

# Heritability of Cognitive Abilities in Adult Twins: Comparison of Minnesota and Swedish Data

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Cross-sectional reports suggest heritability of cognitive ability increases throughout adulthood. To investigate this hypothesis, quantitative genetic analyses were conducted on four measures of cognitive ability (verbal, spatial, perceptual speed, memory). Data from Minnesota and Swedish twin studies of aging were compared. Heritability estimates and the factor structure of cognitive abilities could be equated across younger twins (age, 27–50) and middle-aged twins (age, 50–65) from both studies, suggesting stability of heritability during adulthood. The heritability of 81% for a general cognitive factor confirmed earlier findings of high heritability in younger and middle-aged samples. Older Swedish twins (age, 65–85) demonstrated significantly lower heritability estimates for cognitive abilities (54%) and a significantly different factor structure of cognitive ability.

**KEY WORDS:** Cognitive abilities; twins; heritability; age differences; sample differences.

## INTRODUCTION

Heritability of cognitive abilities has been the focus of behavior genetic investigations for decades. Summaries of the world's existing IQ data converge on the conclusion that heritability for IQ is at least 0.50 (Bouchard and McGue, 1981; Chipuer *et al.*, 1990). The majority of that information, however, came from children and adolescents. A number of developmental behavior geneticists have suggested that heritability actually increases through adulthood (McCartney *et al.*, 1990; McGue *et al.*, 1993b; Plomin, 1986). It is not unlikely that heritability differs across the life span. For instance, it is possible that environmental influences

accumulate over the life span, resulting in a decrease late in the life span in the proportion of variance accounted for by genetic factors (Baltes *et al.*, 1980). It is equally possible that genetic effects existing early in development are amplified throughout the life span (Plomin, 1986), resulting in an increase in the amount of variance accounted for by genetic factors. A third possibility is that relationship between age and heritability of cognitive abilities is not linear; perhaps heritability increases during some portions of the life span and decreases during others.

Recent reports (McCartney *et al.*, 1990; McGue *et al.*, 1993b) have suggested that the relative influence of genetic factors on cognitive abilities may increase throughout adulthood. In a summary of existing twin data, McGue *et al.* (1993b) reported an increase in heritability from approximately 50% in childhood and adolescence to more than 80% in adulthood. McCartney *et al.* (1990) reported a positive correlation of at least 0.36 between age and heritability estimates for intelligence in their meta-analysis of twin data. In

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**Table I.** Sample Characteristics for Both the Minnesota and the Swedish Twin Studies (Reared Together Twins Only)

Variable	MTSADA			Swedish		
	Younger	Middle-aged	Older	Younger	Middle-aged	Older
MZ twin pairs	47	54	18	21	41	31
DZ twin pairs	23	34	15	30	32	51
Age range	27–49	50–64	65–88	27–49	50–64	65–85
Mean age (SD)	38.9 (4.4)	60.1 (3.9)	69.3 (5.8)	41.1 (5.9)	58.8 (4.3)	71.6 (4.8)

both studies adulthood was treated as a single entity, combining data across more than 60 years of the life span. They have, in effect, reported a linear relationship between age and heritability, defined either by two points or by a correlation coefficient. Contrary to common practice in gerontology, none of these studies has focused on nonlinear trends or the old-old. Given the ramifications of the hypothesized relationship between age and heritability, it is imperative that genetic influences on cognitive abilities be assessed at different points in adulthood to obtain a more accurate picture of the exact nature of the relationship.

Stability in the heritability of intellectual performance does not preclude changes in the structure of general cognitive ability. Previous quantitative genetic research has not focused on possible structural changes in cognitive abilities across the adult life span. Research on cognitive aging has demonstrated marked age differences in variables such as the rate of information processing (e.g., Cerella, 1990), memory (e.g., Craik and Jennings, 1992), and measures of fluid abilities (Horn, 1970), but less so for measures of crystallized abilities (Horn, 1970). As a result of these age changes, it is possible that the structure of a general cognitive ability factor may change dramatically with age, independent of any stability or change in heritability.

In the present investigation, univariate and multivariate quantitative genetic analyses were conducted using data from two samples of adult twins that were divided into three age ranges to investigate further the pattern of differences in the heritability and structure of cognitive abilities across age cohorts. The analyses differ from previous investigations of these data in two important

ways. First, the structure of cognitive abilities is explored using a common pathway quantitative model that allows for investigation of genetic and environmental influences at two levels. This model has not been previously applied to cognitive data from either study. Second, the adult life span is divided into three cohorts, as opposed to treating adulthood as one homogenous entity. Finally, this investigation marks an important collaboration between two large twin studies of aging. At all points along the life span, sample differences in the heritability and structure of cognitive abilities are examined.

