4 constrains the additive genetic component to be represented by a single common factor as shown in figure 2.

Table 3

Model Comparisons for 7 Year Data.

Model	-2 x log	vs.	$\chi 2$	df	p
1) ACE	6260.70				
2) AE	6275.20	1	14.72	6	<.05
3) CE	6322.42	1	61.72	6	<.001
4) A_1CE	6261.20	1	.50	3	>.90
5) AC_2E	6264.00	1	3.30	5	>.50
6) A_1C_2E	6271.05	1	10.35	8	>.30
7) No E common factors	6284.10	1	23.40	3	<.001

Note. A = additive genetic component; C = common environmental component; E = individual environmental component. Subscript 1 denotes a single common factor loading on each measure (figure 2). Subscript 2 denotes a single factor loading on WISC-R only.

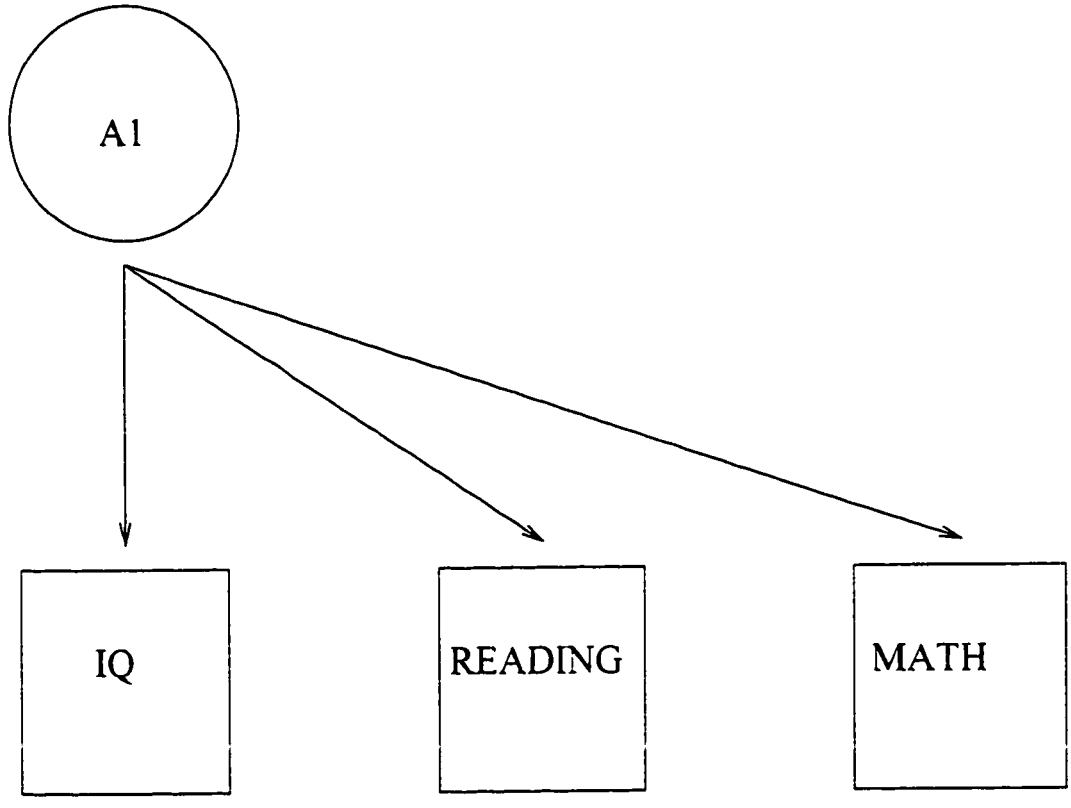


Figure 2

Single common factor at age 7.

The resulting chi-square of .5 with 3 df indicates this model is a more parsimonious model and better represents the data. In this case only one factor is needed to explain the genetic variation of all three measures. Model 5 constrains the common environmental component to a single factor loading on just the WISC-R. Many other models were fit, and this one proved to best represent C with a chi-square of 3.3 with 5 df. Model 6 combines the best representation of A and C and results in a chi-square of 10.35 with 8 df, again showing a more parsimonious model. Finally, model 7 shows one of many attempts at dropping parameters for the individual environmental component. All of theses attempts, however resulted in worse fitting models, so E is needed in full form.

ADE Model Comparisons

Because the DZ twin correlations were less than half that of the MZ correlations in some cases, dominance was explored by model comparisons shown in Table 4. The full ADE model resulted in -2 times the log likelihood of 6274.08. Constraining the dominance component to be zero (model 2) resulted in a chi-square of 1.12 with 6 degrees of freedom. This is a more parsimonious model, indicating

that dominance is not needed.

In model 3, the additive genetic component was constrained to zero and resulted in a worse fitting model (chi-square = 26.56, df = 6).

Parameters for the best fitting model are presented in Figure 3.

Table 4

Model Comparisons with Dominance for 7 Year Data.

Model	-2 x log	vs.	$\chi 2$	df	p
1) ADE	6274.08				
2) AE	6275.20	1	1.12	6	>.90
3) DE	6300.64	1	26.56	6	<.001

Note. A = additive genetic component; D = dominance component; E = individual environmental component.

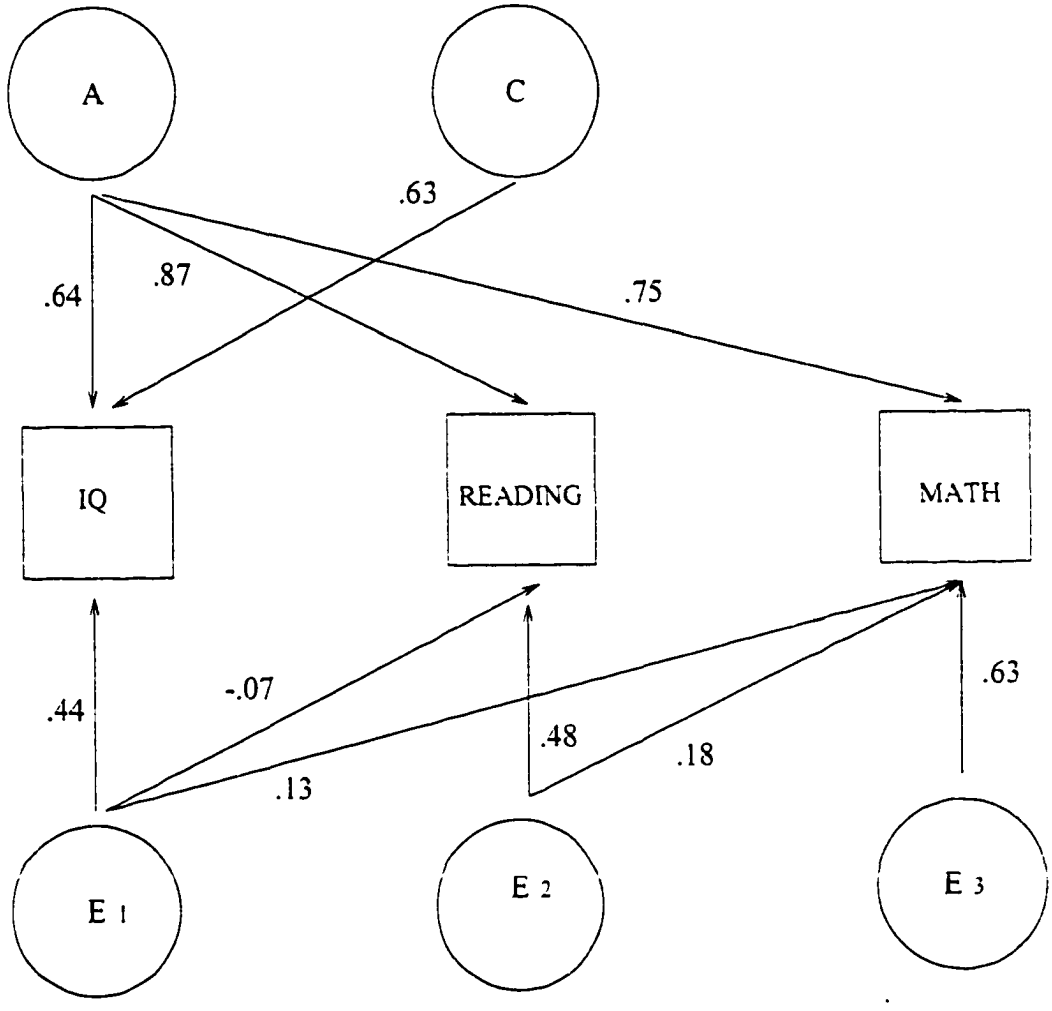


Figure 3

Path diagram for most parsimonious model at age 7.

The most parsimonious model resulted in a single genetic component loading on all three variables, a single common environmental component loading on just WISC-R, and a full model representing the individual environmental component. The standardized path coefficients can be squared to account for the total variance of the phenotypes as shown in Table 5.

Table 5

Heritability, Common and Individual Environmental Influences for Age 7.

Measure	h^2	c^2	e^2
IQ	.41	.40	.19
READING	.76	0	.23
MATH	.56	0	.43

Note. h^2=heritability; c^2=common environment; e^2=individual environment

Most of the variance in the three measures can be accounted for by genetic effects. Reading had the highest heritability with 76% of the variance accounted for by genes. Heritability accounted for 56% of the variance for math. The proportion of variance in WISC-R accounted for by genes was 41%. The only measure that the common environment influenced was WISC-R where 40% of the variance was accounted for. The individual environment accounted for 43% of the variance in math, 23% of the variance in reading and 19% of the variance in the WISC-R score.

Genetic Correlations

The genetic correlations were calculated and are shown in Table 6.

Table 6

Genetic Correlations for 7 Year Data.

Measure	IQ	Reading	Math
IQ	1.00		
Reading	.90	1.00	
Math	.90	.98	1.00

The genetic correlations between IQ and academic achievement are .90. It is slightly higher between reading and math at .98. Virtually all of the co-variation between IQ and academic achievement can be accounted for by shared genetic factors.

Discussion

The findings of this study show results that have similarities to some studies, (Thomas, 1991; Brooks, Fulker & DeFries, 1990 & Gillis, DeFries & Fulker, 1992) as well as differences (Martin, 1975; Gill et al., 1985 & Wadsworth, 1994). Like other studies, we found that most of the observed covariation between IQ and academic achievement can be accounted for by genetic factors.

Unlike Martin (1975) and Gill et al (1985), the current analysis found IQ to be less heritable than academic achievement. This difference may be due to the different measures of academic achievement. As Martin acknowledged, their study used large impersonal testing programs to measure academic achievement.

