

Application of Hierarchical Genetic Models to Raven and WAIS Subtests: A Dutch Twin Study

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Hierarchical models of intelligence are highly informative and widely accepted. Application of these models to twin data, however, is sparse. This paper addresses the question of how a genetic hierarchical model fits the Wechsler Adult Intelligence Scale (WAIS) subtests and the Raven Standard Progressive test score, collected in 194 18-year-old Dutch twin pairs. We investigated whether first-order group factors possess genetic and environmental variance independent of the higher-order general factor and whether the hierarchical structure is significant for all sources of variance. A hierarchical model with the 3 Cohen group-factors (verbal comprehension, perceptual organisation and freedom-from-distractibility) and a higher-order *g* factor showed the best fit to the phenotypic data and to additive genetic influences (A), whereas the unique environmental source of variance (E) could be modeled by a single general factor and specifics. There was no evidence for common environmental influences. The covariation among the WAIS group factors and the covariation between the group factors and the Raven is predominantly influenced by a second-order genetic factor and strongly support the notion of a biological basis of *g*.

KEY WORDS: Hierarchical genetic models; Confirmatory Factor Analysis; IQ; *g*; twins.

INTRODUCTION

The relationships between dimensions of cognitive functioning such as verbal and perceptual processing and memory capacity have been a major focus of interest in intelligence research. Methodological advances in factor-analytic techniques, in which from a matrix of second-order moments a general second-order (*g*) factor and first-order group factors can be extracted, have led to the development of a hierarchical concept of intelligence. Hierarchical models of cognitive ability proposed by Burt (1949), Horn and Cattell (1966),

and Vernon (1950), for example, are now testable and widely accepted.

The first analyses of the factorial structure of the Wechsler Adult Intelligence Scale (Cohen, 1957) yielded a strong general second-order factor and three first-order factors that since have become known as the *Cohen Factors*: verbal comprehension (VC), perceptual organisation (PO) and freedom from distractibility (FD). Factor analyses of the Wechsler Intelligence Scale for Children (WISC) standardization data yielded three similar factors (Cohen, 1959), later replicated by Kaufman (1975). The combination of the various verbal and nonverbal tests to obtain a full-scale IQ was supported by the identification of a large general factor.

Most phenotypic factor analytic studies of multiple cognitive ability tests have arrived at factor patterns reflecting general skills operating across diverse cognitive tasks, and specific abilities operating within distinct cognitive domains. However, the phenotypic

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grouping of specific intellectual abilities contains no information on the nature of these associations. With multivariate genetic analyses it is possible to analyze the sources of variation that underlie and differentiate the different factors comprised by typical subtest loadings, hence, the different aspects of intelligence. Analyses of this kind determine the extent to which phenotypic relationships among different subtests result from common genetic and common environmental influences.

Multivariate genetic analyses of the WAIS-R and WISC-R factorial structure have been conducted in a limited number of studies. For the WAIS, a model with Cohen factors for the genetic and environmental structures in addition to one general genetic factor and specific within-family environmental influences was reported by Tambs, Sundet, and Magnus (1986). It was shown that the major part of the covariance between the phenotypic VC, FD, and PO factors was due to genetic effects. Shared environmental influences were only of some importance for PO. In contrast, multivariate genetic analyses of the WISC-R, conducted in reading-disabled and matched control twin pairs, showed the phenotypic factor structure (VC, PO, and FD) to be caused by both genetic and shared environmental influences (Casto, DeFries, and Fulker, 1995).

None of these studies explicitly tested a higher-order factor that accounts for the covariation between the group factors. With ordinary independent pathway or factor models the genetic and environmental covariances are accounted for by common factors to different variables which themselves may be correlated. With hierarchical models it can be tested whether group factors possess genetic and environmental variance independent of the second-order general factor and, conversely, whether the second-order factor is significant for all sources of variance. Moreover, we can quantify the effect of the higher-order factor independent of the effect of the group factors. The definition of general intelligence, g , corresponds with this higher-order factor; thus, hierarchical factor models can provide important information on the structure of intelligence.

General intelligence can be seen as either a unitary (molar) cognitive process in which the extent to which different tests correlate is reflected by the extent to which they overlap on this common process, or as a collective action of a set of independent cognitive processes that have all been brought to bear on a complex cognitive problem (i.e., g is componential or modular). Petrill (1997) discussed how hierarchical genetic models might help resolve the molarity-versus-modu-

larity debate in intelligence research. The molarity explanation would best fit the data if the same genetic factors were found to influence different dimensions of cognitive abilities. If cognitive dimensions were influenced by different genetic factors, then a modular explanation would best fit.

In the Colorado Adoption Project, hierarchical models have been applied to specific cognitive ability test batteries, including the WISC (Cardon *et al.*, 1992). Apart from a significant second-order general genetic factor, genetic influences were found to operate on group factors as well. The application of hierarchical models to twin data is sparse. In the Western Reserve Twin Project, phenotypic and genetic hierarchical factor models were applied to both the WISC-R and a specific cognitive ability battery (Luo, Petrill, and Thompson, 1994). Both general genetic and genetic group factors were significant for the two batteries. General shared environmental and general genetic influences were equally important for the WISC-R, but genetic factors were observed to be more influential in structuring individual differences in specific cognitive abilities than were shared environmental influences. Petrill, Luo, Thompson, and Detterman (1996) found significant phenotypic group factors (VC, PO, FD, and Speed) in addition to a second-order general factor in children, and, therefore, a modular system of intelligence was concluded to best fit the data. The hierarchical structure was significant for the genetic source of variance, but not for shared and unique environmental influences, which could be explained by one general factor and specifics.

The contribution of hierarchical genetic models to the understanding of the nature and structure of intelligence can be summarised as follows: Phenotypic g is strongly related to genetic g , which supports the view that general intelligence is a strong indicator of common genetic variance among cognitive tests (Luo, Petrill, and Thompson, 1994); There is genetic variance specific to distinct first-order cognitive dimensions, including reaction-time tasks (Petrill *et al.*, 1996) in addition to general genetic variance common to all tests; thus, genetic covariance among cognitive ability tests (i.e., genetic g) is not unitary.

In the present study, subtest scores of the Dutch version of the Wechsler Adult Intelligence Scale (WAIS) and the Raven Progressive Matrices were analysed for 194 adolescent Dutch twin pairs. The aim of this paper is (1) to test the fit of a phenotypic hierarchical factor model underlying the subtest scores of the WAIS; (2) to test how this hierarchical model fits

a genetic model: whether first-order group factors possess genetic and environmental variance independent of the second-order general factor and whether the second-order factor is significant for all sources of variance (A, C and E); and (3) to explore the nature of the covariance of Raven Standard Progressive test scores of the same sample (Rijsdijk, Boomsma, and Vernon, 1995) with the WAIS IQ tests. The Raven is claimed to be a nonverbal test of reasoning and a good measure of *g*, which has very low loadings on other factors (Jensen, 1998).