## METHOD

### Subjects

The present sample consisted of same-sex twins from the Minnesota Twin Study of Adult Development and Aging (MTSADA; Finkel and McGue, 1993; McGue *et al.*, 1993a) and from the Swedish Adoption/Twin Study of Aging (SATSA; Pedersen *et al.*, 1984, 1991). Sample ascertainment and characteristics of both studies have been described previously (Finkel and McGue, 1993; Lykken *et al.*, 1990; Pedersen *et al.*, 1984, 1991). In brief, both the Minnesota and the Swedish samples were ascertained from birth records. Both samples can be divided into three age groups: younger subjects (age 27 to 50 years), middle-aged subjects (age 50 to 65 years), and older subjects (age 65 to 88 years); age and sample size information are reported in Table I.

Minnesota twins were assessed in their homes during two 2.5-h interviews, while middle-aged and older Swedish twins were assessed in a location

convenient to their homes in one 4-h interview. Younger Swedish twins were assessed at the Karolinska hospital (Bartfai *et al.*, 1991). A recent comparison of memory data from the two studies (Finkel *et al.*, 1995) has demonstrated that the samples are similar in age ranges and means, IQ scores, and ratios of monozygotic (MZ) to dizygotic (DZ) twins and males to females. MTSADA includes only twin pairs who were reared in the same household. Because one of the primary goals of this investigation was a comparison between the Minnesota and the Swedish samples, Swedish twins who were reared apart were not included in these analyses. In addition, as Block Design was added to the MTSADA protocol approximately 2 years after the study's inception, complete data were available for a smaller sample of older Minnesota twins than has been included in previous reports.

### Measures

As part of a large assessment battery, the MTSADA collects 10 of the 11 scales of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981). The SATSA includes a battery of several tests from various sources designed to parallel and augment the WAIS-R (Pedersen *et al.*, 1992). A subset of four tests that MTSADA and SATSA have in common was chosen to tap four major areas of cognitive ability (Pedersen *et al.*, 1992). The Information subscale includes questions of increasing difficulty concerning general information and is included as a measure of *verbal ability*. Block Design assesses reasoning and *spatial ability* by requiring subjects to arrange colored blocks to match provided patterns. Digit Symbol requires subjects to apply a number-symbol code as quickly as possible and thus measures *perceptual speed*. *Memory ability* is measured using the Digit Span task, requiring subjects to repeat increasingly lengthy lists of digits. Principal-components factor analyses of these four scales in each sample as a whole and within each age group produced a single factor in every case, accounting for from 82 to 97% of the total variance.

### Statistical Method

Quantitative genetic analysis involves apportioning the total variance in a trait into three sep-

