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The genetic basis of the relation between speed-of-information-processing and IQ

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Abstract

The relationship of speed-of-information-processing (SIP), as derived from reaction times (RTs) on experimental tasks, and intelligence has been extensively studied. SIP is suggested to measure the efficiency with which subjects can perform basic cognitive operations underlying a wide range of intellectual abilities. Observed phenotypic correlations between RT and IQ typically are in the -0.2 to -0.4 range, and the question is addressed to what extent this relationship is determined by genetic or environmental influences. In a group of Dutch twins the heritabilities for RT tasks at age 16 and 18 years were estimated longitudinally and the nature of the RT-IQ relationship was investigated. At age 16 years heritabilities for a simple reaction time (SRT) and choice reaction time (CRT) were 64 and 62% and the average phenotypic correlations between the RTs and IQ, assessed by the Raven standard progressive matrices, was -0.21. At the second test occasion lower heritabilities were observed for the RTs, probably due to modifications in administration procedures. The mean correlations between the RTs and WAIS verbal and per formal subtests were -0.18 and -0.16. Multivariate genetic analyses at both ages showed that the RT-IQ correlations were explained by genetic influences. These results are in agreement with earlier findings (Baker et al., Behav Genet 1991;21:351–67; Ho et al., Behav Genet 1988;18:247–61) and support the existence of a common, heritable biological basis underlying the SIP-IQ relationship.

Keywords: Reaction times; WAIS; Raven; Twins; Multivariate genetic analyses; Structural equation modelling; Heritability; IQ

1. Introduction

In the search for determinants of human intelligence the relationship between measures of timed performance on experimental tasks and scores on psychometric tests of intelligence is the most extensively studied and well established. Performance on reaction time (RT) tasks are supposed to be a reflection of the speed-of-information processing (SIP). The idea of studying RTs as correlates of intelligence goes back to Galton [1], but it was not until after the 1960s that a great deal of research on RTs and intelligence was successfully conducted. One of the major contributors to this area is Jensen [2,3]. The history of the research on RTs is extensively reviewed elsewhere [4].

A theoretical model for the relationship between RTs and IQ was given by the 'neural efficiency' model [2,5], in terms of three characteristics of the short term memory (STM) system in which basic cognitive operations are carried out: the limited capacity of the STM system; the rapid decay of information in absence of continued rehearsal and the trade-off between the amount of information that can simultaneously be stored and processed. SIP is regarded as the fourth property which can prevent the capacity threshold from being exceeded. The speed or efficiency with which individuals can execute basic cognitive operations at

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each step in solving a given problem is expected to have an effect on the success of their performance. Beyond showing the existence of RT-IQ correlations, the nature of this relationship can further be explored by means of twin data.

Twin and family studies indicate considerable genetic influences for individual differences in intelligence [6]. Moderate heritability estimates ($h^2 = 46$ and 54%) for RT tasks were observed in studies of reared-apart adult twins [7,8]. Another adult twin study reported a heritability of 49% for a 'general speed of response' factor [9]. In younger populations common environmental factors seem to play a bigger role in explaining individual differences for SRT and CRT [10].

Only a few studies have investigated the genetic and environmental covariance between SIP and IQ. Results of multivariate genetic analysis indicated that phenotypic correlation between two RT factors (rapid automatic naming and symbol processing speed) and full-scale IQ (both r's -0.42) was largely attributable to correlated genetic effects [11]. This was also the case when adult RT twin data of the Vernon study [9] was re-examined. The phenotypic correlations of RT with verbal and performance IQ data (both r's -0.59) were entirely mediated by genetic factors [12]. Individual differences in the speed with which cognitive operations can be executed were suggested to be responsible for individual differences in IQ as a consequence of differences in neurophysiological properties of the brain that may be hypothesized to underlie both SIP and IQ. What was shown by these results is a common genetic (biological) basis for IQ and SIP. More recently, in children, multivariate analyses of RT tasks and the WISC-R subtests, showed the SIP-IQ covariance to be predominantly determined by shared family environment (C) [13]. This result is in accordance with significant C effects on IQ for this age interval (6–13 years).