SUBJECTS AND METHOD

Subjects were 194 Dutch twin pairs who participated in a longitudinal project that investigated variation in peripheral nerve conduction velocity, reaction times, and intelligence (Rijsdijk *et al.*, 1995, Rijsdijk and Boomsma, 1997, Rijsdijk, Vernon, and Boomsma, 1998) and genetic and environmental influences on brain development (Van Beijsterveldt *et al.*, 1995, 1996).

The Raven Standard Progressive Matrices test scores were obtained at the first visit of the twins to the laboratory (mean age, 16.13; SD, .56). The Raven score is simply the number of correct answers (without a time limit). The Dutch version of the Wechsler Intelligence Scale was individually administered (Stinissen, Williams, Coetsier, and Hulsman, 1970) on the second visit, 1 1/2 years later (mean age, 17.6; SD, .54). The scoring table applied was based on the joint normative samples of males and females between age 15 and 18 (age 18 meaning 18 years, 11 months and 31 days). Mean age was equal for males and females. IQ data were available for 37 MZM, 31 DZM, 46 MZF, 36 DZF and 44 DOS twin pairs.

For 117 same-sex twin pairs, zygosity was determined by blood group and DNA typing and for the others by using items from a questionnaire concerning physical similarity and the frequency with which family members and strangers confuse the twins. For the blood and DNA typed group, questionnaire data were available for 85 pairs. The percentage correctly classified zygosity based on the questionnaire information compared with blood group polymorphisms and DNA was 95%.

Phenotypic Analyses

Kaufman factor scores were computed by summing subtest scores that load on the different factors:

VC (subtests 1, 2, 4, 6); FD (subtests 3, 5, 7); and PO (subtests 8–11). Sex differences in means for the subtest scores, Kaufman factors, verbal, performance, full-scale IQs and Raven scores were assessed by likelihood-ratio χ^2 tests using Mx (Neale, 1997). These tests compare the fit of a model that constrained parameter estimates for mean scores to be equal across sexes to one that allowed them to vary in males and females, while taking into account the dependency that exists between observation from twins (Boomsma *et al.*, 1993).

Sex differences in phenotypic correlations among the WAIS subtests and the Raven were also analysed in Mx. To the variance-covariance matrices of males and females a model was fitted in which maximum-likelihood correlations, as well as the standard deviations, were obtained. First, the standard deviations were tested for sex differences by comparing the fit of models that constrained standard deviations to be equal across groups with models in which they are allowed to vary. With the same strategy, correlations were tested for sex differences at the next step. Significance of correlations was tested by evaluating the significance of χ^2 changes of models in which correlations were constrained to be zero.

Confirmatory phenotypic factors analysis was conducted on the variance-covariance matrix of the whole sample. Models were fitted in which the phenotypic variance and covariance was accounted for by a specified number of group factors and also by specific factors accounting for the variance unique to each subtest. Significance of alternative phenotypic factor models was compared by χ^2 tests.

Univariate Genetic Analyses

Univariate genetic model fitting was carried out on variance-covariance matrices from the 5 sex-by-zygosity groups. Sources of phenotypic variation considered were *A*, additive genetic variation, (i.e., the sum of the average effects of the individual alleles at all loci); *D*, dominance genetic influences that represent interactions between alleles at the same locus; *C*, common or shared environmental variation; and *E*, a random environmental deviation that is not shared by family members. Confidence intervals for heritability estimates were computed in Mx (Neale and Miller, 1997). All WAIS subtest scores, Kaufman factors, verbal, performance, full-scale WAIS IQ, and Raven scores were tested for sex-differences in genetic architecture.

Multivariate Genetic Analyses

Because no major sex-differences in genetic architecture were observed, zygosity groups were pooled across sexes and multivariate analyses were conducted on the phenotypic mean-squares-between pairs (MSB) and mean-squares-within pairs (MSW) matrices. These matrices can be estimated by, for example, MANOVA. The limitation of this method is the inability to test sex differences. The advantage is the use of $v \times v$ input matrices, instead of $2v \times 2v$ covariance matrices, which is of greater importance when the numbers of variables (v) is large (Martin and Eaves, 1977); Boomsma and Molenaar, 1986).

The MSW estimate the within-pairs covariance σ_w^2 . The expectation for MSB is twice the between-pair variance plus the within-pairs variance ($2\sigma_B^2 + \sigma_w^2$). Expected MSB and MSW pair matrices for MZ and DZ twin pairs can be expressed as a function of genetic and environmental factors that effect the phenotypic covariance. Because MZ twins are genetically identical, genetic factors do not contribute to phenotypic differences between members of a pair. The MZ within-pair variance thus reflects only unique environmental differences ($\sigma_w^2 = \mathbf{E}$). Influences that cause differences between MZ twin pairs (and make members of one pair more alike) are the genetic and common environmental factors ($\sigma_B^2 = \mathbf{A} + \mathbf{C}$). As DZ twins share half of their genes on average, DZ within-pair covariances not only reflect differences in unique environment but differences caused by a different genotype as well ($\sigma_w^2 = 0.5 \mathbf{A} + \mathbf{E}$). Influences that cause differences between DZ twin pairs are also the additive genetic and common environmental influences ($\sigma_B^2 = 0.5 \mathbf{A} + \mathbf{C}$). These derivations apply to all genetic and environmental factors, that is, for factors \mathbf{A} , \mathbf{C} , and \mathbf{E} and specific factors \mathbf{A}_{SP} , \mathbf{C}_{SP} , \mathbf{E}_{SP} . The expected MSW and MSB for MZ and DZ pairs based on the expected variance components are:

$$\Sigma MSB_{mz} = 2(\mathbf{A} + \mathbf{C}) + \mathbf{E} + [2(\mathbf{A}_{sp} + \mathbf{C}_{sp}) + \mathbf{E}_{sp}] \quad (1)$$

$$\Sigma MSW_{mz} = \mathbf{E} + [\mathbf{E}_{sp}] \quad (2)$$

$$\Sigma MSB_{dz} = 1\frac{1}{2}\mathbf{A} + 2\mathbf{C} + \mathbf{E} + [1\frac{1}{2}\mathbf{A}_{sp} + 2\mathbf{C}_{sp} + \mathbf{E}_{sp}] \quad (3)$$

$$\Sigma MSW_{dz} = \frac{1}{2}\mathbf{A} + \mathbf{E} + [\frac{1}{2}\mathbf{A}_{sp} + \mathbf{E}_{sp}] \quad (4)$$