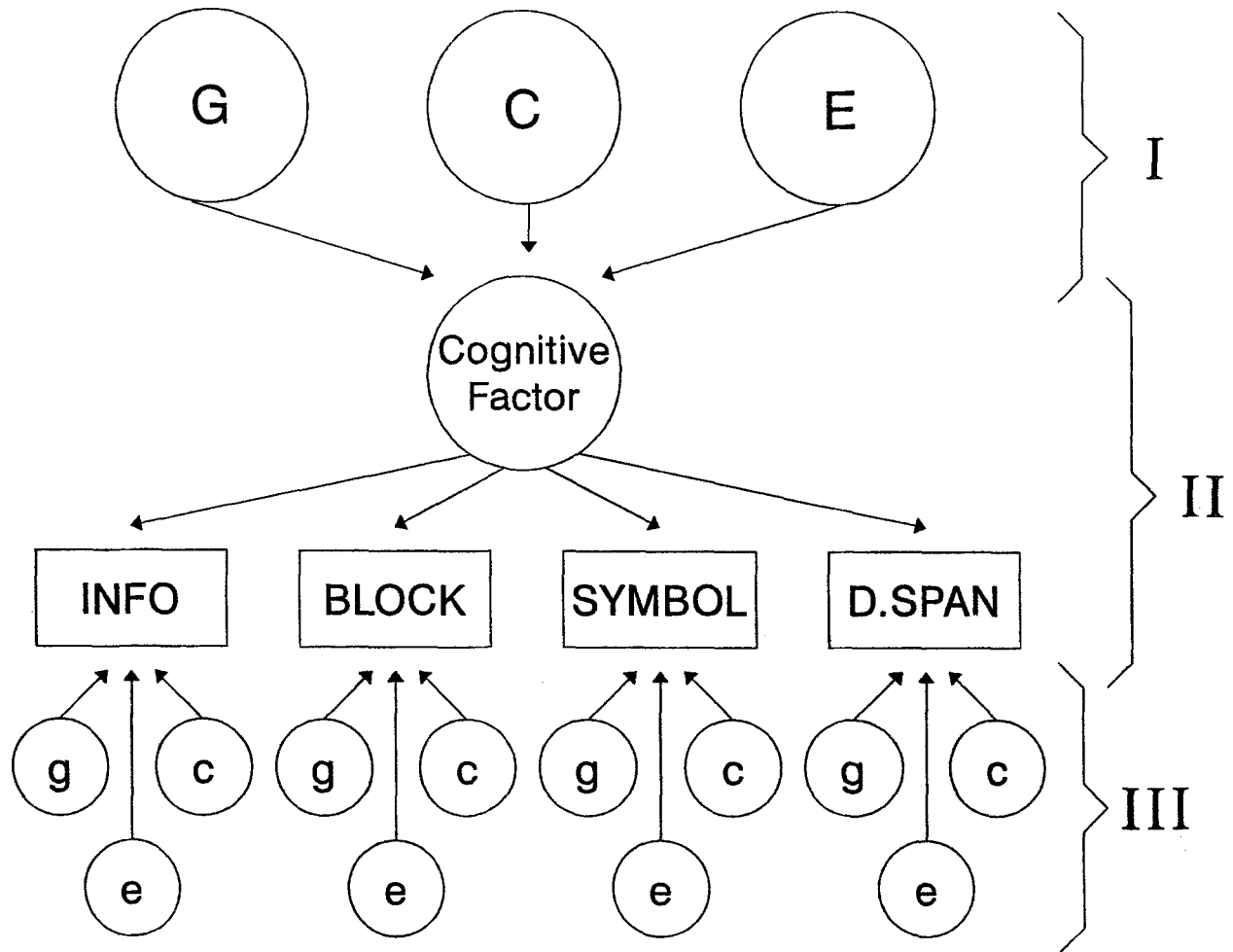
arate influences: additive genetic effects ( $G$ ); common environmental effects, i.e., influences shared by individuals reared in the same family ( $C$ ); and nonshared environmental effects and/or error ( $E$ ). The phenotypic correlation between two individuals ( $P_1$  and  $P_2$ ), assuming that the three components of variance are uncorrelated, can be expressed

$$\text{corr}(P_1, P_2) = h^2\text{corr}(G_1, G_2) + c^2\text{corr}(C_1, C_2)$$

where  $\text{corr}(G_1, G_2)$  is the additive genetic correlation for twins, which is 1.0 for MZ twins and 0.5 for DZ twins. Similarly,  $\text{corr}(C_1, C_2)$  is the correlation between shared environmental influences, which is 1.0 for all individuals reared in the same household. The three parameters  $h^2$ ,  $c^2$ , and  $e^2$  account for all portions of the phenotypic variance and consequently sum to 1. By analyzing the observed MZ and DZ covariance matrices, we can estimate the parameters  $h^2$ ,  $c^2$ , and  $e^2$ : the proportion of total phenotypic variance accounted for by the variance in genetic factors (i.e., heritability), shared environmental factors, and nonshared environmental factors, respectively.

The goals of this investigation were to apply a structural model that incorporated the phenotypic factor structure of the cognitive measures and to compare that model (1) across age groups within each study and (2) across studies. To this end, the common pathway model (Kendler *et al.*, 1987; Neale and Cardon, 1992) presented in Fig. 1 was fit to the data. There are three levels of interest in this path model. The phenotypic factor structure exists in the level labeled II in Fig. 1. Here the observed cognitive measures are combined to create a latent cognitive ability factor, and the path coefficients represent the factor loadings of each measure on the factor. Genetic and environmental influences on cognitive ability were assessed at two levels. First, general genetic and environmental influences were assessed at the level of the latent cognitive factor (level I in Fig. 1). Second, residual genetic and environmental influences were assessed at the level of the specific cognitive measures (level III in Fig. 1).

The three levels in the common pathway model allow for several comparisons both within and between studies. Across age groups within each study, three comparisons are possible: (1) comparison of the factor structure of cognitive abilities across age by equating the parameters at level



**Fig. 1** Common pathway model. Level I denotes the genetic (*G*), shared environmental (*C*), and nonshared environmental (*E*) influences on the cognitive ability factor. Level II denotes the factor structure of four cognitive measures combined into a single factor; the path coefficients represent factor loadings. Level III denotes the specific genetic (*g*), shared environmental (*c*), and nonshared environmental (*e*) influences on the individual cognitive tests, INFO, Information; BLOCK, Block Design; SYMBOL, Digit Symbol; D.SPAN, Digit Span.

II, (2) comparison of the general genetic and environmental influences on the latent cognitive factor across age by equating the parameters at level I, and (3) comparison of the specific genetic and environmental influences on the observed cognitive measures across age by equating the parameters at level III. Given that the cognitive measures used in each study are equivalent if not identical, it was possible to make the same three comparisons across studies.