Wadsworth's 1994 adoption study found lower heritabilities for academic achievement than this study. Again, the differences may be due to the way in which academic achievement was measured. However, Gillis, DeFries & Fulker (1992) found very similar heritabilities

for reading and math in their twin study as we found in the current analysis.

Our most striking finding was that the common environment did not account for any of the variation in academic achievement, while it accounted for 40% of the variance in IQ. While striking, the results aren't necessarily surprising. IQ, as measured by the WISC-R is a global measure, while academic achievement in this study is specific to the classroom. In Colorado it is common for twins to be assigned different classrooms, hence they do not share the classroom environment. It is possible that the teaching methods, ways of grading, how noisy the classroom is etc. would influence the children's achievement. These differences would contribute to the individual environmental component as opposed to the common environmental component.

The correlations between teacher ratings and standardized tests in math and reading, while statistically significant, are lower than one would expect if they were measuring the same thing.

In conclusion, academic achievement as measured by how well a child performs in a classroom, may be a different measure than how well a child performs on a standardized test, whether it is an IQ test or an academic achievement test. When measured in this way, the factors that influence IQ and academic achievement are

somewhat different although the genetic correlations between IQ and reading and math achievement remain high.

Chapter III

Longitudinal Analysis of IQ and Academic Achievement.

Methods

Subjects

Participants of the LTS have been described in previous sections. The current study focuses on measures of intelligence (IQ) and academic achievement in children ranging from 14 months to age nine. Sizes for the variables at each age are shown in Table 7 among with the means and standard deviations.

Measures

To assess IQ in infancy and early childhood, the Bayley Scales of Infant Development (Bayley, 1969) were administered at 14, 20, and 24 months. At ages three and four years, the Stanford-Binet Intelligence Scale (Terman & Merrill, 1973) was used to measure IQ. A full scale Wechsler Intelligence Scale for Children - Revised or WISC-R (Wechsler, 1974) was administered to assess IQ at age seven. Academic achievement was assessed at ages 7 and 9 and was determined by teacher report. The teachers completed the Child Behavior Checklist (CBC) (Achenbach & Edelbrock, 1986) in which they were asked to rate the

Table 7

Sample Size, Means, and Standardized Deviations for Longitudinal Analysis.

Measure	MZ	DZ	Mean	Std Dev
IQ				
14 mo	212	186	104.47	13.91
20 mo	174	149	104.32	17.45
24 mo	196	175	107.46	19.04
3 yr	182	164	103.16	17.49
4 yr	192	167	103.83	14.13
7 yr	156	134	106.54	13.59
Achievement				
Math 7	112	91	3.39	.83
Read 7	112	93	3.28	1.00
Math 9	62	43	3.47	.83
Read 9	62	43	3.50	.98

child's performance in reading and math on five point scale ranging from far below grade level (1) to far above grade level (5).

Analysis

Multivariate genetic analysis was carried out to investigate the covariance between IQ and academic achievement. Genetic influence on the covariation between IQ and academic achievement is indicated when crosstwin correlations (correlating one twin's IQ with the cotwins academic achievement) are greater for MZ than for DZ twins (Plomin & Petrill, 1997). The genetic correlation determines how much of the covariation between the measures is accounted for by shared genetic factors.

Traditional twin models have been explained in previous chapters. The path model for the additive genetic component is illustrated in Figure 4.

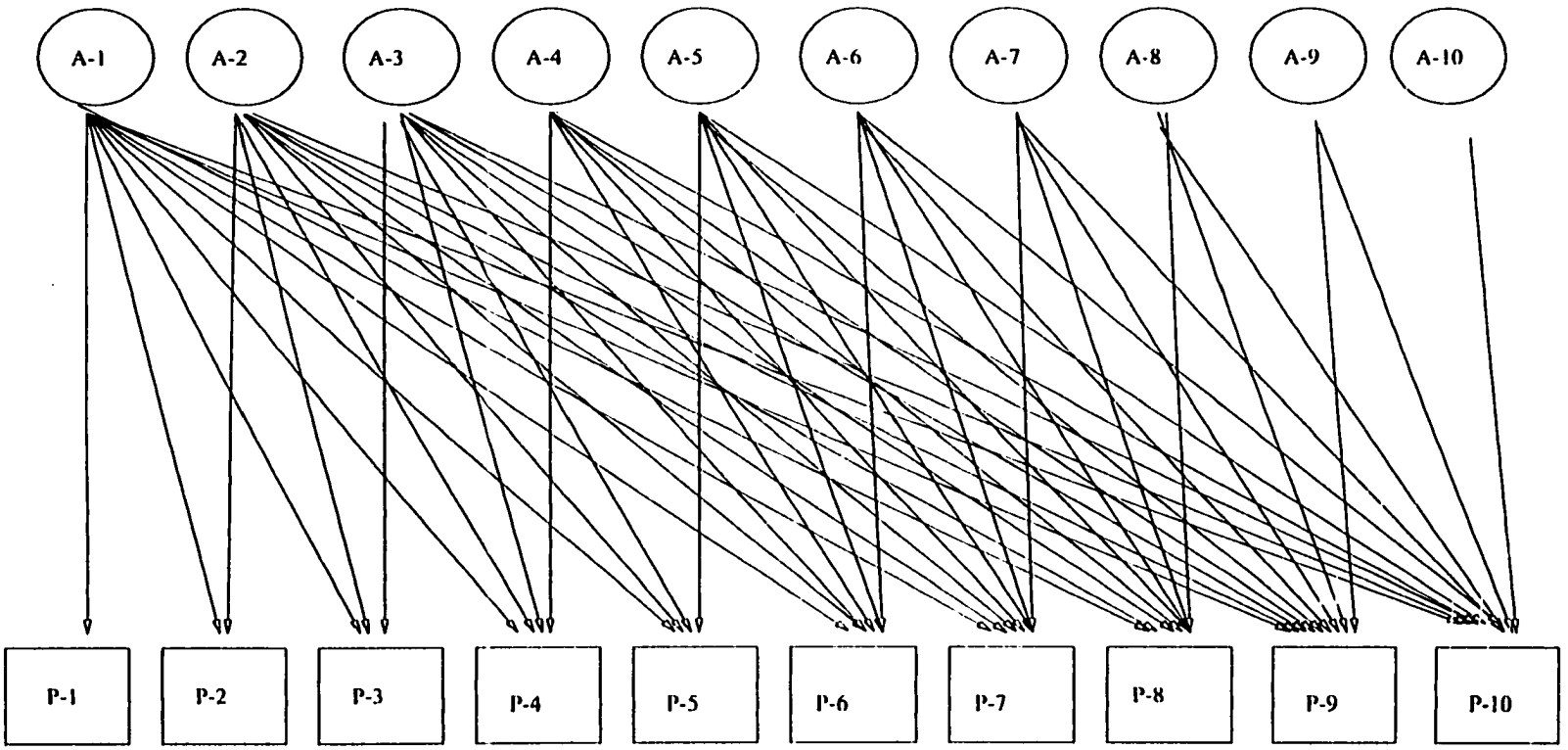


Figure 4

Cholesky decomposition model showing the additive genetic path model for longitudinal analysis.

The first additive genetic component (A1) may be interpreted as the genetic effects that influence scores on the Bailey Scales at age 1. These same genetic effects may influence subsequent intelligence as well as academic achievement. This is depicted in Figure 3 by the arrows originating in A1 and ending in P2, P3 etc.. The second genetic component (A2) represents the possibility that new genetic effects may enter at age 2 and also carry over to subsequent years (i.e. paths from A2 to P3, P4, etc.).

The common environment and the unique environment are parameterized in a similar fashion to the additive genetic effects.

The notation for the path coefficients (factor loadings) are represented by lower case letters, a for additive genetics effects, c for common environmental effects and e for non-shared environmental effects. The first subscript of the lower case letters indicates the variable the path is leading to (endogenous variable), while the second corresponds to where the path begins (exogenous variable). The endogenous variable is directly caused or influenced and the exogenous variable acts as a cause (Hayduk, 1987). The full model and more restricted models (with some of the paths set to zero) were fit to determine which was the most parsimonious model that adequately fit the data

For each of the three components of the model (A, C, and E) a matrix of the coefficients between phenotype and genotypes may be constructed. Here that coefficient matrix will be denoted as lambda (Λ) with a subscript to denote the appropriate component. For example, the coefficient matrix for A would be,

$$\Lambda_A = \begin{bmatrix} a_{1,1} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{2,1} & a_{2,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{3,1} & a_{3,2} & a_{3,3} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{4,1} & a_{4,2} & a_{4,3} & a_{4,4} & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{5,1} & a_{5,2} & a_{5,3} & a_{5,4} & a_{5,5} & 0 & 0 & 0 & 0 & 0 \\ a_{6,1} & a_{6,2} & a_{6,3} & a_{6,4} & a_{6,5} & a_{6,6} & 0 & 0 & 0 & 0 \\ a_{7,1} & a_{7,2} & a_{7,3} & a_{7,4} & a_{7,5} & a_{7,6} & a_{7,7} & 0 & 0 & 0 \\ a_{8,1} & a_{8,2} & a_{8,3} & a_{8,4} & a_{8,5} & a_{8,6} & a_{8,7} & a_{8,8} & 0 & 0 \\ a_{9,1} & a_{9,2} & a_{9,3} & a_{9,4} & a_{9,5} & a_{9,6} & a_{9,7} & a_{9,8} & a_{9,9} & 0 \\ a_{10,1} & a_{10,2} & a_{10,3} & a_{10,4} & a_{10,5} & a_{10,6} & a_{10,7} & a_{10,8} & a_{10,9} & a_{10,10} \end{bmatrix}$$

Let ΣP denote the within individual covariance matrix. In terms of the model,

$$\Sigma_p = \Lambda_A \Lambda_A' + \Lambda_C \Lambda_C' + \Lambda_E \Lambda_E'$$

Let Σ_t denote the cross-twin covariance matrix. Then

$$\Sigma_c = \Upsilon\Lambda_A \Lambda_A' + \Lambda_C \Lambda_C'$$

where Υ is the correlation between the genetic values of the twins. For MZ pairs, Υ=1 and for DZ pairs, Υ=½ . The actual covariance matrix, denoted here as Σ, contains both the within individual covariances and the cross twin covariances.

There are considerable amounts of missing data in the LTS, particularly at the later ages due to the simple fact that a large number of twins have yet to reach the later ages. In order to use each observation in the analysis, a pedigree or raw data analysis was employed using an MX statistical package (Neale, 1995). A log-likelihood for each case is calculated separately as:

$$L_i = -1/2 \log [\Sigma_i] - 1/2 (x-\mu)' \Sigma i (x-\mu) + \text{constant}$$

where Σ_i is the expected covariance matrix for the ith pedigree, $\mathbf{x}$ is the vector of observed data within that pedigree, and $\boldsymbol{\mu}$ it the vector of means for the pedigree (Neale & Cardon, 1992).