The observed mean-square matrices (Σ) are full and the specific matrices (\mathbf{A}_{SP} , \mathbf{C}_{SP} , \mathbf{E}_{SP}) diagonal, both of dimension $v \times v$. The dimension and type of the factors \mathbf{A} , \mathbf{C} , and \mathbf{E} depend on the model. Initially, a

Cholesky decomposition was specified in which \mathbf{A} , \mathbf{C} , and \mathbf{E} are lower matrices of dimension $v \times v$. In the hierarchical model, \mathbf{A} and \mathbf{E} are both comprised of four matrices according to the Schmid-Leiman **transformation**. In Mx notation this **transformation** is specified as: $\mathbf{X} * (\mathbf{Y}|\mathbf{Z}) * (\mathbf{X} * (\mathbf{Y}|\mathbf{Z}))' + \mathbf{R} * \mathbf{R}'$, where \mathbf{X} is a full $v \times f$ matrix, and f is the number of first-order group factors (i.e., three for the Cohen factorial model); \mathbf{Y} is a full $f \times 1$ matrix (one second-order, general factor influencing the f group factors); \mathbf{Z} is an $f \times f$ identity matrix in order to fix the residual first-order factor variances to unity; and \mathbf{R} is a $v \times v$ diagonal matrix in which subtest specific effects are estimated (first-order error variances). The standardised solution of the factor structure will yield a $v \times (1 + f)$ matrix, with the first column representing the standardised general factor loadings and the following f columns representing the standardised group factor loadings (see for details example script at web site <http://statgen.iop.kcl.ac.uk>). In the final analysis the Raven test score was included in the best-fitting hierarchical model to explore the nature of the common variance with the WAIS subtests. To identify this model the specific genetic (\mathbf{A}_{SP}) parameter was omitted (Fig. 1).

RESULTS

Phenotypic Analyses

Sex differences were observed for arithmetic and picture completion, for which males had a significantly higher score than females (6.98 versus 6.51 and 6.38 versus 5.95, respectively); and for coding, for which females had a significantly higher score than males (7.62 versus 6.78). However, these sex differences were small and did not weigh heavily on the total subtest scores, which is reflected in the equality of means across sexes for the verbal, performance, and full-scale IQ scores (VIQ, PIQ, and FSIQ) (Table I).

The sample's mean WAIS full-scale IQ of 113.5 was higher, and the standard deviation of 11.8 was lower than the theoretical parameters ($M = 100$; $SD = 15$). A validation study ($N = 601$) of four subtests of the Dutch version of the WAIS (Mulder, Decker, and Decker, 1995) showed that scores on all four tests were higher than the scores of the Dutch normative sample (Stinissen *et al.*, 1970). Bouma, Mulder, and Lindboom, (1996) suggested that this observation might be a consequence of increasing population IQ and that WAIS IQ scores based on the 1970 norms may, in fact, be somewhat overestimated.

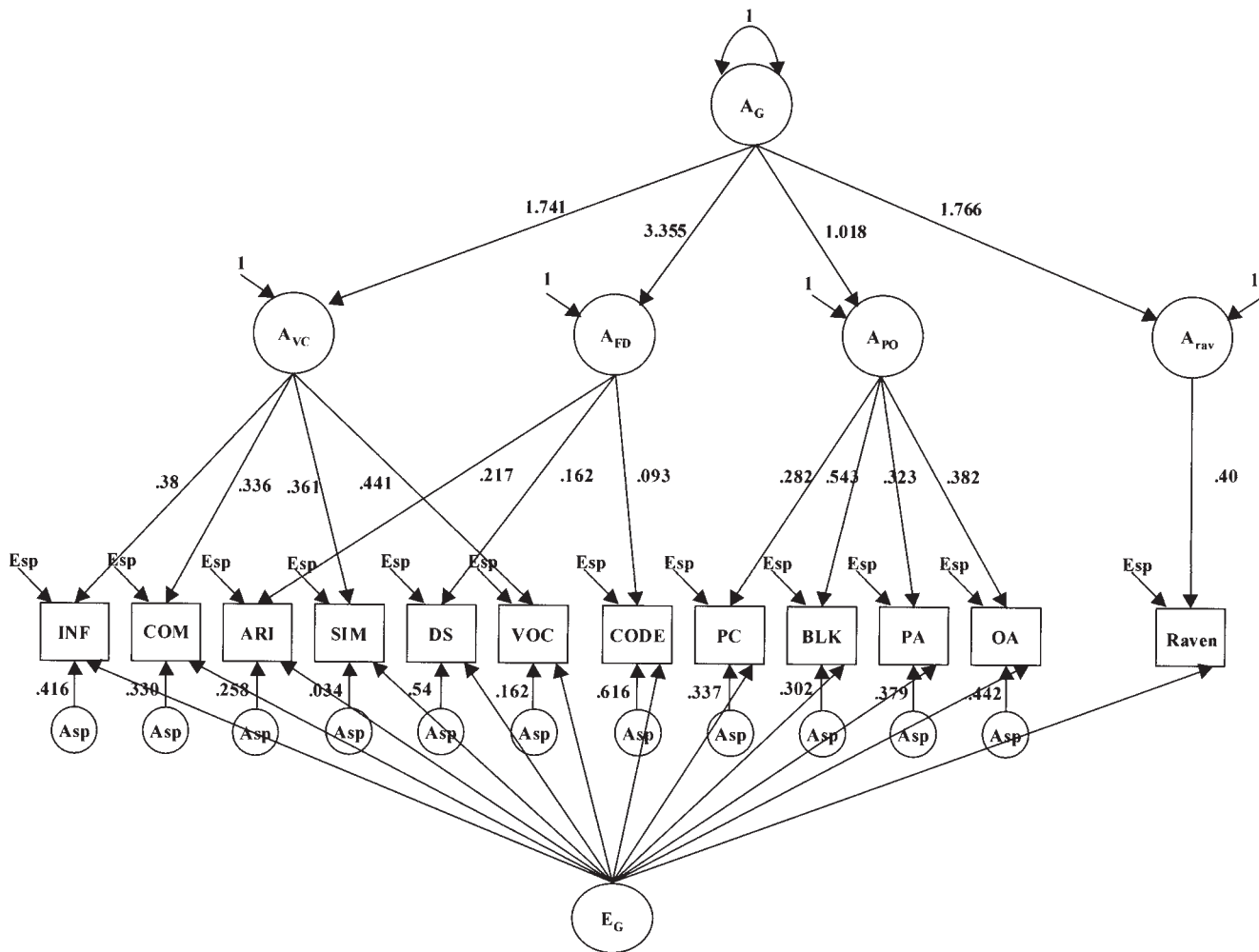


Fig. 1. Standardised path-coefficients of the best-fitting hierarchical model of the WAIS subtests, including the Raven. For clarity the estimates for the E factors were omitted, but can be extracted from Table VII.