MZ and DZ covariance matrices were computed and subjected to both univariate and multivariate model fitting procedures using LISREL VII

methods (Jöreskog and Sörbom, 1986). Tests of subsets of the structural model were conducted to determine whether the fit of the model to the data was significantly reduced when one or more of the parameters was removed from the model, when parameters were equated across age groups, or when parameters were equated across studies (Neale *et al.*, 1989). The best-fit models were chosen by minimizing Akaike's (1987) information criteria [AIC;  $\chi^2 - 2 * \text{degrees of freedom (df)}$ ] and by comparing Jöreskog's  $\chi^2/\text{df}$  (Jöreskog and Sörbom, 1979) as recommended by Loehlin (1987) when fitting many models with large degrees of

**Table II.** Intraclass Correlations for All Four Cognitive Measures, the Phenotypic Factor Generated in Each Nationality, and an Average Score Generated from the Four Cognitive Measures<sup>a</sup>

Variable	Younger		Middle-aged		Older	
	MZ <sup>b</sup>	DZ	MZ	DZ	MZ	DZ
<b>MTSADA</b>						
Information	.76	.56	.73	.43	.75	.12
Block Design	.75	.54	.73	.44	.85	.55
Digit Symbol	.73	.14	.55	.24	.60	.06
Digit Span	.52	.29	.53	.13	.76	.07
Phenotypic factor <sup>c</sup>	.80	.54	.80	.52	.86	.47
Average score <sup>d</sup>	.78	.41	.80	.56	.84	.19
<b>Swedish</b>						
Information	.65	.24	.88	.09	.64	.15
Block Design	.76	.29	.72	.34	.65	.33
Digit Symbol	.73	.19	.66	.38	.46	.28
Digit Span	.47	.11	.58	.06	.28	.12
Phenotypic factor	.83	.26	.79	.32	.55	.27
Average score	.74	.20	.84	.20	.59	.14

<sup>a</sup> Correlations have been corrected for age and sex within in each age group.

<sup>b</sup> Sample sizes are given in Table I.

<sup>c</sup> Phenotypic factor is the factor score resulting from a principal-components factor analysis of the four cognitive measures within each nationality (i.e., the factor structure is the same across age groups).

<sup>d</sup> Average score is the average of the four cognitive measures, with no differential weights on the basis of differential loading on the factor (i.e., identical across nationalities).

freedom. A value for Jöreskog's  $\chi^2/df$  of less than 2.0 indicates an adequate fit of the model to the data.

## RESULTS

Within age group and nationality, all measures were corrected for the effects of age and sex (McGue and Bouchard, 1984). Intraclass correlations for the four measures and the general cognitive ability factor are presented in Table II. The cognitive ability factor has the same structure across age groups but not quite the same factor loadings across nationalities. For this reason, correlations are also reported for a simple average of the four cognitive tests. Correlations are remarkably similar across age groups and nationalities, with the exception of older Swedish twins. MZ correlations for older Swedish twins are consistently smaller than all other MZ correlations listed in Ta-

ble II, whereas DZ correlations do not differ consistently across nationality or age group.

Univariate quantitative genetic analysis of the general cognitive factor including all age groups from both studies suggested that heritability estimates for all groups except older Swedish twins could be equated without significantly reducing model fit ( $\chi^2 = 5.8$ ,  $df = 14$ ,  $p = .97$ ). Equating parameters across all groups significantly reduced model fit ( $\chi^2 = 20.0$ ,  $df = 16$ , change in  $\chi^2 = 14.2$ ,  $p < .01$ ). Heritability of the cognitive factor was 81% for all MTSADA twins and for the younger and middle-aged Swedish twins but was only 54% for the older Swedish twins. Analogous results were found for the average score: heritability was 80% for all MTSADA twins and the younger and middle-aged Swedish twins and 56% for the older Swedish twins. The same pattern of equivalent heritabilities across all age groups and nationalities except older Swedish twins was found for Information, Digit Symbol, and Digit Span. Heritability estimates for Block Design could be equated across all groups without significantly reducing the model fit. Shared environmental influences were not significant.