Genetic studies of RTs, suggest moderate to high heritabilities in adult and adolescent samples. The RT-IQ correlation is mainly due to genetic factors. In the present study results of a longitudinal genetic study on RTs and IQ are reported. In a sample of Dutch twin pairs performance on a SRT and CRT task and IQ scores were examined at age 16 and 17.5 years. The genetic relationship between RTs and IQ was examined in a multivariate design including all variables. In contrast, earlier studies [11,12] employed phenotypically derived factor scores of SIP and IQ in the genetic analyses. A disadvantage of the later method may be that phenotypic factors may yield quite a different pattern than is observed for the genetic and environmental factors when employing the complete set of variables in a multivariate genetic design.

2. Subjects and methods

2.1. Subjects

Subjects were 213 Dutch twin pairs who participated in a longitudinal project which investigated variation in peripheral nerve conduction velocity and intelligence [14,15] and genetic and environmental influences on brain development [16]. Mean age on occasion I was 16.13 years (S.D., 0.56), on occasion II 17.6 years (S.D., 0.54). Data on the first test occasion were available for 80 monozygotic (MZ) and 108 dizygotic (DZ) twin pairs (including 44 opposite sex pairs). On test occasion II data were available for 74 MZ and 100 DZ pairs (including 39 opposite sex pairs). The drop-out pairs did not significantly differ in IQ score compared to the other participants. For 117 same-sex twin pairs zygosity was determined by blood group and DNA typing and for the others by questionnaire information.

2.2. RT tasks

SRT, the display of a reaction stimulus, either a digit or a letter, requiring a right-key response (72 trials).

CRT, the display of a digit requiring a right-key response and that of a letter a left-key response (72 trials).

The RT tasks, part of a battery of five tasks, were administered via a computer with attached response console. The sum of the 'decision time' (time from onset of the stimuli to the release of a home key) and the 'movement time' (time from releasing the home key to pressing the response key) was employed as the measure for speed of performance (msec). For each subject (per task), outlier trials exceeding three standard deviation (S.D.) units above or below an initially computed individual mean, were removed. In addition to this individual screening of outliers per RT task, all subjects with mean RTs exceeding ± 3 S.D. units from the group means were excluded. Subjects with less than 50% correct responses on a RT task were excluded as well. Subjects received at least 10 practice trials for each task. At test occasion II the same battery was administered with a few modifications. The number of trials was reduced to 60 to shorten the administration time. After each response, the RT was displayed and feedback was given whenever it was slower than an established target RT value for that specific task. Correct responses were rewarded with 5 cents.

2.3. IQ tests

On the first visit the Raven standard progressive matrices [17] and on the second visit the Dutch version of the WAIS [18] were administered.