For standard deviations of the subtests similarity and digit span, higher values were observed for males (1.92 versus 1.67 and 1.87 versus 1.53, respectively). Maximum-likelihood estimates of phenotypic correlations among subtests are shown in Table II. Sex differences were only observed for the correlation between coding and digit span: 0.29 for males and 0.07 for females (ns). The mean correlation between subtests comprising different scales was 0.27 for both males and females. Correlations between subtests loading on the verbal scale averaged 0.52 and on the performance scale 0.25. In the Dutch normative population these correlations are 0.57 and 0.44, respectively. The coherence of the performance scale subtests appears to be weaker than the relation among the

subtests from different scales. The phenotypic correlation of the Raven score with the verbal and performance scale WAIS subtests was on average 0.47 and 0.30, respectively, and with VIQ, PIQ, and full-scale IQ, 0.63, 0.51 and 0.66, respectively. Vernon (1983) reported correlations of 0.36 and 0.46 on average for the Raven with verbal and performance subtests, respectively, and 0.57, 0.70, and 0.72 with VIQ, PIQ, and full-scale IQ, respectively. In contrast with our results, the Raven test shared more variance with the performance scale.

The hierarchical phenotypic factor model of the WAIS subtests was composed of a second-order factor and the three first-order Kaufman factors: verbal comprehension (VC), perceptual organisation (PO),

Table I. Estimates of Means and Standard Deviations for the WAIS Subtests and Scales, Kaufman Factors, and the Raven Test.

Sub-tests	Males (<i>N</i> = 180)		Females (<i>N</i> = 208)		Sex Differences*
	M	(SD)	M	(SD)	$\Delta\chi^2$ (df = 1)
INF	6.09	(1.32)	5.84	(1.48)	2.51
COM	5.94	(1.67)	5.90	(1.66)	0.17
ARI	6.98	(1.90)	6.51	(1.84)	4.15†
SIM	7.07	(1.92)	7.29	(1.67)	0.56
DS	6.21	(1.87)	6.35	(1.53)	1.23
VOC	5.87	(1.65)	6.08	(1.64)	1.14
CODE	6.78	(1.84)	7.62	(1.61)	19.2†
PC	6.38	(1.68)	5.95	(1.78)	5.22†
BLO	7.24	(1.91)	7.42	(1.96)	0.14
PA	7.02	(2.01)	7.01	(1.89)	0.02
OA	6.46	(1.96)	6.64	(1.81)	0.44
Factors and Scales					
VC	25	(5.6)	25.1	(5.4)	0.05
FD	20	(4.3)	20.5	(3.5)	1.5
PO	27.1	(5.3)	27	(5.3)	0.03
VIQ	109.9	(12.7)	109.6	(11.6)	0.06
PIQ	115.7	(11.9)	117.1	(11.9)	0.87
FSIQ	113.5	(11.8)	114.0	(11.7)	0.07
RAVEN	4.95	(0.64)	4.94	(0.56)	0.08

WAIS Subtests: INF = Information; COM = comprehension; ARI = arithmetic; SIM = similarities; DS = digit span; VOC = vocabulary; CODE = coding; PC = picture completion; BLK = block design; PA = picture arrangement; OA = object assembly; *Kaufman Factors:* VC = Verbal comprehension; FD = freedom of distractibility; PO = perceptual organisation; *WAIS Scales:* VIQ = Verbal IQ; PIQ = performance IQ; FSIQ = full scale IQ. *Raven Score* = number of correct items divided by 10. *N* = number of individuals. * Significance test for means; † = $\Delta\chi^2(df = 1) > 3.84$, and implies a significant difference for 1 df.

freedom-from-distractibility (FD) and specific factors, accounting for the unique phenotypic variance for each subtest ($\chi^2_{41} = 78.08$, $P < 0.001$, AIC = -3.92). The negative AIC fit index of this model in-

dicates that it is a better fit to the data compared with a two-factor verbal (subtests 1 to 6) and performance (subtests 7 to 11) model ($\chi^2_{42} = 110.31$, $P < 0.001$, AIC = 26.3).

Table II. Maximum-likelihood Estimates of Phenotypic Correlations Among WAIS Subtests and Raven Test

	INF	COM	ARI	SIM	DS	VOC	CODE	PC	BLO	PA	OA	Raven
INF	—											
COM	0.55	—										
ARI	0.52	0.48	—									
SIM	0.55	0.59	0.53	—								
DS	0.39	0.34	0.46	0.38	—							
VOC	0.67	0.66	0.56	0.67	0.43	—						
CODE	0.21	0.16	0.26	0.14	0.29*	0.21	—					
PC	0.30	0.39	0.30	0.31	0.20	0.34	0.22	—				
BLK	0.35	0.31	0.47	0.36	0.29	0.34	0.19	0.30	—			
PA	0.28	0.36	0.29	0.33	0.21	0.32	0.08 ^{ns}	0.33	0.32	—		
OA	0.20	0.25	0.25	0.25	0.08 ^{ns}	0.21	0.07 ^{ns}	0.28	0.49	0.26	—	
Raven	0.49	0.47	0.51	0.47	0.38	0.51	0.25	0.31	0.40	0.32	0.25	—

Note: Number of subjects = 388. Mean correlation between subtests comprising the verbal scale = 0.52. Mean correlation between subtests comprising the performance scale = 0.25. Mean correlation between sub-tests from different scales = 0.27. ^{ns} = Nonsignificant correlation. * = Sex differences in correlation: 0.29 for males and 0.7^{ns} for females.

Univariate Genetic Analyses

The twin-correlations for subtest scores, Kaufman factors, VIQ, PIQ, FSIQ, and Raven scores for the five sex-by-zygosity groups, as well as for the MZ and DZ group, are given in Table III and indicate either dominance genetic or C effects. In the univariate genetic analyses an ADE and ACE sex-differences model fitted equally well. Given the small sample size, we report only additive genetic effects. The AE no-sex-differences model showed to be the best for the IQ scores and for all but the digital span subtest (Table IV). The univariate estimates of heritability for the verbal scale subtests show the additive genetic factor to account for 56% to 75% of the phenotypic variance (65% on average). Subtests of the performance scale exhibit a lower heritability on average (47%). The lowest heritabilities were observed for picture completion and picture arrangement (32% and 40%, respectively). Heritabilities for VIQ, PIQ, and FSIQ were 84%, 68%, and 82%, respectively. Because of the absence of major sex differences, the five groups were pooled across sexes for the multivariate analyses.