Multivariate analyses served to support and augment the univariate results. The multiple comparisons made within the common pathway model in the MTSADA are summarized in Table III. Fitting the common pathway model in MTSADA without equating parameters across age groups resulted in an adequate  $\chi^2/df$  value (model 1, Table III); however, equating the parameters across age groups at all three levels in Fig. 1 provided a better fit of the model to the data (model 17, Table III; change in  $\chi^2 = 43.7$ ,  $df = 36$ , *ns*). In addition, dropping shared environmental influences further improved the model fit ( $\chi^2 = 289.2$ ,  $df = 203$ ,  $\chi^2/df = 1.42$ ,  $AIC = -116.8$ ). The final estimate of the path coefficient for the genetic loading on the cognitive factor was .98 in the MTSADA. The fact that the path coefficient was as near to unity as possible, given the upper limit of the reliability of the measures, suggests that the cognitive ability factor actually represents the genetic components of cognitive performance. It would be inappropriate to interpret the square of this path coefficient (95%) as the heritability of the cognitive factor; recall that univariate analyses demonstrated that the heritability of the factor was 81%.

Table III. Summary of Model-Fitting for MTSADA Twins<sup>a</sup>

Model	$\chi^2$	df	<i>p</i>	AIC	$\chi^2$ /df	Versus full model	
						$\chi^2$ change	df
1. Full	243.9	162	.00	-80.1	1.51		
Level I (general influences)							
2. Y=MA	247.7	164	.00	-80.3	1.17	3.8	2
3. Y=O	244.0	164	.00	-84.0	1.49	0.1	2
4. MA=O	247.6	164	.00	-80.4	1.51	3.7	2
5. Y=MA=O	248.4	166	.00	-83.6	1.50	4.5	4
Level II (factor structure)							
6. Y=MA	249.5	166	.00	-82.5	1.50	5.6	4
7. Y=O	245.3	166	.00	-86.7	1.48	1.4	4
8. MA=O	250.9	166	.00	-81.1	1.51	7.0	4
9. Y=MA=O	254.5	170	.00	-85.5	1.50	10.6	8
Level III (specific influences)							
10. Y=MA	256.4	174	.00	-91.6	1.47	12.5	12
11. Y=O	258.6	174	.00	-89.4	1.49	14.7	12
12. MA=O	262.4	174	.00	-85.6	1.51	18.5	12
13. Y=MA=O	273.0	186	.00	-99.0	1.47	29.1	24
All parameters							
14. Y=MA	267.2	180	.00	-92.8	1.48	23.3	18
15. Y=O	260.0	180	.00	-100.0	1.44	16.1	18
16. MA=O	270.6	180	.00	-89.4	1.50	26.7	18
17. Y=MA=O <sup>b</sup>	287.6	198	.00	-108.4	1.45	43.7	36

<sup>a</sup> Y, younger twins; MA, middle-aged twins; O, older twins. Levels I, II, and III refer to the levels in the quantitative model in Fig. 1.

<sup>b</sup> The model in which all parameters were equated across groups provided the best fit to the data.

The results of model-fitting in the Swedish data did not demonstrate the same uniformity across age groups (model-fitting summary is presented in Table IV). Fitting the common pathway model to each age group in the Swedish sample independently resulted in an adequate  $\chi^2$ /df value (model 1, Table IV); however, equating the parameters across age groups at all three levels in Fig. 1 resulted in a decreased fit of the model to the data (model 17, Table IV; change in  $\chi^2 = 58.3$ ,  $df = 36$ ,  $p < .01$ ). The best fit of the model to the data resulted when all parameters were equated across younger and middle-aged twins and specific influences (Level III) were equated across all age groups (model 18, Table IV). Again, the model fit improved when shared environment was deleted from the model ( $\chi^2 = 228.5$ ,  $df = 202$ ,  $\chi^2$ /df = 1.13, AIC = 175.5). The estimates of the path coefficients for the genetic loading on the cognitive factor were .94 for younger and middle-aged twins and .74 for older twins.

The second major goal of this investigation was to compare parameter estimates across studies to determine whether the Minnesota and Swedish twin data are in agreement. To that end, based on the results of the analyses within each study, four types of covariance matrices were included in a single common pathway model: (1) MTSADA younger and middle-aged, (2) MTSADA older, (3) younger and middle-aged Swedish, and (4) older Swedish twins. The multiple comparisons made in the common pathway model between Swedish and MTSADA twins are summarized in Table V.

A full model estimating separate parameters for each of the four groups provided an adequate fit to the data (model 1, Table V). Model fit was maximized when all parameters were equated across the groups from MTSADA (as expected from previous results), and parameters at level I and level III were equated across MTSADA and the younger and middle-aged Swedish twins (model 14, Table V). In other words, the pheno-