To test the significance of each component, sub-models were evaluated by setting certain parameters to zero. For example, to test the significance of genetic effects, the additive genetic parameters (i.e. $a_{i,j}$) were set to zero, thus excluding A from the model. The

resulting model would then be a CE model. To compare models, -2 times the log-likelihood from the first model (ACE) is subtracted from that of the constrained model (CE). This statistic is asymptotically distributed as chi-square where the degrees of freedom equal the number of parameters fit in the first model less the number of parameters fit in the constrained model. If the p value of the chi-square is <.05, then the constrained model is rejected (Hayduk, 1987).

I began by testing the significance of each component (i.e. A=0 and C=0). Afterwards, the nature of each component was further explored in terms of significant parameters within the component. For example, as shown in Figure 5, only one common genetic factor is allowed to influence the variables.

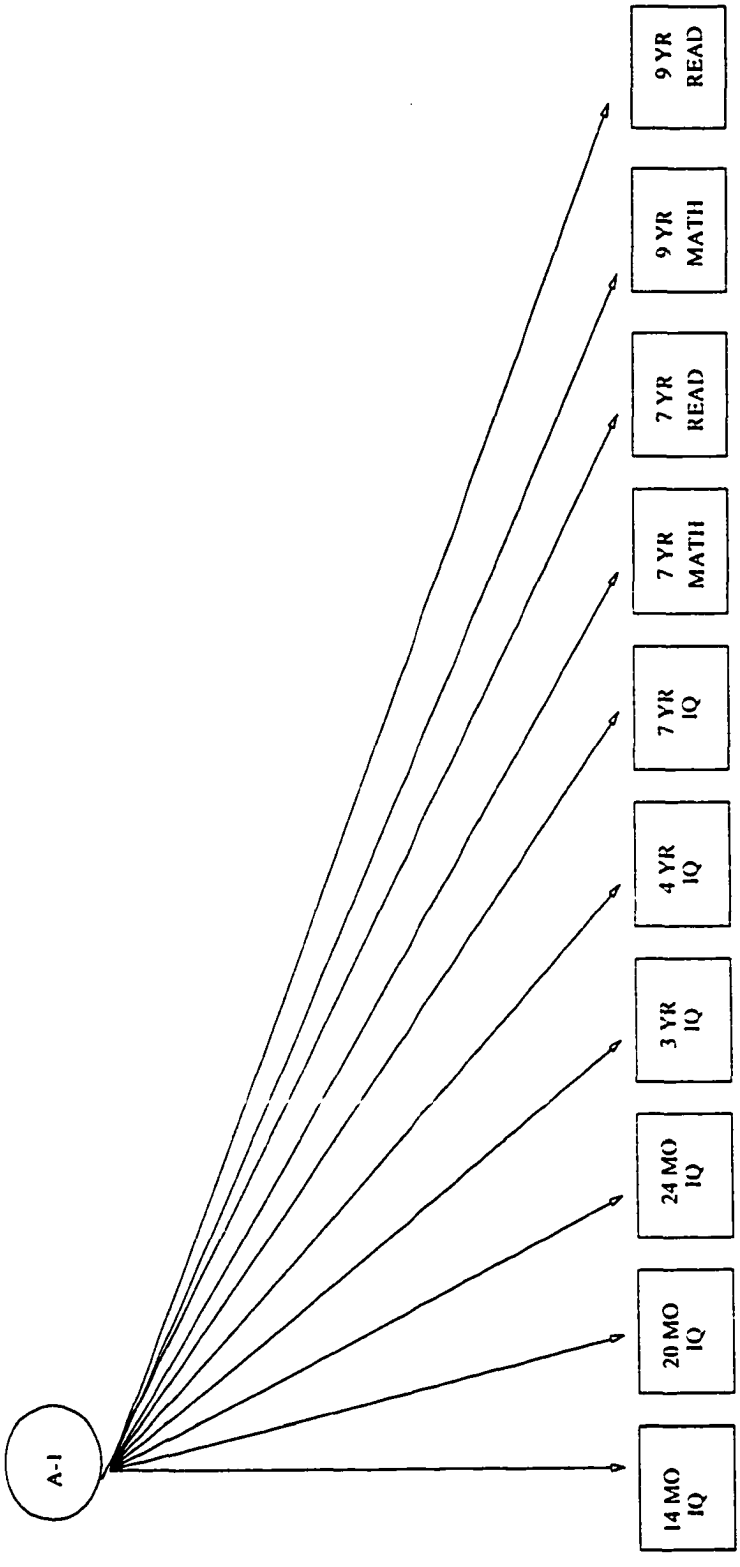


Figure 5

Single genetic factor for the longitudinal analysis.

This model tests the substantive hypothesis of "innate genetic ability" or the possibility that genes "hardwire" us at birth for intelligence and the genetics effects simply carry over from one age to another.

I tested the following models against the general model:

1. That there is a single genetic factor influencing IQ and academic achievement at all ages (see figure 4).
2. That there is a single environmental factor influencing IQ and academic achievement at all ages.
3. That there are two genetic factors, one influencing IQ and the other influencing academic achievement (see figure 5).
4. That there are two genetic factors, one influencing IQ and academic achievement and the other influencing only academic achievement (see figure 6).
5. That there is a single common factor influencing all of the measures as well as age specific factors (see figure 7).
6. That there is a genetic factor for each measure (see figure 8).

Results

Descriptive Statistics and Correlations

The correlations between twins are shown in Table 8.

Table 8

Correlations Between Twins at Each Age for the Longitudinal.

Measure	MZ	DZ
IQ		
14 mo	.58	.41
20 mo	.81	.65
24 mo	.84	.63
3 yr	.77	.51
4 yr	.77	.51
7 yr	.81	.58
Achievement		
Math 7	.62	.28
Read 7	.79	.33
Math 9	.67	.34
Read 9	.63	.32

For all of the measures, the correlations for MZ twins are greater than that of the DZ twins. For IQ, the difference in the correlations between the MZ and DZ twins tends to increase with age, indicating an increase in heritability. All of the correlations are statistically significant.

The within-individual correlations between IQ measures are shown in Table 9.

Table 9

Within-Individual Correlations Between IQ Measures for the Longitudinal Analysis.

	IQ 14-mo	IQ 20-mo	IQ 24-mo	IQ 3-yr	IQ 4-yr
IQ 20-mo	.48				
IQ 24-mo	.42	.68			
IQ 3 yr	.29	.55	.63		
IQ 4 yr	.25	.53	.59	.68	
IQ 7 yr	.23	.48	.51	.56	.59

The correlations range from .23 (IQ at 14 months and age 7) to .68 (IQ at 20 and 24 months and IQ at ages 3 and 4). The correlations decrease as time between the measures increase giving a simplex-like pattern to the correlation matrix. All of the correlations are significant.

The within-individual correlations between IQ and achievement are shown in Table 10.

Table 10

Within-Individual Correlations Between IQ and Achievement Measures for the Longitudinal Analysis.

	IQ 14-mo	IQ 20-mo	IQ 24-mo	IQ 3-yr	IQ 4-yr	IQ 7-yr
Math 7	.14	.34	.28	.29	.34	.52
Read 7	.10	.39	.32	.32	.39	.46
Math 9	.04*	.20	.22	.33	.31	.50
Read 9	.07*	.19	.24	.35	.41	.51

Note. *correlations not significant at .05

The correlations between IQ and achievement measures range from .04 (IQ at 14 months and math at 9) to .52 (IQ and math at age 7). The only correlations that are not significant are the correlations between IQ at 14 months and achievement at age 9. The correlations tend to increase as the age discrepancy decreases. There are no trends to indicate that either math or reading correlate more highly with IQ.

The within individual correlations between achievement measures are shown in Table 11.

Table 11.
Within-Individual Correlations Between Achievement Measures for the Longitudinal Analysis.

	Math 7	Read 7	Math 9
Read 7	.72		
Math 9	.51	.46	
Read 9	.46	.56	.66

The correlations are all significant and range from .46 to .72. The highest correlation is between math and reading at age 7.

Genetic Analysis

Heritability

Heritability was calculated from the full model and the results are shown in Table 12. The first number (.38) is the squared path coefficient for the path going from A1 to IQ at 14 months. It is interpreted as "38% of the variance in IQ at 14 months can be accounted for by additive genetic effects." The second number(.11) is the path coefficient from A1 to IQ at 20 months. The third number (.20) is the path coefficient from A2 to IQ at 20 months, and so on.

The amount of variance in IQ that can be accounted for by genetic factors ranges from 31% to 46% and has a tendency to increase with age. For academic achievement, between 38% and 61% of the variation can be accounted for by genetic effects.

Table 12

Proportion of Variance Accounted for by Heritability for the Longitudinal Analysis.

	A1	A2	A3	A4	A5	A6	A7	A8	A9	A10	TOTAL
14 mo	.38										.38
20 mo	.11	.20									.31
24 mo	.14	.06	.13								.33
3 yr	.07	.01	.09	.23							.40
IQ 4 yr	.05	.04	.02	.26	.03						.40
IQ 7 yr	.07	.02	0	.19	.02	.16					.46
MATH 7 yr	.08	.04	0	.04	0	.27	0				.43
READ 7 yr	.01	.21	0	.12	0	.26	.01	0			.61
MATH 9 yr	0	0	0	.07	0	.21	.10	0	0		.38
READ 9 yr	.01	.08	0	.12	.13	.14	.07	0	0	0	.55

The genetic effects on IQ at early ages appear to "die down" at later ages. There is a noticeable shift from the first three ages to age four. The component coming in at age 4 has lasting effects, as does the component coming in at age 7. This also corresponds with the change of measuring IQ from the Bayley to the Stanford Binet and from the Stanford Binet to the WISC-R.

The genetic effect from early IQ on academic achievement may be non-zero, but it is small. Most of the genetic effect comes from age 4 on and is particularly strong at age 7.

Almost all of the genetic effects on academic achievement can be predicted by the genetic effects on IQ.

Common Environment

The amount of variation in IQ accounted for by the common environment is shown in Table 13 and ranges from 17% to 50%.

Overall, the common environment influences IQ more than achievement. There are two primary environmental components accounting for most of the variation, one present at 14 months and one at 20 months. There is very little new or residual common environmental affects after coming in after 20 months.

Table 13

Variation Accounted for by the Common Environment for the Longitudinal Analysis.