Multivariate Genetic Analyses

A full Cholesky decomposition for **A**, **C**, and **E** was fitted to the MZ and DZ between and within mean cross-product matrices. First, the significance of the shared environmental structure was tested (Table V).

The **C** structure could be omitted without deterioration in fit [$\Delta\chi^2(77) = 18, P = 0.99$]. The heritability estimates for this model closely resemble the univariate estimates. Higher heritabilities were obtained for the verbal scale subtests (58% to 75%, 65% on average) than for the performance scale subtests (34% to 67%, 48% on average). The lowest heritability estimates were again observed for picture completion and picture arrangement (34% and 39%, respectively). The Cholesky-AE model showed a reasonable fit. The genetic correlations showed a pattern similar to that of the phenotypic correlations. The mean genetic correlations among subtests of the verbal scale and performance scale were 0.72 and 0.48, respectively. The genetic correlation among subtests loading on different scales averaged 0.44 and was almost equal to the mean performance scale correlation. This rather high correlation suggested a general genetic factor influencing all subtests. The mean correlation among verbal scale subtests resulting from within-pair environmental influences (*E*) was small ($r = 0.12$), whereas that among the performance scale subtests and among different scale subtests was practically zero. The environmental (*E*) correlation matrix exhibited a pattern suggesting a general *E* factor.

In subsequent model fitting, a hierarchical AE three-factor model (Model 3) was specified (see Table V), in which the genetic structure was composed of three first-order factors (A_{VC} , A_{PO} , and A_{FD}), a second-order factor (A_G) and specific factors (A_{SP}); the non-

Table III. Twin Correlations for the WAIS Subtests, Kaufman Factors, WAIS Scales, and Raven Test

Sub-tests	MZM (<i>N</i> = 37)	DZM (<i>N</i> = 31)	MZF (<i>N</i> = 46)	DZF (<i>N</i> = 36)	DOS (<i>N</i> = 44)	MZ (<i>N</i> = 83)	DZ (<i>N</i> = 111)
INF	0.73	0.43	0.80	0.52	0.23	0.76	0.36
COM	0.66	0.17	0.73	0.33	0.32	0.70	0.27
ARI	0.63	0.20	0.64	0.45	0.18	0.64	0.27
SIM	0.72	0.11	0.58	0.04	0.22	0.66	0.16
DS	0.65	0.36	0.46	0.001	0.37	0.58	0.26
VOC	0.82	0.16	0.77	0.27	0.27	0.79	0.25
CODE	0.39	0.30	0.47	0.11	0.30	0.44	0.29
PC	0.25	0.32	0.28	0.05	0.35	0.28	0.24
BLK	0.70	0.25	0.65	0.57	0.44	0.67	0.44
PA	0.43	0.26	0.51	0.11	-0.11	0.46	0.05
OA	0.69	0.46	0.33	0.40	0.21	0.50	0.36
VC	0.90	0.27	0.86	0.35	0.36	0.88	0.33
FD	0.72	0.22	0.73	0.16	0.24	0.72	0.23
PO	0.72	0.31	0.67	0.48	0.27	0.70	0.35
VIQ	0.87	0.31	0.87	0.26	0.25	0.89	0.27
PIQ	0.74	0.23	0.67	0.47	0.26	0.70	0.34
FSIQ	0.86	0.19	0.84	0.44	0.24	0.85	0.30
RAVEN	0.77	0.24	0.50	0.35	0.42	0.66	0.39

Table IV. Univariate Model Fitting Results of the WAIS Subtests, Kaufman Factors, WAIS Scales and Raven Test Fitted to the 5 Sex-by-Zygoty Group Covariance Matrices

Sub-tests	AE sd model df = 11		AE nsd model df = 13		Variance components		
	χ^2	<i>P</i>	χ^2	<i>p</i>	<i>A</i>	<i>E</i>	h^2
INF	13.26	0.28	16.82*	0.21	1.502	0.491	0.75
COM	15.35	0.17	15.70*	0.27	0.775	0.970	0.65
ARI	7.96	0.72	9.32*	0.75	2.195	1.313	0.63
SIM	9.85	0.54	14.67*	0.33	1.813	1.319	0.58
DS	13.48*	0.26	20.43	0.09	1.606	1.284	0.56
VOC	7.91	0.72	8.30*	0.82	2.005	0.681	0.75
CODE	7.01	0.80	11.40*	0.58	1.344	1.658	0.45
PC	6.54	0.84	7.05*	0.90	0.960	2.026	0.32
BLK	5.94	0.88	6.02*	0.95	2.482	1.180	0.68
PA	7.23	0.78	8.42*	0.82	1.471	2.232	0.40
OA	9.88	0.54	14.46*	0.34	1.751	1.745	0.50
VC	8.71	0.65	10.24*	0.67	24.73	4.67	0.84
FD	17.64	0.10	18.90*	0.13	10.21	5.19	0.66
PO	4.4	0.96	4.6*	0.98	18.77	8.83	0.68
VIQ	9.62	0.57	11.19*	0.61	122.4	23.93	0.84
PIQ	3.62	0.98	3.84*	0.99	93.65	43.60	0.68
FSIQ	8.84	0.64	9.13*	0.76	110.7	24.23	0.82

Note: ADE and ACE sex differences models fitted equally well. The C factor could be dropped without significant decline in fit. Given the small sample size we report only additive genetic influences. For DS the sex-differences model fitted slightly better: h^2 females 0.43; h^2 males 0.64. * Indicates best-fitting model.

shared environmental influences were composed in a similar way (E_{VC} , E_{PO} , E_{FD} , E_G , E_{SP}). This model showed a good fit. The genetic structure showed a strong general component, whereas for the *E* structure the E_G loadings of VC, FD, and PO were very low. The AIC index of this model also indicated a better fit to the data compared with a hierarchical two-factor model with a verbal and performance group factor (Model 4,

$\chi^2_{216} = 263.85$, $P = 0.16$). In Model 5 the significance of the three first-order genetic factors was shown. Next, with Model 3 as a reference, more parsimonious structures for *E* were examined. In model 6 for *E*, only a general factor and specific factors were specified ($\chi^2_{217} = 229.5$, $P = 0.27$). The drop in fit compared with Model 3 was nonsignificant [$\Delta\chi^2(3) = 5.07$] and this model was chosen as the best fitting model.