Table IV. Summary of Model-Fitting for Reared-Together Swedish Twins<sup>a</sup>

Model	$\chi^2$	df	<i>p</i>	AIC	$\chi^2/df$	Versus full model			
						$\chi^2$ change	df		
1. Full	191.8	162	.06	-132.2	1.18				
Level I (general influences)									
2. Y=MA	191.9	164	.07	-136.1	1.17	0.1	2		
3. Y=O	195.8	164	.05	-132.2	1.19	4.0	2		
4. MA=O	199.8	164	.03	-128.2	1.22	8.0	2	*	
5. Y=MA=O	201.5	166	.03	-130.5	1.21	9.7	4	*	
Level II (factor structure)									
6. Y=MA	195.9	166	.06	-136.1	1.18	4.1	4		
7. Y=O	208.1	166	.02	-123.9	1.25	16.3	4	**	
8. MA=O	218.1	166	.00	-113.9	1.31	26.3	4	**	
9. Y=MA=O	226.7	170	.00	-113.3	1.33	34.9	8	**	
Level III (specific influences)									
10. Y=MA	200.1	174	.09	-147.9	1.15	8.3	12		
11. Y=O	202.2	174	.07	-145.8	1.16	10.4	12		
12. MA=O	208.4	174	.04	-139.6	1.20	16.6	12		
13. Y=MA=O	215.3	186	.07	-156.7	1.16	23.5	24		
All parameters									
14. Y=MA	206.2	180	.09	-153.8	1.15	14.4	18		
15. Y=O	220.2	180	.02	-139.8	1.22	28.4	18	*	
16. MA=O	233.4	180	.00	-126.6	1.30	41.6	18	**	
17. Y=MA=O	250.1	198	.01	-145.9	1.26	58.3	36	**	
Best fit									
18. Level I: Y=MA Level II: Y=MA Level III: Y=MA=O	221.7	192	.07	-162.3	1.15	29.9	30		

<sup>a</sup> Y, younger twins; MA, middle-aged twins; O, older twins. Levels I, II, and III refer to the levels in the quantitative model in Fig. 1.

\* *p* < .05.

\*\* *p* < .01.

typic factor structure in Level II could not be equated across studies, nor could it be equated across age groups in the Swedish sample. The genetic and environmental influences on the factor and the individual measures could be equated across studies, with the exception of the older Swedish twins. Dropping shared environment from the model again improved the model fit ( $\chi^2 = 342.9$ , *df* = 259,  $\chi^2/df = 1.32$ , AIC = -175.1).

The factor loadings and variance components resulting from the best-fit model are presented in Table VI. The  $\beta$  matrix contains the factor loading of each cognitive measure on the cognitive factor (level II in Fig. 1). Although analogous, the factor loadings for MTSADA and the younger Swedish twins differed significantly (i.e., equating these parameters resulted in a reduction in model fit). For the older Swedish twins, however, the factor load-

ings suggest that in late adulthood, the structure of general cognitive ability is dominated by Digit Symbol, a measure of perceptual speed.

General heritability in Table VI denotes the portion of the heritability of the individual cognitive tests that arose from the general cognitive factor (levels I and II in Fig. 1), while specific heritability denotes residual genetic variance (i.e., not in common with the general cognitive factor, level III in Fig. 1). Heritability estimates for MTSADA did not differ significantly from the estimates for the younger and middle-aged Swedish twins, while the heritability estimates for older Swedish twins were significantly smaller. Contrary to expectations from reviews of earlier studies involving generally younger subjects, no increase in the heritability of cognitive ability was found in these samples. Data from MTSADA suggested sta-

**Table V.** Summary of Model-Fitting for Comparing Swedish and MTSADA Twins<sup>a</sup>

Model	$\chi^2$	df	<i>p</i>	AIC	$\chi^2/df$	Versus full model			
						$\chi^2$ change	df		
1. Full	307.4	216	.00	-124.6	1.42				
Levels I & III (general & specific influences)									
2. My=Sy & Mo=So	338.1	244	.00	-149.9	1.39	30.7	28		
3. My=Mo & Sy=So	349.3	244	.00	-138.7	1.43	41.9	28	*	
4. My=Mo	325.6	230	.00	-134.4	1.42	18.2	14		
5. My=Sy	322.8	230	.00	-137.2	1.40	15.4	14		
6. My=Mo=Sy	337.8	244	.00	-150.2	1.38	30.4	28		
7. Equate all groups	379.3	258	.00	-136.7	1.47	71.9	42	**	
Level II (factor structure)									
8. My=Sy & Mo=So	344.0	224	.00	-104.0	1.54	36.6	8	**	
9. My=Mo & Sy=So	344.2	224	.00	-103.8	1.54	36.8	8	**	
10. My=Mo	313.5	220	.00	-126.5	1.43	6.1	4		
11. My=Sy	324.1	220	.00	-115.9	1.47	16.7	4	**	
12. My=Mo=Sy	325.8	224	.00	-122.2	1.45	18.4	8	*	
13. Equate all groups	381.8	228	.00	-74.2	1.67	74.4	12	**	
Best fit									
14. Levels I & III: My=Mo=Sy									
Level II: My=Mo	339.8	248	.00	-156.2	1.37	32.4	32		

<sup>a</sup> My, younger and middle-aged MTSADA twins; Mo, older MTSADA twins; Sy, younger and middle-aged Swedish twins; So, older Swedish twins.