	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	TOTAL
14 mo	.21										.21
20 mo	.26	.24									.50
24 mo	.15	.30	.04								.49
3 yr	.11	.25	0	0							.36
IQ 4 yr	.08	.21	.03	0	0						.32
IQ 7 yr	.02	.23	.01	.06	.01	.02					.35
MATH 7 yr	0	.04	.02	.05	.06	0	0				.17
READ 7 yr	.01	.03	0	.11	.03	0	0	0			.18
MATH 9 yr	.03	.12	.10	.03	.01	0	0	0	0		.29
READ 9 yr	.03	.07	.04	.02	.01	0	0	0	0	0	.17

49

Individual Environment

The amount of the variation in IQ and achievement accounted for by the individual environment is shown in Table 14. The individual environment accounts for between 17% to 41% of the variation in IQ and academic achievement. The largest environmental component is present at 14 months and has no residual effects. The individual environmental effects are by and large, time specific.

Genetic Correlations

Genetic correlations are shown in Table 15. The covariation between IQ and achievement that can be accounted for by shared genetic effects varies. Within the IQ measures, the genetic correlation ranges from .35 to .91. For achievement the range is between .68 to .91.

Math at age 9 has the weakest genetic correlations with IQ ranging from .04 at 14 months, increasing to .70 at age 7. The other correlations also have a tendency to increase with age. By age 7, between 70% and 89% of the covariation between IQ and achievement can be accounted for by shared genetic factors.

Table 14

Variation Accounted for by the Individual Environment for the Longitudinal Analysis.

	E1	E2	E3	E4	E5	E6	E7	E8	E9	E10	TOTAL
14 mo	.41										.41
20 mo	0	.18									.18
24 mo	0	0	.17								.17
IQ 3 yr	0	.01	0	.22							.23
IQ 4 yr	0	0	0	0	.23						.23
IQ 7 yr	0	0	0	0	0	.17					.17
MATH 7 yr	0	.01	0	0	0	0	.36				.36
READ 7 yr	0	0	0	0	0	0	.03	.16			.19
MATH 9 yr	0	0	0	0	0	.01	0	0	.28		.29
READ 9 yr	0	0	0	.03	0	0	0	0	.03	.21	.27

Table 15

Genetic Correlations for the Longitudinal Analysis.

	IQ 14-mo	IQ 20-mo	IQ 24-mo	IQ 3-yr	IQ 4-yr	IQ 7-yr	MATH 7-yr	READ 7-yr	MATH 9-yr
IQ 20-mo	.59								
24-mo	.66	.73							
3-yr	.41	.36	.64						
4-yr	.35	.47	.50	.91					
7-yr	.39	.41	.38	.70	.79				
Math 7	.42	.50	.39	.43	.50	.89			
Read 7	.13	.55	.33	.46	.61	.86	.91		
Math 9	.04	.06	.03	.24	.31	.70	.74	.68	
Read 9	.14	.40	.18	.40	.65	.82	.77	.87	.77

Model Comparisons

The model comparisons are shown in Table 16. Model comparisons were analyzed to test different hypothesis regarding the etiology of IQ and academic achievement. The significance of each component was tested and was further explored in terms of significant parameters within the component.

The full ACE model resulted with -2x log likelihood of 34514.41. The second model dropped the additive genetic component and the chi-square resulting from comparing this model to the full model is 159.88 with 55 degrees of freedom and $p < .01$. This finding indicates that it is a less parsimonious model.

Model 3 omits the common environmental component. The resulting chi-square is 67.20 with 55 degrees of freedom and a $p = .13$ indicating a more parsimonious model. This model was further explored by constraining all of the additive genetics path coefficients that are less than .05 to zero (Model 4). The resulting chi-square when compared to the AE is 31.81 with 14 degrees of freedom. The $p < .00$ indicates a less parsimonious model.

Table 16

Model Comparisons for the Longitudinal Analysis.

Model	-2x log	vs	chi-square	df	p
1) ACE	34514.41	1			
2) CE	34674.29	1	159.88	55	.00
3) AE	34581.61	1	67.20	55	.13
4) A_1E	34613.42	3	31.81	14	.00
5) A_2CE	34582.09	1	67.68	45	.02
6) A_3CE	34609.83	1	94.42	45	.00
7) A_4CE	34577.20	1	62.79	41	.02
8) A_5CE	34619.27	1	102.89	45	.00
9) A_6CE	34541.01	1	26.60	35	.85
10) A_6C_7E	34592.20	9	51.19	36	.05
11) A_6C_8E	34559.23	9	18.22	28	.92
12) $A_6C_8E_9$	34610.32	10	51.09	42	.16

Note. A=additive genetic component; C=common environmental component; E=individual environmental component. Subscript 1 denotes constraining path coefficients <.05 to zero. Subscript 2 denotes a single genetic factor loading on all ages. Subscript 3 denotes one genetic factor loading on only IQ and one factor loading on academic achievement. Subscript 4 denotes one genetic factor loading on all measures and one factor loading on academic achievement. Subscript 5 denotes one genetic factor per measure (e.g. One factor for Bayley, one for Stanford Binet etc.) Subscript 6 denotes one factor to IQ measures, one factor to age 3 and over, and one factor to age 7 and over. There are also age specific factors at ages 20 and 24 months. Subscript 7 denotes one common environmental factor loading on all components as well as age/measurement specific components. Subscript 8 denotes two factors to all measures, age specific factors to ages 3 and 4, and one factor influencing all measures at age 7 and up.

Model 5 leaves the common and environmental component in full form and constrains the additive genetic component to a single common genetic factor influencing all of the measures as illustrated in Figure 5. This test the hypothesis that genes "hardwire" us at birth for intelligence and ability and simply carry over from one age to another. Comparing this to the full model results in a chi-square of 67.68 with 45 degrees of freedom. The p=.02 indicates a less parsimonious model.

Model 6 constrains the path coefficients to 2 genetic factors, one loading on only on IQ and one loading on academic achievement. This model is shown in Figure 6. Model 6 tests the hypothesis that there is one genetic factor present at birth that influences IQ through age 7 and another genetic factor that is present or "turns on" at age 7 that influences academic achievement. In this case there are two different and separate genetic components influencing IQ and achievement. Comparing this to the full model results in a chi-square of 94.42 with 45 degrees of freedom and a p<.00 indicating a less parsimonious model.

Model 7 constrains the genetic path coefficients to two factors, one loading on all the variables and one loading only on academic achievement. This path diagram is illustrated in Figure 7.

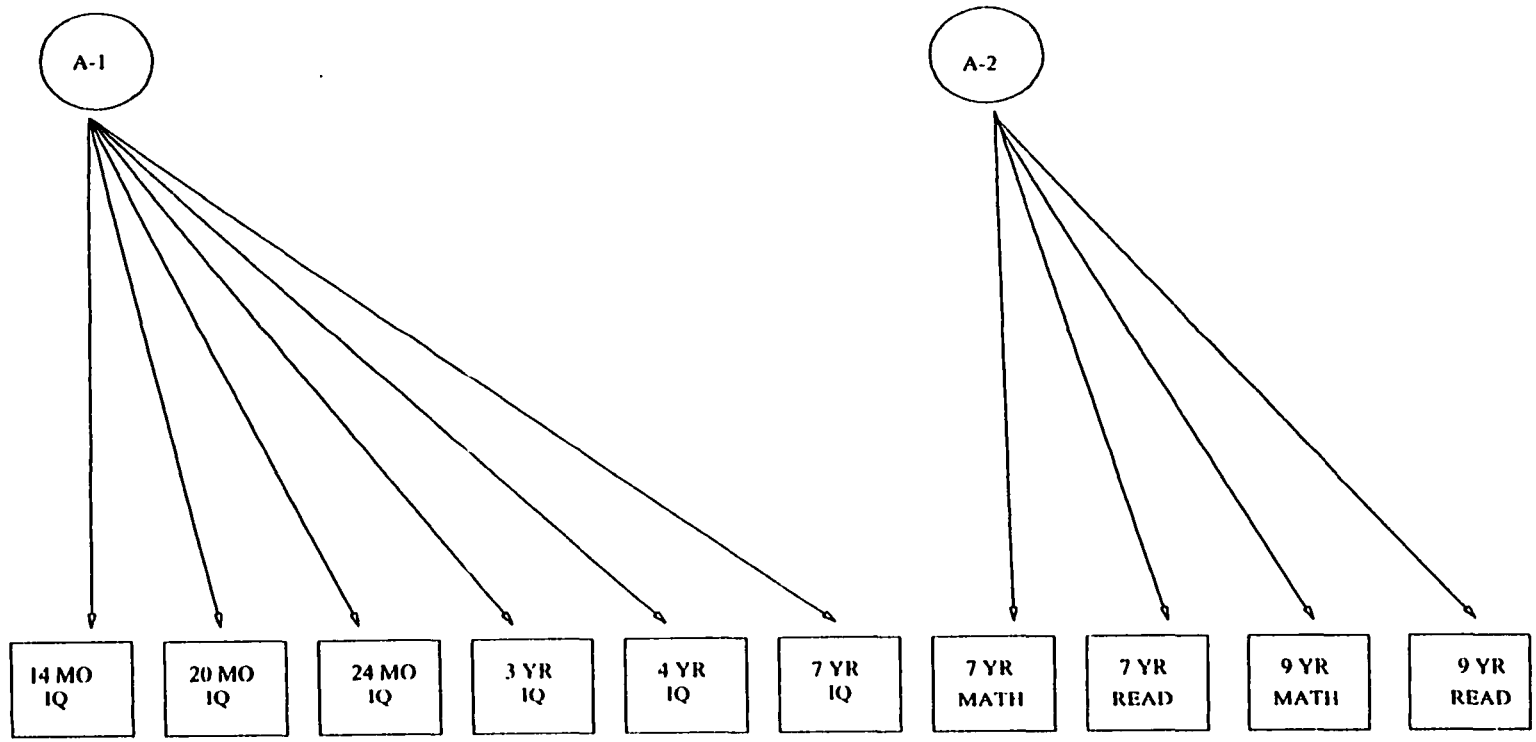


Figure 6

One IQ and one achievement factor for the longitudinal analysis.