Table V. Fit Indices for Multivariate Models Fitted to Between- and Within-Mean Product Matrices of MZ and DZ Pairs

Model	χ^2	df	<i>P</i>	AIC	$\Delta\chi^2$	Δ df	<i>P</i>
1. Cholesky ACE decomposition	109.40	66	0.001	-23	-	-	-
2. Cholesky AE decomposition	127.2	132	0.6	-137	18	77	ns
3. Hierarchical three-factor model	224.43	214	0.29	-203	-	-	-
4. Hierarchical two-factor model	236.85	216	0.16	-195	-	-	sig
5. Same as 3, but for A only A_G	251.08	217	0.06	-183	26.65	3	sig
6. Same as 3, but for E only E_G^*	229.50	217	0.27	-205	5.07	3	ns
7. Raven included in Model 6	279.11	261	0.21	-243	-	-	-

Note: Twin groups pooled across sexes: 83 MZ, 111 DZ. $\Delta\chi^2$ = Change in chi-square. Δ df = change in number degrees of freedom, ns = nonsignificant $\Delta\chi^2$, sig = significant $\Delta\chi^2$. * = Best-fitting model. Factors specified in Model 3: A_C , A_{VC} , A_{PO} , A_{FD} ; E_G , E_{VC} , E_{PO} , E_{FD} and A_{SP} ; E_{SP} . Factors specified in Model 4: A_G , A_V , A_P ; E_G , E_V , E_P , and A_{SP} ; E_{SP} . Subscripts: G = General, VC = Verbal Comprehension, PO = Perceptual Organisation, FD = Freedom of Distractibility, SP = Specific, V = Verbal, P = Performal.

The question of how the shared variance of the Raven and the WAIS is mediated was addressed by including the Raven scores as a subtest in the multivariate design. The Raven was included in Model 6 and allowed to load on the genetic VC, PO, and FD factors, respectively. For all three models, inclusion of the Raven distorted the Cohen factor pattern in a way that the respective factors would lose coherence and, therefore, the second-order factor would gain strength. Abandoning this approach, we allowed the Raven to load on a fourth, first-order genetic factor (see Fig. 1). The fit of this model was good. The covariance of the Raven test score with the WAIS subtests was largely accounted for by the general second-order genetic factor. The mean genetic correlation of the Raven with the verbal subtests was higher than with the performance subtests: 0.73 and 0.51, respectively (Table VI). Genetic and environmental correlations among WAIS subtests, as well as heritability estimates, did not change significantly when the Raven was included in the multivariate analysis. Heritability estimates for the Raven (64%) and the WAIS subtests, along with information about their precision in the form of likelihood-based 80% confidence intervals, are reported in Table VII.

DISCUSSION

Although hierarchical models of intelligence are widely accepted, application of these models to twin data is sparse. The aim of this paper was to address the question of how the hierarchical model of the WAIS subtests (collected in a Dutch sample) fits a genetic model. We investigated whether first-order group factors possess genetic and environmental variance inde-

pendent of the higher-order general factor and whether the hierarchical structure was significant for all sources of variance. The hierarchical model with the three Cohen group factors (VC, PO, and FD) and a second-order g factor showed the best fit to the phenotypic data. The genetic analyses showed no evidence for common environmental influences. Additive genetic influences (A) were best explained by the three Cohen group factors and a second-order g factor, whereas the unique environmental source of variance (E) could be modeled by a single general factor. For A, test-specific factors were modeled to account for genetic variance that is not shared between tests. For E these test specific factors need to be specified to account for measurement error. These results indicate that the factorial structure of the WAIS subtests is determined by individual differences in genetic structure (phenotypic g is strongly related to genetic g). To the extent that these results can give us information on the structure of intelligence in adults, they support (1) a modular genetic system (genetic group factors possess independent variance after controlling for the higher-order genetic g); (2) environmental molarity; and (3) the notion that general intelligence g has a biological basis.

In contrast with earlier findings (Tambs *et al.*, 1986; LaBuda, DeFries, and Fulker, 1987; Casto *et al.*, 1995), the influences of shared-family environment were nonsignificant and genetic influences were high. Shared environmental influences on the WAIS-R subtest scores of the Tambs *et al.* study were low. Results were suggested to be interpreted with caution because of the small sample size and relatively low phenotypic correlations observed among subtests. The significant shared environmental variance of the WISC-R data in

Table VI. Genetic (below diagonal) and Nonshared Environmental (above diagonal) Correlations for the WAIS Subtests and Raven Test (estimates based on Model 7)

	INF	COM	ARI	SIM	DS	VOC	CODE	PC	BLK	PA	OA	Raven
INF	—	0.07	0.03	0.04	0.02	0.04	0.00	0.05	0.01	0.05	0.02	0.03
COM	0.79	—	0.14	0.19	0.10	0.22	0.00	0.27	0.02	0.23	0.09	0.14
ARI	0.69	0.71	—	0.08	0.04	0.09	0.00	0.11	0.01	0.09	0.04	0.06
SIM	0.88	0.90	0.79	—	0.06	0.12	0.00	0.15	0.01	0.13	0.05	0.08
DS	0.53	0.54	0.69	0.60	—	0.07	0.00	0.08	0.01	0.07	0.03	0.04
VOC	0.86	0.88	0.77	0.98	0.59	—	0.00	0.17	0.02	0.15	0.06	0.09
CODE	0.34	0.35	0.44	0.39	0.34	0.38	—	0.00	0.00	0.00	0.00	0.00
PC	0.42	0.43	0.50	0.47	0.38	0.47	0.25	—	0.02	0.18	0.07	0.11
BLK	0.51	0.52	0.60	0.58	0.46	0.57	0.30	0.71	—	0.02	0.01	0.01
PA	0.42	0.43	0.50	0.48	0.38	0.47	0.25	0.59	0.72	—	0.06	0.09
OA	0.42	0.43	0.50	0.48	0.39	0.47	0.25	0.60	0.72	0.60	—	0.04
Raven	0.66	0.68	0.79	0.75	0.61	0.74	0.39	0.48	0.58	0.48	0.48	—

Table VII. Percentages Genetic and Environmental Variance and Heritabilities with 80% CI for the WAIS Subtests and Raven (estimates based on Model 6) (% Variance Accounted for by the Genetic and Environmental Factors)