\* *p* < .05.

\*\* *p* < .01.

**Table VI.** Estimated Factor Loadings and Variance Components Resulting from the Best Fit of the Model in Fig. 1 to the Data from Younger, Middle-Aged, and Older Twins in the MTSADA and the Swedish Sample<sup>a</sup>

Variable	$\beta$ matrix	Heritability			Nonshared environment		
		Gen.	Sp.	Total	Gen.	Sp.	Total
MTSADA, all twins							
Information	.65	.39	.38	.77	.03	.20	.23
Block Design	.72	.49	.24	.73	.04	.23	.27
Digit Symbol	.50	.24	.38	.62	.02	.36	.38
Digit Span	.42	.16	.39	.55	.01	.44	.45
Swedish, younger and middle-aged twins							
Information	.60	.33	.42	.75	.03	.22	.25
Block Design	.74	.51	.23	.74	.04	.22	.26
Digit Symbol	.66	.41	.29	.70	.03	.27	.30
Digit Span	.54	.27	.33	.60	.02	.38	.40
Swedish, older twins							
Information	.59	.19	.43	.62	.15	.23	.38
Block Design	.74	.31	.32	.63	.25	.13	.37
Digit Symbol	.93	.49	.00	.49	.39	.12	.51
Digit Span	.31	.05	.28	.33	.04	.63	.67

<sup>a</sup> Beta matrix contains loadings of each test on the cognitive factor (level II in Fig. 1). Gen., general effects through the cognitive factor (levels I and II in Fig. 1); Sp., effects specific to the individual cognitive tests (level III in Fig. 1); total = Gen. + Sp.



bility across age cohorts, while Swedish data suggested stability and a possible decline in heritability in late adulthood.

To investigate further the discrepancy between MTSADA and Swedish parameters, univariate and multivariate analyses were replicated using the reared-apart twins from SATSA. The reared-apart sample includes 13 MZ pairs and 20 DZ pairs of younger twins, 21 MZ pairs and 52 DZ pairs of middle-aged twins, and 24 MZ pairs and 37 DZ pairs of older twins. Univariate analyses of the cognitive factor including all 12 groups of SATSA twins (including twins reared apart and twins reared together) indicated that estimating a separate heritability for older twins provided a significantly better fit ( $\chi^2 = 4.0$ ,  $df = 14$ ,  $p = .99$ ) than equating parameters across all groups ( $\chi^2 = 14.9$ ,  $df = 16$ , change in  $\chi^2 = 10.9$ ,  $df = 2$ ,  $p < .01$ ). Heritability was estimated at 58% for the combined sample of older Swedish twins and 81% for younger Swedish twins. Applying the common pathway model to reared-apart twin data indicated that the most parsimonious model that afforded adequate fit to the data estimated separate level I and level II parameters for older twins ( $\chi^2 = 271.1$ ,  $df = 192$ ,  $\chi^2/df = 1.41$ ,  $AIC = -112.9$ ), identical to the model providing the best fit for reared-together SATSA twins. Equating all parameters across age groups resulted in a significant reduction in fit ( $\chi^2 = 294.4$ ,  $df = 198$ , change in  $\chi^2 = 23.3$ ,  $df = 6$ ,  $p < .01$ ). Thus, both univariate and multivariate analyses were successfully replicated in the reared-apart Swedish twins.

Two differences between the older Minnesota subjects and the older Swedish subjects may account for the sample differences found in model-fitting. First, the older MTSADA sample was much smaller and, thus, may not have provided sufficient power to detect differences in heritability. Examination of the correlations and parameter estimates produced independently in each age group, however, did not indicate large, undetected differences between younger and older MTSADA twins. Second, the select sample of older MTSADA twins for whom complete data were available was significantly younger than the older Swedish sample. While 78% of the older MTSADA twins were between 65 and 70 years of age, only 47% of the older Swedish twins fell in that age range—the remaining 53% were older than 70 years. Thus, if heritability is less in older cohorts, the older

MTSADA twins included in this analysis may not have been old enough to manifest the effect.<sup>6</sup>