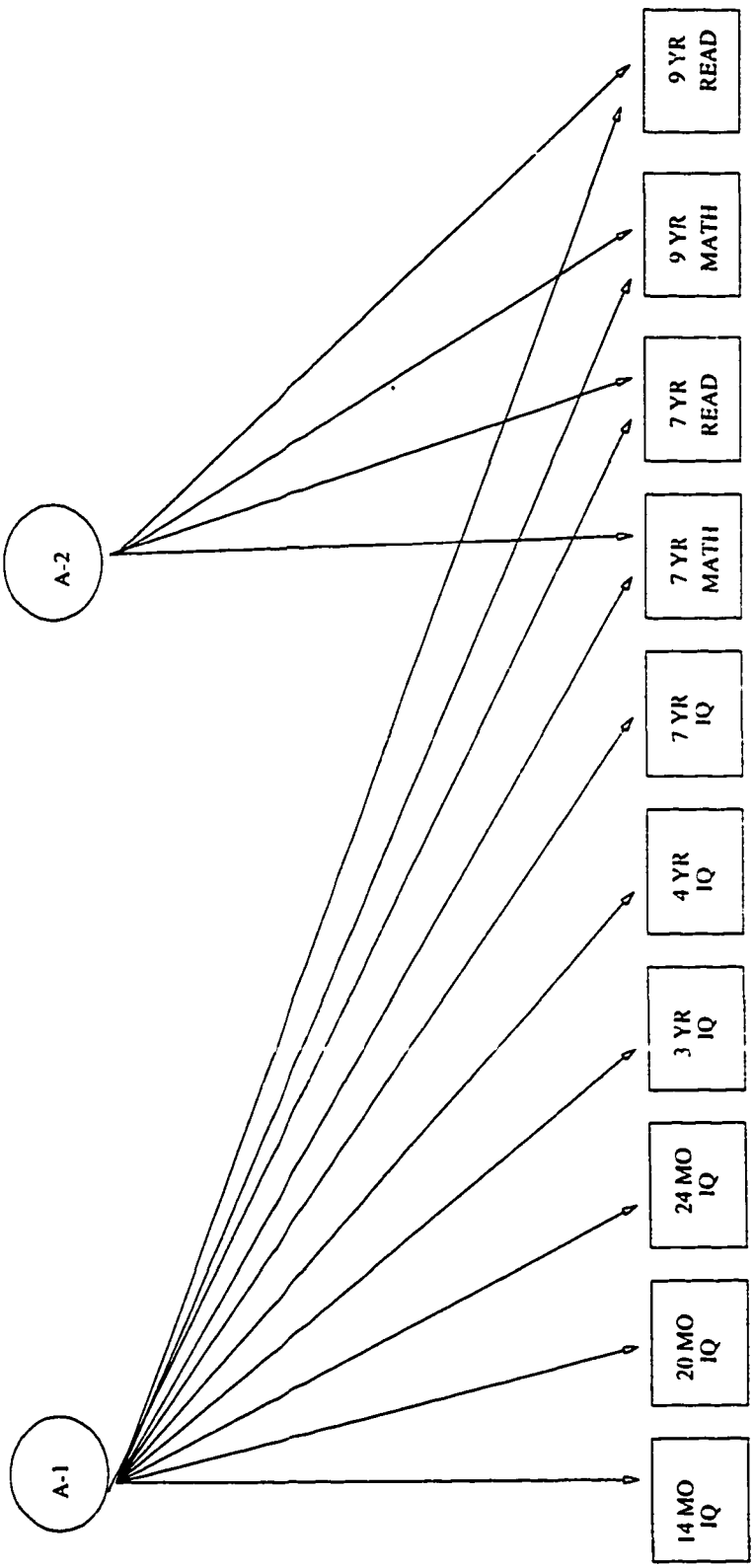


Figure 7

One factor influencing all measures and one achievement factor.

Comparing this to the full model results in a chi-square of 62.79 with 41 degrees of freedom. The resulting p =.02 indicates a less parsimonious model.

Model 8 constrains the path coefficients to represent one genetic factor per measure as shown in figure 8. Model 8 tests the hypothesis that there is a different set a genes influencing each measure (Figure 8). This would indicated that at birth we have genes that determine how one would score on the Bayley at 14, 20, and 24 months, a new set of genes would come in at age 3 and determine the performance on the Stanford-Binet, etc.. If the data fit this model, it might also indicated that Stanford Binet, Bayley, and WISC-R were not measuring the same thing. Comparing this to the full model results in a chi-square of 102.89 with 45 degrees of freedom. The resulting $p<.001$ indicates a less parsimonious model.

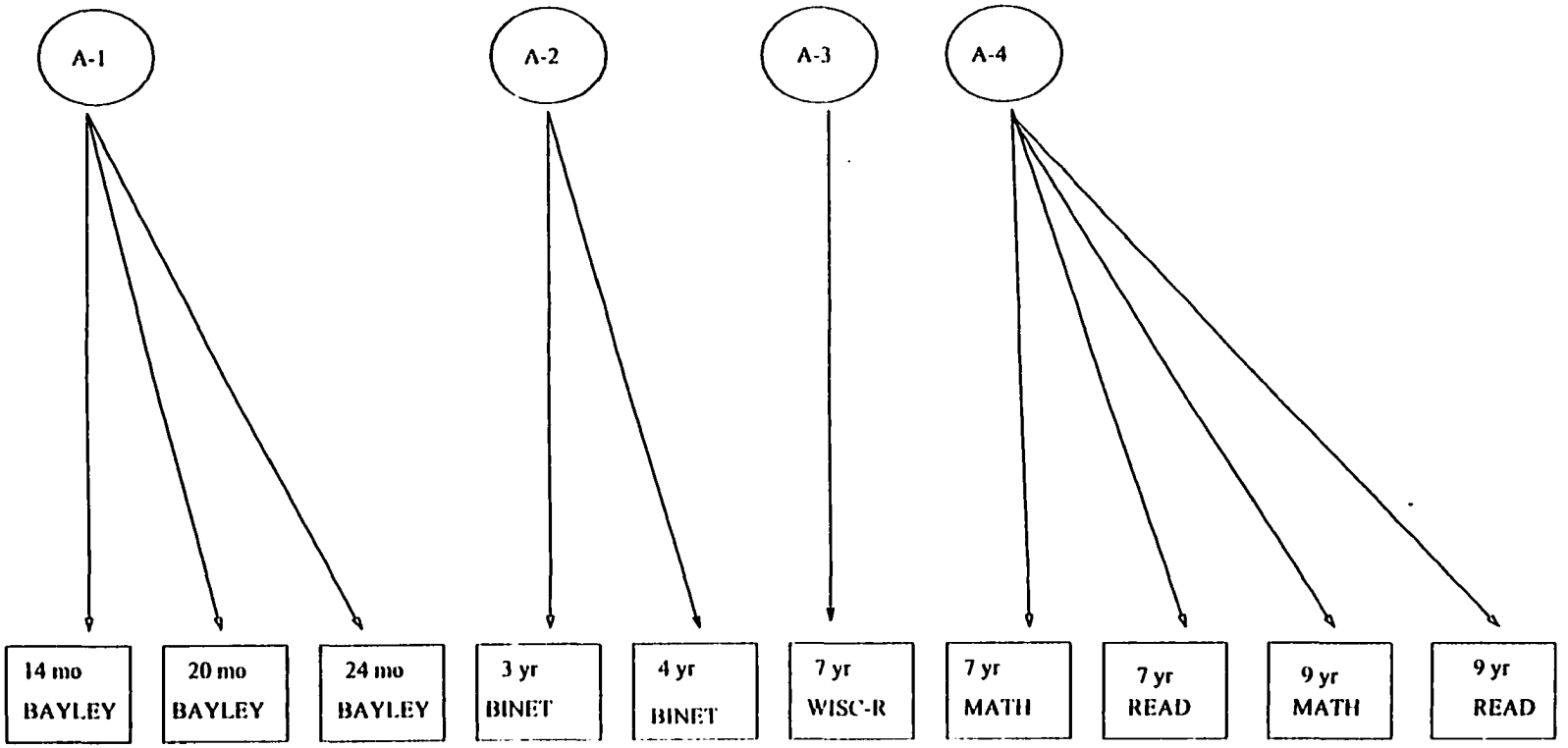


Figure 8

One genetic factor per measure.

From this point the parameter estimates were examined, and specific paths were constrained accordingly to obtain the most parsimonious model representing the additive genetic component. Model 9 constrains the additive genetic component to one factor loading on the IQ measures, age specific factors at 20 and 24 months, one factor loading on all measures from age 3 on, and one factor loading all measures at ages 7 and 9. This path diagram with the parameter estimates is shown in figure 9.

These finding indicate that there are three major genetic components influencing IQ and achievement, one present at birth, one coming in at age three, and one coming in at age 7. Comparing this to the full model results in a chi-square of 26.60 with 35 degrees of freedom and a p=.85 indicating a more parsimonious model. The path coefficients range from .22 to .59. This is the best fitting additive genetic model.