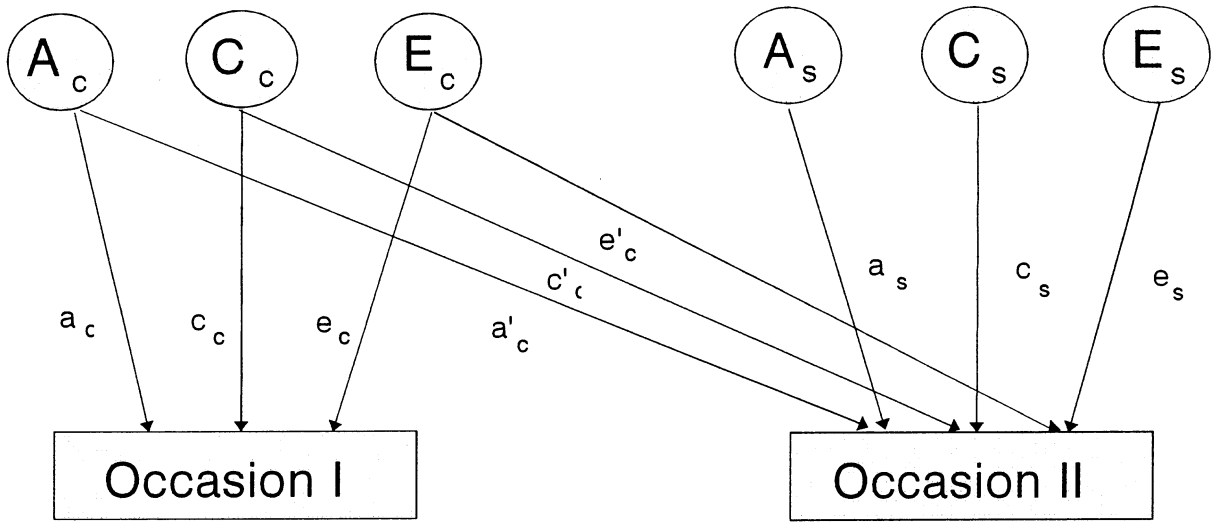

Fig. 1. Longitudinal genetic model for RTs of test occasion I and II. *A*, *C* and *E* are the additive genetic, the shared environmental and the unique environmental components. A_C, C_C and E_C represent influences common to the RT scores of test occasion I and II. A_S, C_S and E_S represent influences specific to the RTs of test occasion II.

2.4. Statistical analyses

2.4.1. Phenotypic analyses

Phenotypic correlations among RTs and IQ scores were estimated by maximum-likelihood (ML). This was accomplished with the structural equation modelling program Mx [19]. To raw data a model was fitted for the covariances in which ML correlations and S.D. were obtained. This model can be denoted as:

$$\Sigma_{YY} = \mathbf{S} \times \mathbf{R} \times \mathbf{S}',$$

where Σ_{YY} is the estimated $v \times v$ covariance matrix, **S** is a $v \times v$ diagonal matrix in which the S.D. are estimated, and **R** is a $v \times v$ symmetric matrix in which the correlations among variables are estimated (v = number of variables). A model for the means was specified as well. The fit of models which constrain parameter estimates of means, S.D. or correlations to be equal across groups can be compared to the fit of models which allow them to vary. This is done by subtracting the -2*LL of the unconstrained model from that of the constrained one, yielding a χ^2 distribution. The degrees of freedom (df) for this test is equal to the difference in df of the two models [20]. Significance of single correlations can be tested by evaluating the significance of the χ^2-change when the specific element in matrix **R** is fixed at zero. When for the MZ and DZ groups, two sets of variables for twin1 and twin2 are considered (dimensions of **R** and **S**: $2v \times 2v$), twin correlations can be estimated with the same model.

2.5. Longitudinal genetic analysis

Variation in phenotype was modelled as a function of variation in genotype and environment. Sources of variation considered were *A*, additive genetic variation (i.e. the sum of the average effects of the individual alleles at all contributing loci), *C*, environmental variation shared by family members in the same household and *E*, random, environmental variation that is not shared by family members. The phenotypic variance can be expressed as a simple additive function of the effects of *A*, *C* and *E*:

$$V_P = V_A + V_C + V_E.$$

Twin data are very useful to unravel these sources of variance. Resemblance within MZ pairs is caused by equal genetic constitution and shared environment, differences are caused solely by unique environment. Resemblance in DZ pairs are caused by shared environment, and genetic factors. However genetic factors contribute less to DZ resemblance since they share only 50% of their genes on average. Decomposition of the phenotypic variance of each RT task measured at age 16 and 17.5 years, was carried out in a longitudinal genetic analyses. Per task the RT score of occasion I was selected as first, and that of occasion II as second variable (Fig. 1). The *A*, *C* and *E* matrices were composed of a common factor (A_C, C_C, E_C) influencing the RT scores of both occasions and a specific factor (A_S, C_S, E_S) influencing the RT score of occasion II.