To test this hypothesis two additional analyses were completed: one in the MTSADA and one in the SATSA. First, univariate analyses were conducted on three of the cognitive measures in the full sample of MTSADA older twins, which matches the age distribution in the Swedish sample. While MZ intraclass correlations were similar to those reported in Table II, DZ correlations were higher (Information = .17, Digit Symbol = .46; Digit Span = .21), resulting in lower heritability estimates. Second, the older Swedish sample was divided into subjects below age 70 (21 MZ pairs and 30 DZ pairs) and those age 70 or above (10 MZ pairs and 21 DZ pairs). To maximize the stability and generalizability of the results, analysis focused on the average of Information, Digit Symbol, Block Design, and Digit Span. The intraclass correlations for the under-70 subset were .63 and .10 for MZ and DZ twins, respectively, and the heritability estimate resulting from univariate model-fitting was 55%. For the over-70 subset, the intraclass correlations were .42 and .10 for MZ and DZ twins, respectively, and the heritability estimate resulting from the univariate model was 38%. Although the heritability estimates were not significantly different (change in  $\chi^2$  as a result of equating variance components across the two groups = 1.19,  $df = 3$ ), the difference was in the expected direction. Together these analyses suggest that heritability of cognitive abilities may be lower after age 70; however, larger sample sizes will be needed to verify this result.

## DISCUSSION

Comparison of the heritability and structure of cognitive ability across age groups and nationalities allowed us to draw three conclusions. First, no differences were found across studies for estimates of genetic and environmental influences on cognitive measures and the general cognitive factor, at least for the younger and middle-aged subjects. Minor discrepancies were found in the structure of the cognitive factor in the same groups, which could easily have arisen from minor differences in ad-

<sup>6</sup> Schaie *et al.* (1992) reported relatively stable parent-offspring correlations for intellectual ability across adulthood, but again, only 28% of the parent sample was over age 70 at the latest time of testing.

ministration and scoring of the tests across studies. Estimates of heritability are population specific. Distinctions in gene pool or typical environmental influences across cultures could result in divergent estimates of the extent to which genetic variance accounts for phenotypic variance. Generalizing the results obtained from a single study to the population at large can be inappropriate. The agreement found between these two independent twin studies of aging suggests that the heritability estimate for the cognitive factor of 81% for younger and middle-aged adults is, in fact, robust.

Second, multivariate quantitative genetic analysis indicated that the structure of the cognitive factor in the older Swedish twins was significantly different from factor structure in the remaining sample. In the older twins, Digit Symbol defined the factor to such an extent that there was no residual genetic influence on Digit Symbol (see Table III). The Digit Symbol task assesses perceptual speed. Decades of research have suggested that behavioral slowing is a fundamental component of the aging process (Cerella, 1990) and is an integral part of cognitive aging, specifically (Salthouse, 1993). Likewise, quantitative genetic analysis demonstrated that perceptual speed becomes increasingly important in defining a factor comprised of the genetic portion of cognitive performance in older subjects. An analogous change in factor structure was not found in MTSADA; however, the Digit Symbol tasks are not identical in each study. WAIS-R Digit Symbol involves writing unusual symbols by an array of digits, while the Swedish Digit Symbol task involves writing known digits by an array of unusual symbols. This may seem a minor difference except to those who have completed both tasks. Because the skill has become automatic, it is much easier to write digits than it is to write novel symbols. For this reason, the Swedish task may be a more pure measure of perceptual speed, while the MTSADA task includes elements of both speed and working memory.

Finally, this investigation found marked stability in heritability estimates across most of the adult life span, while results from the older Swedish twins suggested that the heritability of cognitive ability may decrease in late adulthood. Combining results from this investigation and previous summaries of child and adolescent twins (Bouchard and McGue, 1981; Chipuer, *et al.*, 1990) suggests that the heritability of cognitive ability is characterized

by increases until adulthood and then relative stability. Genetic variance accounts for from 40 to 50% of the variance in intellectual performance during childhood and adolescence. The observed increase in heritability to as high as 81% in early and middle adulthood may reflect the amplification of genetic effects existing early in development (Plomin, 1986). In contrast to the increases suggested by McGue *et al.* (1993b) and McCartney *et al.* (1990), heritability appears to remain stable throughout most of the adult life span. Late in adulthood, perhaps after age 70, heritability estimates for cognitive ability demonstrate a decrease to as low as 54%. This change results not so much from a decrease in genetic variance as from a relative increase in environmental variance. For example, previous investigation of SATSA data (Harris *et al.*, 1992) demonstrated that the non-shared environmental variance for self-reported health increases dramatically in older adults while the genetic variance decreases, especially after age 70. Thus it is possible that the accumulated effect of nonshared environmental influences such as diet, exercise, and disease begin to take a toll on intellectual performance in late adulthood. Examination of even older cohorts and longitudinal research are necessary to verify the observed function, however, this investigation suggests that the nature of the relationship between age and the heritability of IQ is nonlinear, increasing from childhood to early adulthood, leveling off throughout middle age, and perhaps decreasing again in later life.

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