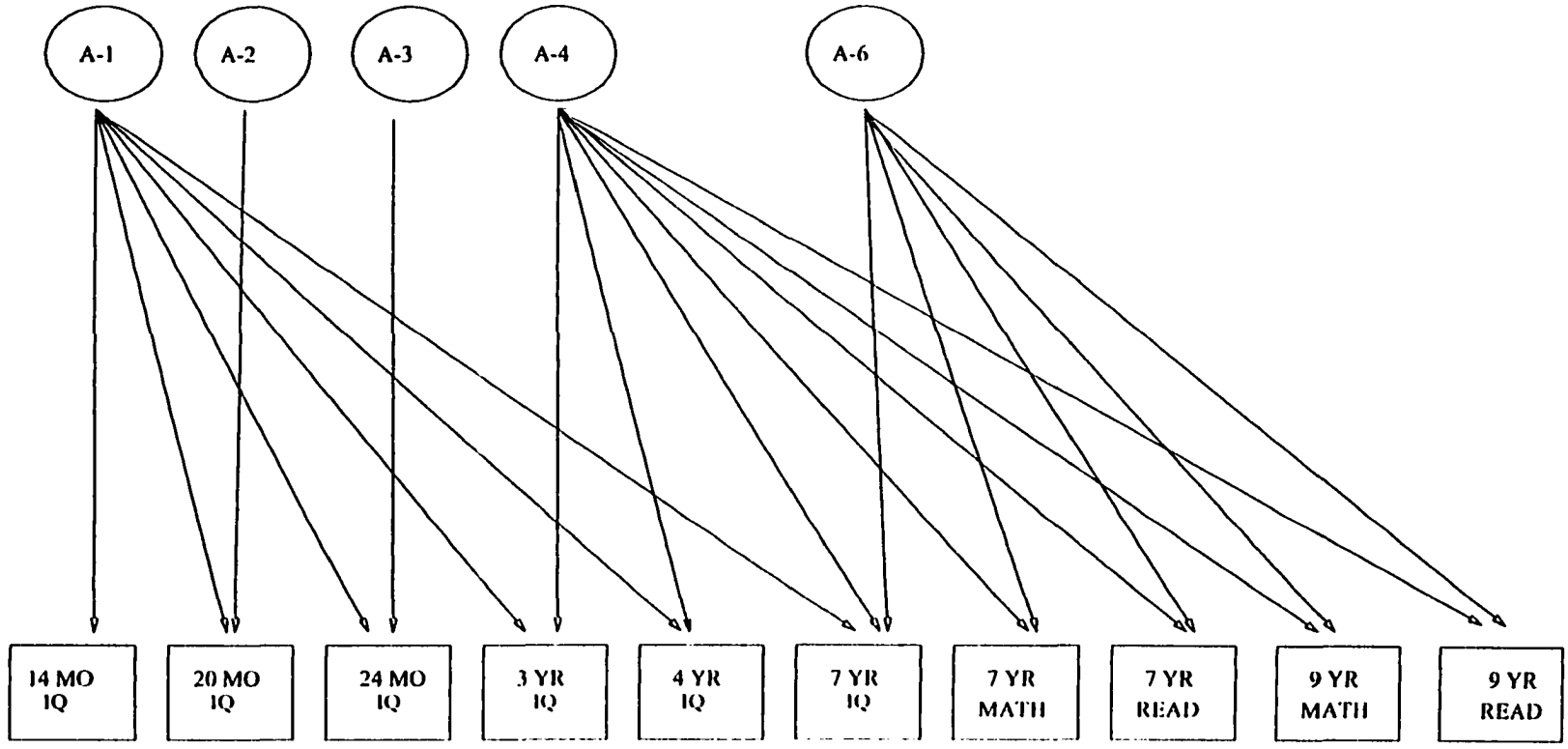


Figure 9

Most parsimonious additive genetic model for the longitudinal analysis.

Model 10 constrains the additive genetic component to the most parsimonious model and further constrains the common environmental component to one factor loading on all components as well as age/measurement specific components. The resulting chi-square from comparing this to model 9 indicates a more parsimonious model (p=.05). The common environmental components were further constrained to obtain the most parsimonious model (model 11) and is illustrated in Figure 10.

In this model, there is a common environmental component present at 14 months and 20 months that loads on all the measures. Two age specific factors come in, one at age 3 and one at age 4. Another common environmental factor comes in at age 7 and loads on all measures at ages 7 and 9. This model results in a chi-square of 18.22 with 28 degrees of freedom and a p=.92.

Model 12 constrains the path coefficients to represent the most parsimonious model for the additive genetic, common environmental, and individual environmental components. The individual environment is best represented by age/measurement specific components as well as a factor present at 24 months that affects math at age 9. The component coming in at age 7 effects both math and reading at that age and the component coming in at age 9 again affects both math and reading.

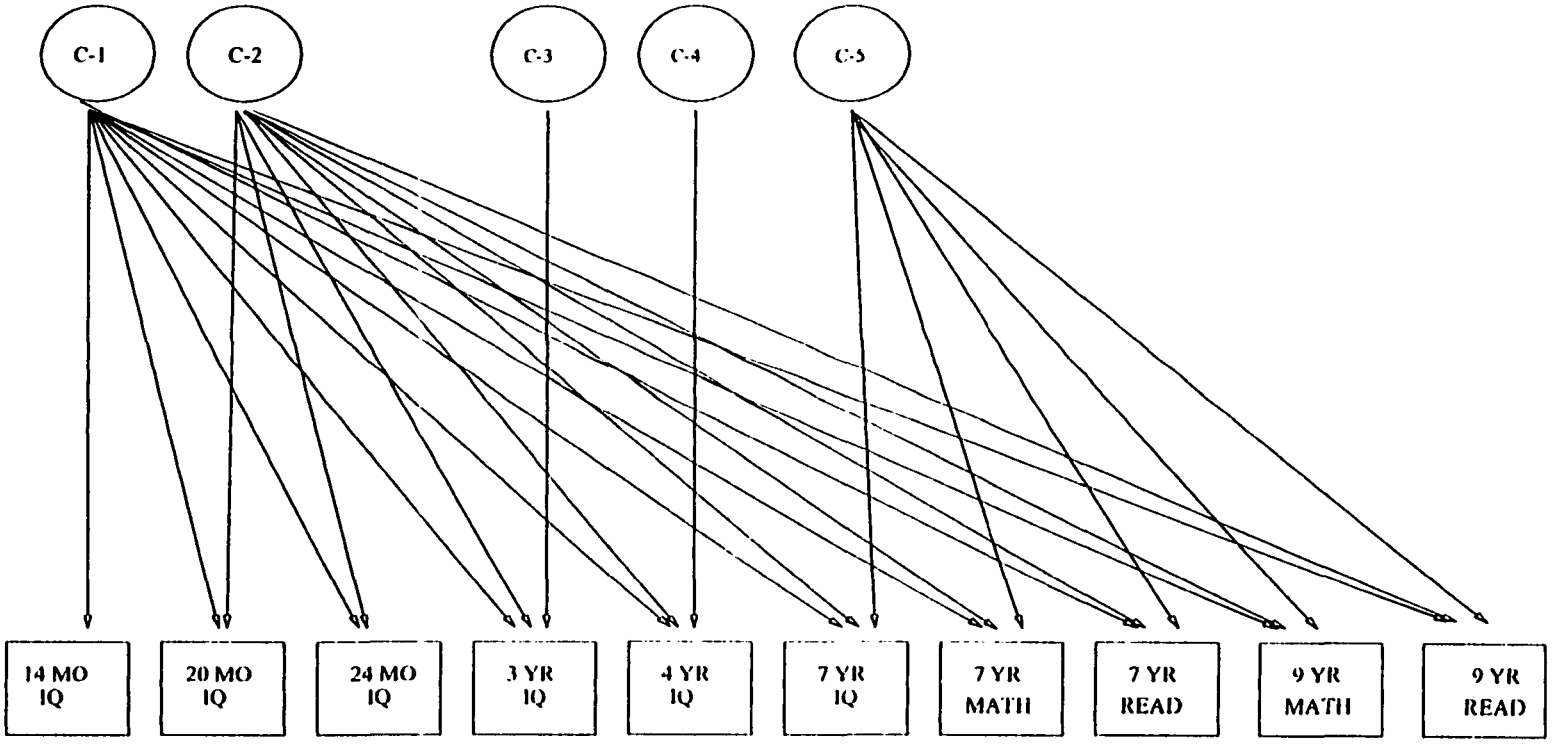


Figure 10

Most parsimonious common environmental model for the longitudinal analysis.

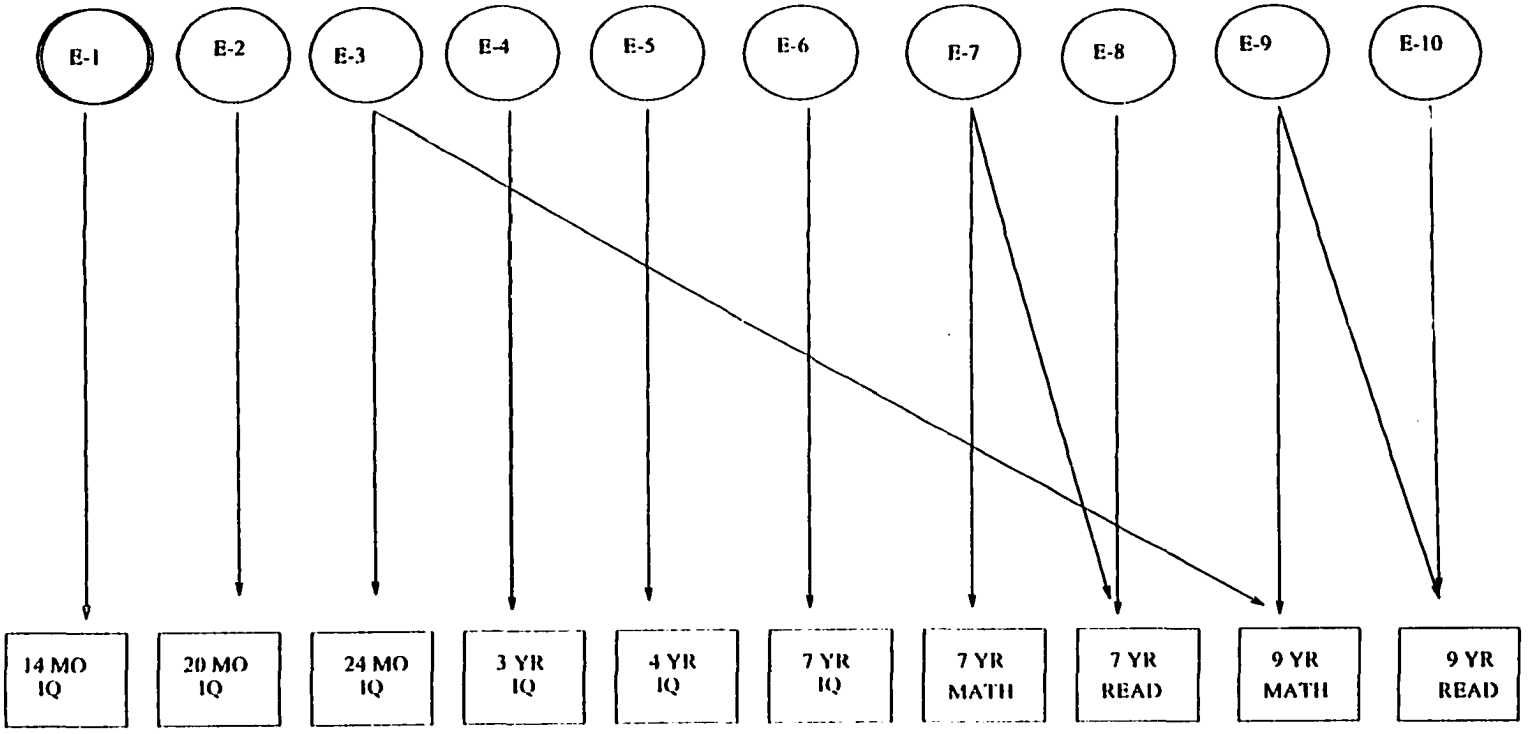


Figure 11

Most parsimonious individual environmental model for the longitudinal analysis

Using the results from the most parsimonious model, the parameter estimates for the additive genetic, common environmental, and individual environmental influences were estimated. The standardized additive genetic parameter estimates are shown in Table 17.

Table 17

Standardized Additive Genetic Parameter Estimates.