The loading of the first RT score on the common genetic factor is represented by the path coefficient a_c and that of the second score by a'_c. The factor A_S represents new genetic influences at age 17.5 years. The relative contributions of genetic and environmental influences to individual RT differences were estimated by ML, conducted on raw MZ and DZ data. This method is especially useful for handling incomplete data. Subjects with missing values for one RT task are not excluded from the sample and, the loss of valuable data is minimized. Goodness-of-fit of alternative nested models in which the *C* structure and specific parameters in *A* and *E* were dropped, were assessed by χ^2-change. The *ACE* model is compared to the saturated model in which the estimated covariance matrix is specified to be $\mathbf{S} \times \mathbf{R} \times \mathbf{S}'$ and the means, S.D. and correlations were unconstrained across groups.

2.6. Multivariate genetic analyses

The RT-Raven covariance was examined by imposing a triangular decomposition upon the **A**, **C** and **E** matrix. In a triangular decomposition the number of latent factors equals the number of variables. The first factor influences all variables, the second factor the second and subsequent variables, and so on. The last factor only contributes to the last variable. To examine the covariance among the RTs and WAIS subtests, models with a number of group factors and specific factors were specified for the **A**, **C** and **E** structures and fitted to the mean-squares-between pairs (MSB) and mean-squares-within pairs (MSW) matrices of the MZ and DZ pairs. Among the Group factors were general factors, loaded by all variables. The dimensions of the **A**, **C** and **E** matrices were $(v \times f)$, where f is the number of group factors. The specific factors $\mathbf{A}_{SP}$, $\mathbf{C}_{SP}$ and $\mathbf{E}_{SP}$ had dimension $v \times v$, representing the variance specific to each variable. Significance of these factors were tested by χ^2-change when fixing particular factors or loadings at zero. Heritability estimates for all variables as well as their 95% confidence intervals (CIs) were computed based on the best fitting model.

3. Results

3.1. Phenotypic analyses of test occasion I and II

The distribution of the Raven score was negatively skewed (-0.98), and a quadratic transformation was conducted to obtain a more symmetric distribution (-0.49). Distributions for the WAIS subtest scores and RT tasks at occasion I and II all showed acceptable symmetry. Descriptive statistics for RTs and Raven IQ occasion I are given in Table 1. Means and S.D. differed slightly between the MZ and DZ group. The correlation between SRT and CRT was 0.73 and between the RTs and the Raven -0.21 and -0.22.

WAIS full-scale IQ (113.8) was higher and the S.D. (11.7) lower than the population mean (100) and S.D. (15). This is most probable an effect of the dated norms for the Dutch WAIS [21]. The mean phenotypic correlation among the verbal WAIS subtests was 0.54, among the WAIS performance subtests 0.26 and between the verbal and performance subtests 0.27 (Table 2). RTs were, on average, almost equally correlated with the verbal and performance subtests (-0.18 and -0.16).

3.2. Longitudinal genetic analyses of RTs from test occasion I and II

Twin correlations for RTs on both occasions are given in Table 3. The fit of the *ACE* model (Table 3) was compared against the saturated model. The *C* structure was not significant and could be dropped for both RTs. Genetic and environmental influences from occasion I on occasion II (a'_c and e'_c) were significant for both RT tasks. The significance of new genetic influences was tested by omitting the specific genetic influences (A_S) from the *AE* model. Expression of new genetic influences was significant for both RTs tasks. New environmental influences (including measurement errors) were highly significant. For Raven IQ and the WAIS subtests an *AE* model was observed to have the best fit (unpublished results).