Subtests	A _G	A _{VC}	A _{FD}	A _{PO}	A _{RAV}	A _{SP}	<i>h</i> ²	80% CI	E _G	E _{SP}	<i>e</i> ²
INF	44	14	—	—	—	17	76	71–81	.3	24	24
COM	34	11	—	—	—	11	56	54–67	15	29	44
ARI	53	—	5	—	—	7	65	53–64	2	33	35
SIM	40	13	—	—	—	0	53	49–60	5	42	47
DS	30	—	3	—	—	29	61	52–67	1	38	39
VOC	52	17	—	—	—	3	72	69–78	4	24	28
CODE	10	—	1	—	—	38	48	38–58	0	52	52
PC	8	—	—	8	—	11	27	19–40	15	58	73
BLK	31	—	—	30	—	9	69	63–74	0	31	31
PA	11	—	—	10	—	14	36	29–49	10	54	64
OA	15	—	—	15	—	19	49	41–57	1	50	51
Raven	48	—	—	—	16	—	64	59–71	2	34	36

Note: A_G = General genetic factor; A_{VC} = verbal comprehension genetic factor; A_{PO} = perceptual organisation genetic factor; A_{FD} = freedom of distractibility genetic factor; A_{SP} = specific genetic factor; E_G = general environmental factor; E_{SP} = specific environmental factor; — means fixed to 0.

the other two studies was not surprising given the mean age of the samples (12.5 and 11.2 years, respectively). The effects of shared environment are suggested to decrease as children begin their formal education, and nonshared family environment becomes more important as children become adolescents (Scarr and McCartney, 1983; Thompson, 1993, Boomsma, 1993). In a longitudinal study, Rietveldt, Dolan, van Baal, and Boomsma, (2001) showed decreasing effects of familial environment and increasing genetic effect on individual differences in both verbal and nonverbal abilities with age (5, 7, 10 years).

In the literature on cognitive development it was suggested that intellectual abilities may become progressively independent (multidimensional) from infancy to childhood. After stabilization of the structure of intelligence in early adulthood, a process of dedifferentiation takes place in late adulthood and old age (Reinert, 1970). Multivariate genetic models, applied on longitudinal data can give useful information on genetic and environmental aspects of cognitive development (Cardon and Fulker, 1993; Price *et al.*, 2000; Rietveldt *et al.*, 2001). At the genetic level, the observed modest correlations between verbal and nonverbal genetic factors do suggest relative independence between verbal and nonverbal cognitive abilities in infancy and childhood. The steady increase in genetic correlation from age 2 to 4 (Price *et al.*, 2000); from 5 to 7 to 10 (0.21, 0.32 and 0.36, Rietveldt *et al.*, 2001) and the genetic correlation between VC and PO factors in this sample of 18-year-old twins (0.87) rather suggests pro-

gressive “unidimensionality” at the genetic level. In other words the genetic general factor *g* becomes more prominent. Although there is evidence for independent genetic effects across different dimensions of cognitive functioning, most genetic effects are general in adults. Petrill (1997) concludes that because the independent genetic influences on verbal and nonverbal ability are small, it will be primarily the environment responsible for the distinctions between different cognitive dimensions.

The Raven is a widely used nonverbal test measuring analytic intelligence and the ability to reason and solve problems involving new information, without relying on acquired knowledge and skills. This implies a high loading on *g*, and thus on the general rather than the group factors. Covariance with the WAIS IQ subtests and other tests of mental ability was, therefore, expected to be mediated by general rather than group factors. The next question was whether this covariance is mediated by general genetic or by general environmental influences. The covariance between the WAIS and the Raven was solely accounted for by the general genetic factor.

A possible limitation of this study was the finding of relatively low correlations among the performance subtests in comparison both to correlations among the verbal subtests and to previous studies. It was also unusual to find the Raven correlating more highly with the verbal than with the performance subtests. At the same time, factor analysis of the WAIS subtests plus the Raven showed that the Raven and the verbal sub-

tests (with the predictable exception of digit span) had the highest g -loadings (averaging 0.73) in comparison to the performance subtests whose g -loadings averaged 0.49. Taken together, these findings may indicate that subjects in the present sample relied more heavily on verbal strategies to solve the Raven items, and the g factor extracted here is more verbally loaded than is typical. It is interesting to note, however, that there is a positive relationship between the subtests' (and Raven's) g -loadings and their heritabilities: the correlation between these is 0.79, which reduces to 0.43 when controlling for the tests' differential reliabilities. Moreover, an even stronger relationship exists between the WAIS subtests' heritabilities and the degree to which each correlates with the Raven: the correlation here is 0.85 (0.55 after controlling for reliabilities). Thus, even though the g factor that was extracted in this sample is more verbally loaded than is typical, it is nonetheless yielding results that have consistently been observed in previous investigations: namely, more g -loaded tests being more heritable, and more heritable tests correlating more highly with another test (the Raven) that has itself been shown in numerous independent studies to be highly g -loaded (Jensen, 1998). Finally, it should be kept in mind that the Raven was administered 1½ years before the WAIS and it is possible that age-specific environmental factors, or simply day-to-day (or "year-to-year") fluctuations, attenuated the WAIS-Raven correlations somewhat. This same effect may also have inflated the environmental variances (E and C) unique to the Raven and the WAIS, respectively.

By revealing additional information about underlying genetic and environmental factor structures, multivariate genetic analysis has helped to answer questions about the nature of the general factor of intelligence (g). The construct g refers to the variance component of individual differences in IQ that is common to all tests of mental ability. The psychometric aspects of g are well established empirically: Vernon (1989), for example, demonstrated the "generality of g " by showing that the g factors extracted from two very different batteries of tests (IQ subtests versus relatively simple reaction time tests) were quite highly correlated, and Thorndike (1987) also provided evidence that the g factors extracted from different and diverse collections of ability tests are very similar to one another. Research has now turned to focus on the biological basis of g (Jensen, 1998). In this search, establishment of a genetic basis of g is of great importance. The covariation among the WAIS subtests and the co-

variation between the subtests and the Raven in our data are predominantly influenced by a second-order genetic factor and thus strongly support the notion of a biological basis of g .