	A1	A2	A3	A4	A5
IQ 14 mo	.56				
IQ 20 mo	.40	.34			
IQ 24 mo	.47		.32		
IQ 3 yr	.51			.32	
IQ 4 yr	.44			.43	
IQ 7 yr	.26			.48	.33
MATH 7 yr				.34	.56
READ 7 yr				.47	.49
MATH 9 yr				.23	.52
READ 9 yr				.59	.26

There is an additive genetic component present at 14 months that influences IQ through age 7. The parameter estimates range from .26 to .56. There are age specific influences at 20 months (.34) and at 24 months (.32). Another genetic component comes in at age 3 and influences IQ and academic achievement from that time forward. The parameter estimates range from .23 to .59. A final genetic component comes in at age 7 and influences IQ and achievement at ages 7 and 9. The parameter estimates range from .26 to .56.

The standardized parameter estimates for the common environmental component is shown in table 18. The common environmental parameter estimates present at 14 months range from .09 to .51 and influence both IQ and academic achievement. Another component is present at 20 months and influences all of the variables from that time point on. The parameter estimates range from .28 to .64. Age specific influences are present at 3 years (.22) and at 4 years (.20). A new common environmental influence comes in at age 7 and affects IQ and achievement at age 7 and 9, ranging from .02 to .38.

Standardized individual environmental parameter estimates from the most parsimonious are shown in Table 19.

Table 18

Standardized Common Environmental Parameter Estimates for the Longitudinal.

	C1	C2	C3	C4	C5
IQ 14 mo	.51				
IQ 20 mo	.47	.56			
IQ 24 mo	.30	.64			
IQ 3 yr	.10	.59	.22		
IQ 4 yr	.09	.58		.20	
IQ 7 yr	.19	.49			.38
MATH 7 yr	.31	.28			.05
READ 7 yr	.28	.35			.30
MATH 9 yr	.14	.46			.02
READ 9 yr	.27	.41			.04

Table 18

Standardized Common Environmental Parameter Estimates for the Longitudinal.

	C1	C2	C3	C4	C5
IQ 14 mo	.51				
IQ 20 mo	.47	.56			
IQ 24 mo	.30	.64			
IQ 3 yr	.10	.59	.22		
IQ 4 yr	.09	.58		.20	
IQ 7 yr	.19	.49			.38
MATH 7 yr	.31	.28			.05
READ 7 yr	.28	.35			.30
MATH 9 yr	.14	.46			.02
READ 9 yr	.27	.41			.04

The individual environmental parameter estimates are primarily age specific and range from .14 to .63. The exceptions are: the component coming in at 24 months also influences math at age 9, the component coming in that influences math at age 7 also influences reading, and the component that influences math at age 9 also influences reading at that age.

Discussion

The purpose of the current analysis is to examine the etiology of IQ and academic achievement as well as the relationship between the two when academic achievement is measured by classroom performance. The etiology of how well a student performs in a classroom is of interest for many reasons. One important reason is that two of the primary considerations college admission boards take into account are how well a student performs on standardized tests (e.g. SAT, GRE) and how well they perform in school as indicated by their grade point average. The current analysis uses a teacher report that compares the individual to other students in the classroom on how well they perform in math and reading. This study hypothesized that the variance in IQ and academic achievement as assessed by teacher report will not be accounted for equally by genes and the environment. IQ is a more global measure than classroom

performance and may be more stable over the years. Two characteristics that may influence achievement are motivation and perseverance. To my knowledge there is not a way to measure these traits.

As found in previous research (Fulker, DeFries, & Plomin, 1988, Plomin & Petrel, 1997), the genetic analysis indicated that the strength of the genetic effect on IQ has a tendency to increase with age. Heritability accounted for 38% of the variance at age 14 months and 46% at age 7. Reading was found to be more heritable than IQ and math, accounting for 61% of the variance at age 7 and 55% of the variance at age 9. Using standardized tests, Wadsworth (1994) also found reading to be more heritable than math (36% and 12% respectively).

The effect of the common environmental influences are present at an early age and persist to later ages as opposed to genetic influences which have a tendency to "wash-out". Early genetic effects for IQ do not predict academic achievement, but early common environmental effects do continue to influence achievement. The common environmental effect is not likely to be due to phenotypic assortment because parents probably assort for adult IQ not infant IQ. Both genes and the common environment suggest a developmental phenomenon at age 7.

The individual environment is primarily age specific

and overall, accounts for more of the variation in achievement than in IQ with the exception of IQ at 14 months.

As with previous studies (Wadsworth et at., 1995, Gillis, DeFries, & Fulker, 1992, Brooks, Fulker, & DeFries, 1990), Thompson et al., 1991) a significant amount of the covariation between the measures can be accounted for by shared genetic effects. Genetic correlations among the IQ measures ranges from .35 to .91. The genetic correlation is also a function of the closeness in time of testing. The correlations are largest for adjacent testings, but decrease as the time between testing increases. This gives important evidence for developmental genetic phenomena in IQ.

Virtually all of the genetic effects influencing academic achievement are the same effects that influence IQ at ages 3 and 7. The genetic effects influencing infant IQ are not the same effects that influence academic achievement.

It should be noted that the new genetic components coming into the model correspond with changing the way we measure IQ. The new genetic effects at age 3 correspond to the change of measuring IQ from the Bayley to the Stanford Binet and the new genetic effects at age 7 correspond with using the WISC-R to measure IQ. However, the new genetic component coming in at 7 influences both

IQ and academic achievement. It may be possible that certain developmental stages exist at these age points and that is the reasons we use different tests to measure IQ at different ages. However, by changing the measures at specific times, we may be measuring different components of IQ.

The Bayley Mental Scale is designed to assess sensory-perceptual acuities and discriminations. It also assesses memory, problem-solving ability and the beginnings of verbal communication. It examines abstract thinking and the ability to form generalizations and classifications.

The Stanford Binet is designed to measure crystallized abilities, which includes verbal and quantitative reasoning. These assess ability in areas such as vocabulary and comprehension. It also measures fluid-analytic abilities, which includes abstract/visual reasoning. These abilities include items such as pattern analysis and matrices. The third item it is designed to measure is short term memory.

The WISC-R contains verbal and performance tests. The verbal tests include categories such as arithmetic, vocabulary, and comprehension, while the performance tests include picture arrangement, block design, and object assembly.

The Stanford Binet and WISC-R appear to have more in

common with each other and achievement than the Bayley does with any of the measures.

The differences in these test may explain why the genetic effects influencing infant IQ are not the same effects that influence academic achievement. However, this does not address the issue of specific developmental stages verses unique aspects of measurement in the tests. One would need to have a test that can be administered at age 2 and 3 as well as 4 and 7, to bridge the gaps between the current tests. It seems unlikely that a single test could be designed that could measure IQ at 14 months and still be effective at age 7.

If the results reflect true genetic changes, our findings support Piaget's periods of cognitive development (Berger, 1988). His theory states that there are four major stages of cognitive development and each one is age related. Infants think through their senses and motor abilities and learn things like objects still exist when out of sight (Sensorimotor stage). Preschool children, beginning at about age 3, use symbolic thinking as reflected in their ability to use language and to pretend (Preoperational stage). School age children, about age seven, begin to think logically and learn to understand the basic ideas of conservation, number, and classification (Concrete Operational stage). His last stage begins about age 12 when adolescents can think

hypothetically and abstractly (Formal Operational stage). While our data does not contain information on age 12, it would be interesting to see if another genetic component comes in at that time.

Future Directions

While this research did answer some questions regarding the etiology of IQ and academic achievement, many questions remain. As the sample grows older, it would be beneficial to add measurements of IQ and achievement at later ages. This could further examine the structure of the genetic and environmental components. We saw that the heritability of IQ has a tendency to increase with age, but did not have data on later ages of achievement to assess such a trend. It would be informative to look at the relationship between achievement at early ages and high school as well as how the relationship between IQ and achievement changes and stays constant during those years.

Modeling standardized tests such as the PIAT and Key Math in a similar way to which this analysis was modeled would supply more information regarding the differences and similarities in standardized tests verses teacher report.

It would also be interesting to look at specific abilities in a longitudinal design as opposed to "g". It

may be that the genetic and environmental contributions to verbal IQ and reading achievement, for example, differ from those in performance IQ and reading achievement.

Chapter IV

CAUSAL MODEL - IQ AND ACHIEVEMENT

Introduction

The previous analyses suggest that there are important genetic contributions that influence IQ and academic achievement. Most social science research, on the other hand, often recognize the fact that genes contribute to IQ and academic achievement, but fail to implement these genetic relationships in their mathematical models.

In social science, a typical way model the effects of IQ on achievement would be to put a causal arrow between the phenotype of IQ and the phenotype of academic achievement.

Recent advances in behavior genetic modeling (Heath et al., 1993; Carey & DiLalla, 1994) show how one can test models about phenotypic causality. In this chapter I begin an exploration of these models using the twin sample.

Methods

Subjects and measures

The subjects and measures for this analysis are

described in Chapter II.

Analysis

Path analysis has been explained in previous chapters and was used to examine the direct effect that IQ has on achievement. The path diagram is illustrated in Figure 12.

In this model the observed IQ scores of the twin pair (P_{IQ1} and P_{IQ2}) are loading on or influencing the observed achievement scores (P_{ACH1} and $P_{ACH}2$). The unobserved residual is represented by U_{ACH1} and U_{ACH2} and are also loading on achievement. The IQ scores are allowed to correlate and are represented by the double headed arrow with the path coefficient r_i . The lower case I represents MZ or DZ twins. The residuals are also allowed to correlate as represented the double headed arrow and the path coefficient "c". The subscript again represents MZ or DZ twins. These path model were run for both the math and reading variables.

Results

The results of the path models are shown in Figure 13 and 14.