3.3. Multivariate genetic analyses of test occasion I

Table 4 shows the multivariate genetic analyses of the RT-Raven covariance. Model 1, a triangular *ACE* model showed a good fit. The drop of the C structure resulted in an even more parsimonious *AE* model. In Model 3 it was tested whether or not the RT-Raven environmental covariances could be fixed at zero. The fit of this model showed no significant decline. This

Table 1
ML estimates of phenotypic correlations among RTs and the Raven standard progressive matrices, means and S.D. (occasion I, age 16)

Subtests	Simple RT	Choice RT	Raven
Simple RT	—		
Choice RT	0.73	—	
Raven	−0.21	−0.22	—
Means	452.91	654.19	247.02
S.D.	66.87	76.75	57.15

RTs in msec; the Raven score = (number of correct items)^2/10.
Number of observations for SRT and CRT: 175 in MZ group, 238 in DZ group.
Number of observations for Raven: 181 in MZ group, 242 in DZ group.

Table 2
ML estimates of phenotypic correlations among WAIS subtests and RTs, means and S.D. (occasion II, age 18)

Subtests	INF	COM	ARI	SIM	DS	VOC	CODE	PC	BLK	PA	OA	SRT	CRT
Information	—												
Comprehension	0.55	—											
Arithmetic	0.53	0.48	—										
Similarity	0.55	0.59	0.52	—									
Digit span	0.38	0.33	0.46	0.37	—								
Vocabulary	0.67	0.66	0.55	0.68	0.43	—							
Coding	0.20	0.15	0.24	0.16	0.20	0.20	—						
Pict compl	0.32	0.39	0.32	0.32	0.19	0.34	0.19	—					
Blok design	0.35	0.31	0.47	0.36	0.29	0.35	0.19	0.30	—				
Pict arrange	0.29	0.36	0.30	0.34	0.22	0.33	0.08^{ns}	0.34	0.34	—			
Obj assembly	0.20	0.25	0.25	0.25	0.09^{ns}	0.23	0.07^{ns}	0.28	0.50	0.27	—		
Simple RT	−0.30	−0.17	−0.24	−0.13	−0.19	−0.23	−0.18	−0.19	−0.20	−0.14	-0.09^{ns}	—	
Choice RT	−0.23	−0.13	−0.17	-0.05^{ns}	−0.18	−0.18	−0.22	−0.16	−0.18	−0.14	-0.05^{ns}	0.58	—
Means	5.96	5.92	6.73	7.19	6.28	5.99	7.23	6.15	7.34	7.01	6.55	270.87	505.43
S.D.	1.41	1.66	1.88	1.79	1.69	1.64	1.77	1.94	1.94	1.94	1.99	69.95	47.33

Number of observations for all WAIS subtests: 166 in MZ group, 222 in DZ group.
For SRT and CRT: 167 in MZ groups, 218 and 217 in DZ group.
ns, non-significant correlation.

Table 3
Longitudinal model fitting results for RTs of test occasion I and II, fitted to raw data

	Twin correlations (95% CI)				Longitudinal model fitting results													
	Occasion I		Occasion II		Saturated model		*ACE*		*AE*		*AE* no a'_c		*AE* no e'_c		*AE* no a_s		Heritabilities (%)	
	MZ	DZ	MZ	DZ	$-2*LL$	df	$\Delta\chi^2_{11}$	*P*	$\Delta\chi^2_3$	*P*	$\Delta\chi^2_1$	*P*	$\Delta\chi^2_1$	*P*	$\Delta\chi^2_1$	*P*	h^2_I	h^2_{II}
SRT	0.58 (0.42–0.70)	0.33 (0.16–0.48)	0.44 (0.26–0.60)	0.29 (0.11–0.45)	5092.7	770	14.0	0.23	0.50*	0.91	32.6	0	18.6	0	11.4	0	64	48
CRT	0.59 (0.44–0.71)	0.31 (0.14–0.46)	0.49 (0.31–0.64)	0.20 (0.02–0.38)	4902.1	769	13.3	0.27	0.01*	0.99	32.8	0	18.4	0	10.6	0	62	49

In the saturated model the S.D., correlations and means are set free across groups, variables and twins.
$\Delta\chi^2$ for *ACE* model is −2*LL of model *ACE* minus −2*LL of saturated model; $\Delta\chi^2$ of the *AE* model is −2*LL of model *AE* minus 2*LL of model *ACE*, yielding 3 df: the fixed c_c, c'_c and c_s.
Δdfs are denoted as subscripts.
h^2_I and h^2_{II}, heritabilities on test occasion I and II, respectively.
Common *A* and *E* influences from age 16 and 18 (a'_c and e'_c) and specific *A* influences at age 18 (a_s) were examined against the *AE* model.
* The best fitting model.