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REFERENCES

- Boomsma, D. I., and Molenaar, P. C. M. (1986). Using LISREL to analyze genetic and environmental covariance structure. *Behav. Genet.* **16**:237–250.
- Boomsma, D. I. (1993). Current status and future prospects in twin studies of the development of cognitive abilities, infancy to old age. In T.J. Bouchard and P. Propping (eds.), *Twins as a Tool of Behavioral Genetics*, John Wiley & Sons, Chichester, pp. 67–82.
- Boomsma, D. I., Kaptein, A., Kempen, H. J. M., Gevers Leuven, J. A., & Princen, H. M. G. (1993). Lipoprotein(a): Relation to other risk factors and genetic heritability. Results from a Dutch parent-twin study. *Atherosclerosis* **99**:23–33.
- Bouma, A., Mulder, J. L., and Lindeboom, J. (1996). *Neuropsychologische Diagnostiek: handboek*. Swets & Zeitlinger B.V., Lisse, The Netherlands.
- Burt, C. L. (1949). *The factors of the Mind: An introduction to Factor Analysis in Psychology*. Macmillan, New York.
- Cardon, L. R., Fulker, D. W., DeFries, J. C., and Plomin, R. (1992). Multivariate genetic analysis of specific cognitive abilities in the Colorado Adoption Project at age 7. *Intelligence* **16**:383–400.
- Cardon, L. R., and Fulker, D. W. (1993). Genetics of specific cognitive abilities. In R. Plomin, and G.E. McClearn (eds.), *Nature, Nurture & Psychology*, American Psychological Association, Washington, pp. 99–120.
- Casto, S. D., DeFries, J. C., and Fulker, D. W. (1995). Multivariate genetic analysis of Wechsler Intelligence Scale for Children-Revised (WISC-R) factors. *Behav. Genet.* **25**:25–32.
- Cohen, J. (1957). The factor structure of the WAIS between early adulthood and old age. *J. Consul. Psychol.* **21**:283–290.
- Heath, A. C., Jardine, R., Eaves, L. J., and Martin, N. G. (1989a). The genetic structure of personality II. Genetic item analysis of the EPQ. *Personality and Individual Differences* **10**:615–624.
- Heath, A. C., Eaves, L. J., and Martin, N. G. (1989b). The genetic structure of personality III. Multivariate analysis of the EPQ scales. *Personality and Individual Differences* **10**:877–888.
- Horn, J. L., and Catell, R. B. (1966). Refinement and test of the theory of fluid and crystallized intelligence. *J. Educ. Psychol.* **57**:253–270.
- Jensen, A. R. (1998). *The g Factor: The Science of Mental Ability*. Praeger, Westport, Connecticut.
- Kaufman, A. S. (1975). Factor analysis of the WISC-R at 11 age levels between 6½ and 16½ years. *J. Consul. Clin. Psychol.* **43**:135–147.
- LaBuda, M. C., DeFries, J. C., and Fulker, D. W. (1987). Genetic and environmental covariance structures among WISC-R subtests: A twin study. *Intelligence* **11**:233–244.
- Luo, D., Petrill, S. A., and Thompson, L. A. (1994). An exploration of genetic g : Hierarchical factor analysis of cognitive

- data from the Western Reserve Twin Project. *Intelligence* **18**:335–347.
- Martin, N. G., and Eaves, L. J. (1977). The genetical analysis of covariance structure. *Heredity* **38**:79–95.
- Mulder, J. L., Dekker, R., and Dekker, P. H. (1995). *Verbale leer- en Geheugen Test. Handleiding voor afname, scoring en interpretatie van de test*. Lisse, The Netherlands: Swets & Zeitlinger B.V.
- Neale, M. C. (1997). *Statistical Modeling with Mx*, Department of Human Genetics, Box 3, MCV, Richmond VA 23298.
- Neale, M. C., and Miller, M. B. (1997). The use of likelihood-based confidence intervals in genetic models. *Behav. Genet.* **27**:113–120.
- Petrill, S. A., Luo, D., Thompson, L. A., and Detterman, D. K. (1996). The independent prediction of general intelligence by elementary cognitive tasks: genetic and environmental influences. *Behav. Genet.* **26**:135–147.
- Petrill, S. A. (1997). Molarity versus modularity of cognitive functioning? A behavioral genetic perspective. *Curr. Direct. Psychological Sci.* **6**:96–99.
- Price, T. S., Eley, T. C., Dale, P. S., Stevenson, J., Saudino, K., and Plomin, R. (2000). Genetic and environmental covariation between verbal and nonverbal cognitive development in infancy. *Child Dev.* **71**:948–959.
- Reinert, G. (1970). Comparative factor analytic studies of intelligence throughout the human life-span. In L. R. Goulet & P. B. Baltes (Eds.), *Life-span Developmental Psychology* (pp. 467–484). New York: Academic Press.
- Rietveld, M. J. H., Dolan, C. V., van Baal, G. C. M., and Boomsma D. I. A. (2001). Twin study of differentiation of cognitive abilities in childhood (Submitted).
- Rijsdijk, F. V., Boomsma, D. I., and Vernon P. A. (1995). Genetic analysis of peripheral nerve conduction velocity in twins. *Behav. Genet.* **25**:341–348.
- Rijsdijk, F. V., and Boomsma, D. I. (1995). Genetic mediation of the correlation between peripheral nerve conduction velocity and IQ. *Behav. Genet.* **27**:87–98.
- Rijsdijk F. V., Vernon P. A., and Boomsma D. I. (1998). The genetic basis of the relation between speed-of-information-processing and IQ. *Behav. Brain Res.* **95**:77–84.
- Scarr, S., and McCartney, K. (1983). How people make their own environments: A theory of genotype-environmental effects. *Child Dev.* **54**:424–435.
- Stinissen, J., Willems, P. J., Coetsier, P., and Hulsman, W. L. L. (1970). *Manual for the Dutch translated and adapted version of the Wechsler Adult Intelligence Scale (WAIS)*. Swets & Zeitlinger B.V, Lisse, The Netherlands.
- Tambis, K., Sundet, J. M., and Magnus, P. (1986). Genetic and environmental contributions to the covariation between the Wechsler Adult Intelligence Scale (WAIS) sub-tests: A study of twins. *Behav. Genet.* **16**:475–491.
- Thompson, L. A. (1993). Genetic contributions to intellectual development in infancy and childhood. In P.A. Vernon (ed.), *Biological Approaches to the Study of Human Intelligence*, Ablex, Norwood, New Jersey, pp. 95–138.
- Thorndike, R. L. (1987). Stability of factor loadings. *Personality and Individual Differences* **8**:585–586.
- Van Beijsterveldt, C. E. M., van Baal, G. C. M., de Geus, E. J. C., Molenaar, P. C. M., and Boomsma, D. I. (1995). Individual differences in P300 amplitude: A longitudinal study in adolescent twins. *Behav. Genet.* **25**:292 (abstract).
- Van Beijsterveldt, C. E. M., Molenaar, P. C. M., de Geus, E. J. C., and Boomsma, D. I. (1996). Heritability of human brain functioning as assessed by Electroencephalography (EEG). *Am. J. Hum. Genet.* **58**:562–573.
- Vernon, P. A. (1983). Speed of information processing and general intelligence. *Intelligence* **7**:53–70.
- Vernon, P. A. (1989). The generality of g. *Personality and Individual Differences* **10**:803–804.
- Vernon, P. E. (1950). *The structure of human abilities*. London: Methuen

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