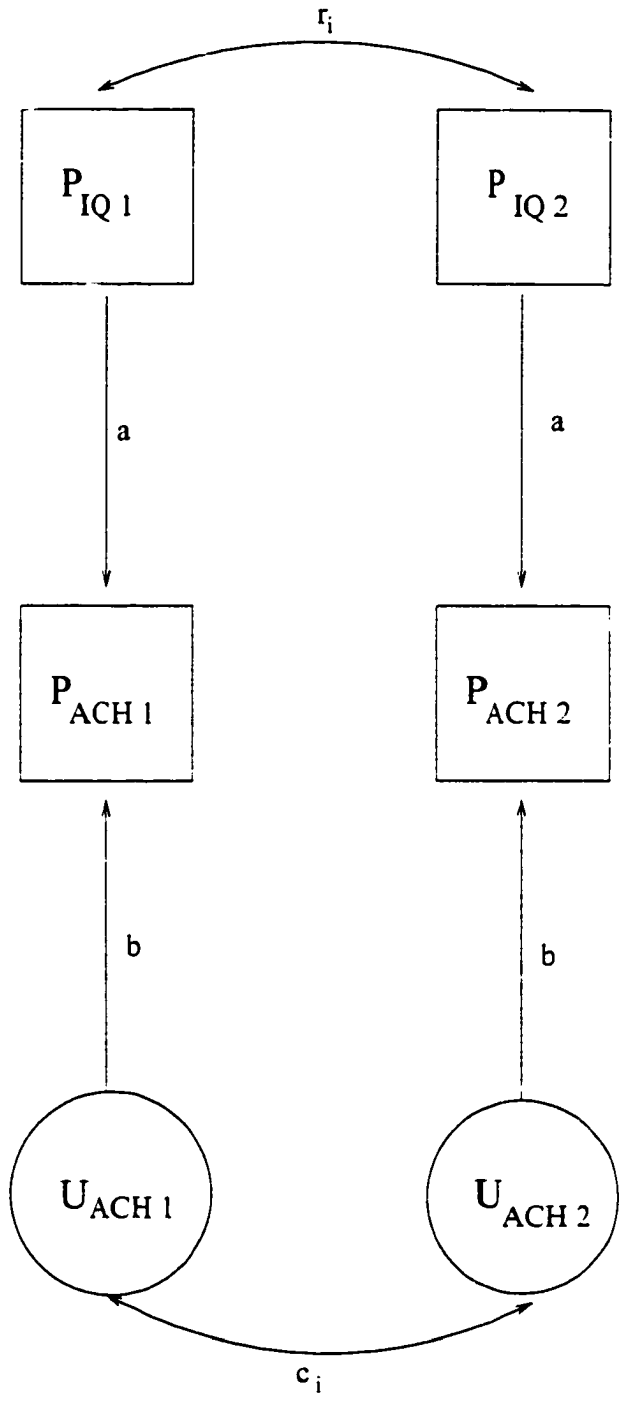


Figure 12

Path diagram for causal model.

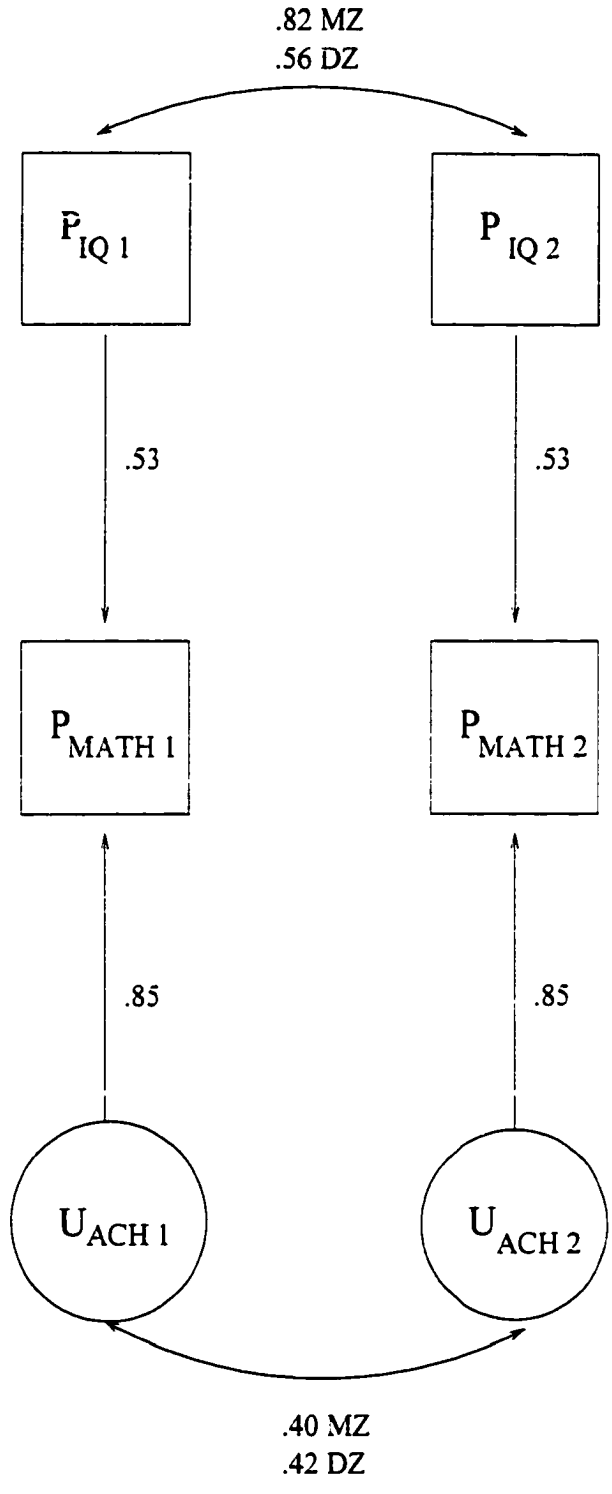


Figure 13

Results of math path diagram.

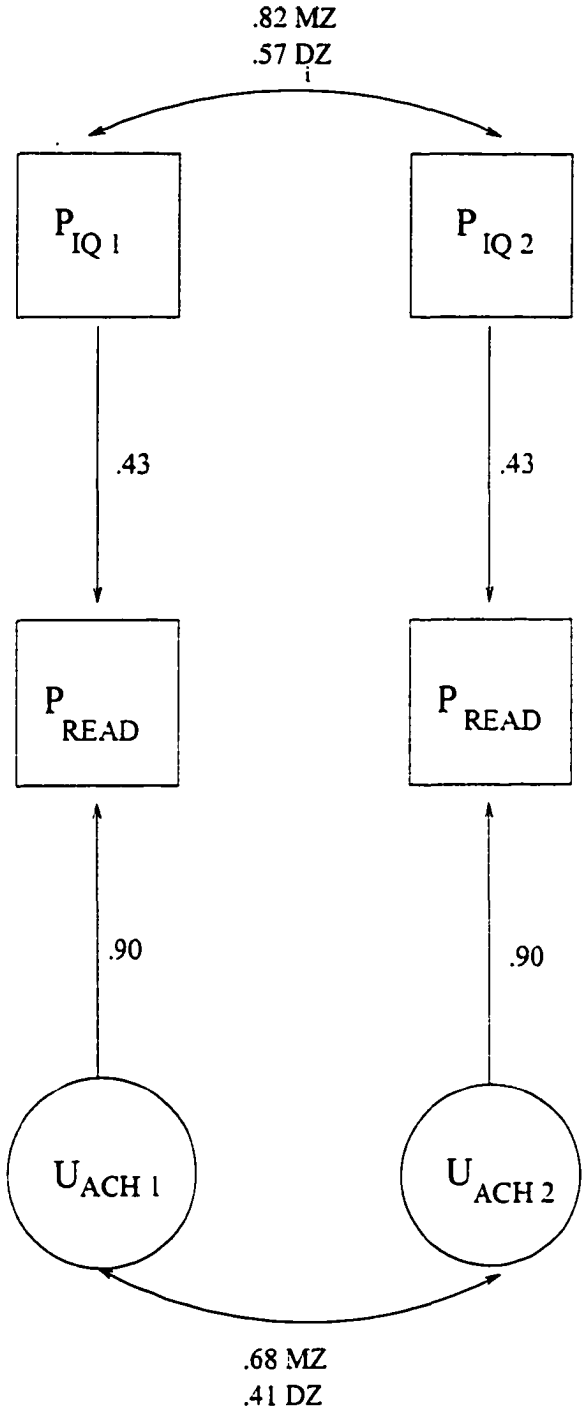


Figure 14

Results of reading path diagram.

In the general model, shown in Figure 15, the path coefficients or correlations are estimated. In other words, all possible correlations are estimated. The causal models are a subset of the general model. The -2xlog for the causal models minus the -2xlog for the general models were used to calculate a chi-square with corresponding degrees of freedom. The chi-square for the math achievement model is 7.41 with 2 df and a resulting p=.02. For the reading achievement, the chi-square = 29.13 with 2 df and a p<.01. The results indicate that these models do not fit the data and are rejected.

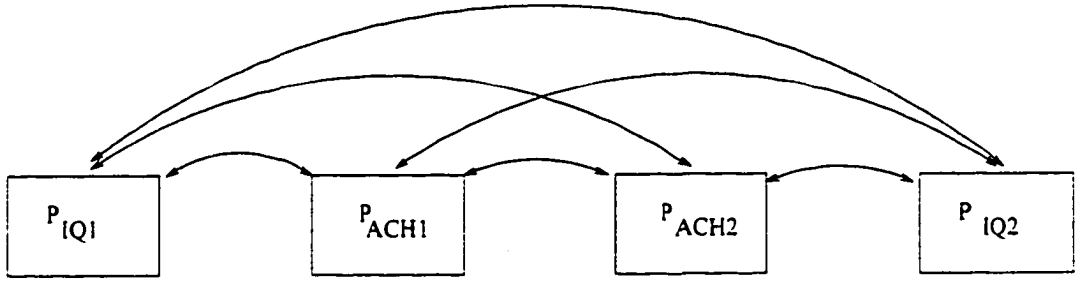
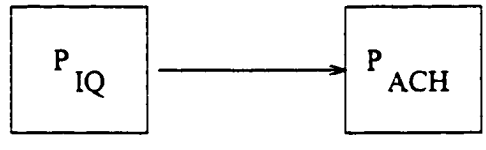


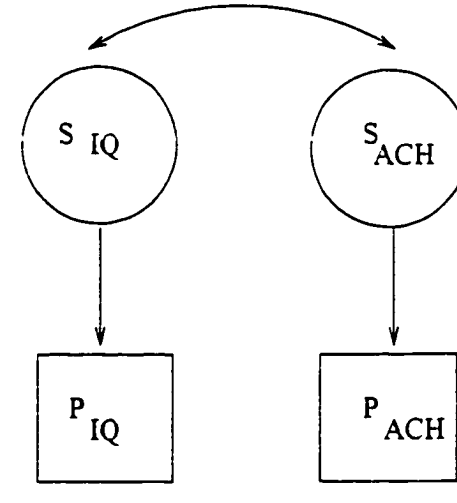
Figure 15.

Path diagram for the general model.

Discussion

In this analysis, using one phenotype to directly cause or influence another phenotype results in models that do not fit the data. Applying this model to twin data in this type of analysis is a new approach and needs to be replicated in other samples. If these finding are replicated, it could have profound implication for social science models that use phenotypic causation, as these models may be mis-specified. In other words, it may be incorrect to have one observed phenotype causing another observed phenotype. Instead, it may be more appropriate to have something else loading on the observed variables that is allowed to correlate. A simple diagram of this is shown below.





Again, this is a novel area of investigation that needs consideration.

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