Table 4
Fit indices for nested sequence of multivariate IQ-RT models fitted to between and within mean product matrices of MZ and DZ pairs (occasion I, age 16)

Model	χ^2	df	P	$\Delta\chi^2$	Δdf	ΔP
1. *ACE* model	2.30	6	0.89			
2. Same as 1, without *C*	6.98	12	0.86	4.68	6	0.59
3. Same as 2, without Raven-RT covariances in *E*	7.11	14	0.93	0.12	2	0.99

Groups: 80 MZ pairs, 108 DZ pairs.
$\Delta P < 0.05$ means a significant change in χ^2.
A, additive genetic variance; C, common environmental variance; E, unique environmental variance.

Table 5
Genetic and environmental correlations between RTs, Raven and heritability estimates with 95% CI based on Model 3

Subtests	Genetic Correlations			h^2	95% CI of h^2	Environmental correlations			e^2	95% CI of e^2
	SRT	CRT	Raven			SRT	CRT	Raven		
SRT	1			0.61	0.46–0.71	1			0.39	0.29–0.54
CRT	0.83	1		0.59	0.44–0.70	0.62	1		0.41	0.30–0.56
Raven	−0.39	−0.36	1	0.58	0.44–0.68	—	—		10.42	0.32–0.56

Table 6
Fit indices for nested sequence of multivariate IQ-RT models fitted to between and within mean product matrices of MZ and DZ pairs (occasion II, age 18)

Model	χ^2	df	P	$\Delta\chi^2$	Δdf	ΔP
1. Four-factor ACE model + specifics e.g. A_G A_V A_P A_{RT} A_{SP}	263.05	250	0.27			
2. Same as 1, without C	315.02	288	0.13	51.9	38	0.07
3. Same as 2, without E_V E_P	330.48	299	0.10	15.5	11	0.16
4. Same as 3, without RT loadings on E_G	331.58	301	0.11	1.10	2	0.58
5. Same as 2: A_G A_V A_P A_{RT} A_{SP}; E_G E_{RT} E_{SP}, without all nonsignificant loadings	333.29	309	0.16	1.7	8	0.99

Groups: 74 MZ pairs, 100 DZ pairs.
$\Delta P < 0.05$ means a significant change in χ^2.
Factors: A, additive genetic factor; C, common environmental factor; E, unique environmental factor.
Factor subscripts: G, general; V, verbal IQ; P, performal IQ; SP, specific.

Table 7
Percentages genetic and environmental variance and heritability estimates with their 95% CI for RTs and WAIS subtests

Subtests	% variance accounted for by genetic and environmental factors												Genetic correlations	
	A_G	A_V	A_P	A_{RT}	A_{SP}	h^2	95% CI of h^2	E_G	E_{RT}	E_{SP}	e^2	95% CI of e^2	STM	CRT
Information	45	13	—	—	18	0.75	0.66–0.82	—	—	24	0.25	0.18–0.35	−0.44	−0.36
Comprehension	30	20	—	—	10	0.59	0.47–0.70	9	—	31	0.41	0.31–0.53	−0.40	−0.33
Arithmetic	49	1	—	—	15	0.64	0.52–0.73	3	—	32	0.36	0.27–0.48	−0.50	−0.40
Similarities	31	23	—	—	—	0.55	0.45–0.64	5	—	41	0.45	0.36–0.55	−0.43	−0.35
Digit span	35	—	—	—	23	0.59	0.44–0.70	—	—	42	0.41	0.30–5.60	−0.44	−0.36
Vocabulary	46	28	—	—	3	0.77	0.68–0.84	1	—	22	0.23	0.15–0.32	−0.44	−0.36
Coding	10	—	—	—	38	0.48	0.31–0.62	—	—	52	0.52	0.38–0.70	−0.26	−0.21
Pic. completion	17	—	—	—	15	0.32	0.15–0.47	17	—	51	0.68	0.53–0.85	−0.41	−0.33
Block design	24	—	44	—	—	0.69	0.58–0.77	3	—	28	0.31	0.23–0.42	−0.34	−0.28
Pic. arrangement	17	—	1	—	13	0.31	0.13–0.45	10	—	59	0.69	0.52–0.87	−0.42	−0.34
Object assembly	7	—	20	—	21	0.49	0.33–0.63	7	—	45	0.51	0.39–0.67	−0.22	−0.18
Simple RT	17	—	—	26	10	0.52	0.37–0.65	—	34	13	0.48	0.35–0.63	1	0.69
Choice RT	11	—	—	20	23	0.54	0.37–0.67	—	13	33	0.55	0.33–0.63	0.69	1

Factors: A, additive genetic factor; E, unique environmental factors.
Subscripts: G, general; V, verbal IQ; P, performal IQ; SP, specific.

indicates that the RT-Raven correlation is entirely mediated by genetic factors, genetic correlations were: -0.39 and -0.36 (Table 5). Heritability estimates with 95% CI are also reported in Table 5. The RT heritabilities are considerable (61 and 59%), almost as high as that of the Raven (58%).

3.4. Multivariate genetic analyses of test occasion II

A model was specified with a general, a verbal IQ, a performance IQ and RT factor in addition to specific factors for each subtest. In order to identify the model, the RT loadings on each specific factor were constrained to be equal. The first model (Table 6) showed a good fit. In subsequent model fittings, the **C** structure, could be dropped (Model 2). In Model 3 the verbal and performal IQ factors in the E matrix could be omitted. It was tested whether the RT-WAIS covariance could be entirely explained by genetic factors. In accordance with the results of occasion I, the loadings of the RTs on E_G were not significant. In the final step all nonsignificant loadings were fixed at zero. Percentages of variance accounted for by the genetic and environmental factors and h^2 estimates with their 95% CI are reported in Table 7. The mean r_g among the verbal subtests (0.74) was higher than among the performance subtests (0.45), whereas the mean environmental correlations were around zero. The mean r_g among the RTs was high (0.69), and so was the mean r_e (0.45) which may in part have resulted from correlated measurement errors. The mean r_g between RTs and verbal was -0.40 and between RTs and performance subtests -0.30.

4. Discussion

Heritabilities for SRT and CRT were considerable on both occasions (48–64%). Heritabilities for verbal WAIS subtest were slightly higher on average (64%) than that for the performance subtests (46%). The mean phenotypic correlation between the RTs and the Raven was within the typically observed range. This correlation was entirely due to genetic influences. Although the RTs correlated lower with the WAIS subtests at occasion II, than with the Raven 2 years earlier, the genetic correlations are considerable. Covariances among WAIS subtests were mainly explained by genetic factors (general, verbal and performance), whereas covariances among RTs were almost equally explained by genetic and environmental factors. The WAIS-RT covariance was entirely mediated by common genetic influences.

The nonsignificant correlations between 'object assembly' (one of the two subtests in which faster performance is rewarded with higher scores) and the RTs support the notion that the SIP-IQ relationship does not seem to be a consequence of the fact that some parts of IQ tests are timed. In another study RT tasks also showed to explain less of the variance of a timed IQ test than that of an untimed administration of the same test [22].

Heritabilities for SRT and CRT were lower on occasion II. It is unclear whether the decrease in both environmental and genetic variance was induced by changes in response strategies caused by modifications of the RT battery: subjects were rewarded for correct responses and encouraged to perform in agreement with target speed values. Mean RTs on both tasks were significantly faster, but the percentages of correctly made trials were significantly lower on occasion II.

In this study, the complete set of variables were included in the multivariate genetic analyses, not composite factor scores. As shown by our results, genetic analyses can yield a quite different pattern than obtained with phenotypic factor analyses. The typically observed Verbal and Performance scales of the WAIS are only expressed in the genetic matrix. The common variance of all WAIS subtests are mainly explained by the general genetic factor and for a small part by the general environmental factor. This method also allows identification of particular aspects of verbal and performance IQ which show higher (genetic) correlations with SIP. Whereas the mean phenotypic correlations between the RTs, verbal and performance subtests were equally high, the RTs showed higher genetic correlations with the verbal subtests, on average. The highest genetic correlations for both RTs were with the subtests information, arithmetic, vocabulary and digit span (a STM task).

Biological determinants of IQ may be translated into neurophysiological and biochemical processes in the central nervous system. In the neural efficiency model of intelligence individual differences in IQ are hypothesized to be attributable to genetic variability in the structure and amount of transmission proteins, which determine speed-of-information-processing. SIP is argued to be an instrument to overcome limiting properties of the working memory and to ensure the correct performance on basic cognitive operations like those involved in IQ tests. In general, genetic studies reveal the importance of genetic effects underlying the established RT-IQ relationship and support this model. In accordance with earlier findings [11,12], the phenotypic RT-IQ correlation in this study, measured at two occasions, was entirely determined by common genetic influences. It is thus possible that common features of both SIP and IQ are determined by the same neurophysiological processes which tap neural speed and efficiency. However, other possibilities have to be considered, e.g. a genetic predisposition to engage in some cognitive activities that increase both SIP and IQ or a causal relationship between the two.

Twin data may be informative for direction of causation tests under certain conditions. This is the case when two correlated traits have different modes of inheritance (e.g. family resemblance is determined by family background for one trait and by genetic factors for the other trait) or if there is a large difference in heritability between the two traits [23]. The RT and IQ data did not meet these criteria. Since, in general, RT and IQ data seem to show rather consistent patterns in genetic architecture and covariance, panel data (measurements on two occasions) may prove useful. However, even when applying data of occasion I and II there was not enough power to distinguish between models predicting either direction of causation.

What is the importance of (small) correlations between IQ and possible biological determinants? Variation in intelligence scores is probably due to multiple factors. This complex trait is unlikely to show a large correlation with any single causal factor. If there is a consistent correlation among a number of biological and psychometric variables, however, and these correlations are shown to be genetic in origin, even small correlations may be of theoretical interest. In addition to the present (and earlier) results, which reveal the genetic basis of the SIP-IQ correlation, the genetic correlation between peripheral nerve conduction velocity and IQ [15] may serve as another example. Genetic analyses are thus considered essential in examining the relationship between biological variables and any complex behavioral trait [24]. Twin studies, therefore, may provide new perspectives in the identification of biological determinants of intelligence.

References

[1] Galton F. Inquiries into Human Faculty and its Development. London: MacMillan, Everyman's library, 1883.
[2] Jensen AR. Reaction times and psychometric *g*. In: Eysenck HJ, editor. A Model for Intelligence. Berlin: Springer-Verlag, 1982.
[3] Jensen AR. Individual differences in the Hick paradigm. In: Vernon PA, editor. Speed of Information Processing and Intelligence. Norwood (NJ): Ablex, 1987.
[4] Vernon, P.A., New developments in reaction time research, In P.A. Vernon (Ed.), Speed of Information Processing and Intelligence. Norwood (NJ): Ablex, 1987:1–20.
[5] Vernon PA. Speed of information processing and general intelligence. Intelligence 1983;7:53–70.
[6] Bouchard TJ, Lykken DT, McGue M, Segal NL, Tellegen A. Sources of human psychological differences: the Minnesota study of twins reared apart. Science 1990;250:223–8.
[7] McGue M, Bouchard TJ, Lykken DT, Feuer D. Information processing abilities in twins reared apart. Intelligence 1984;8:239–58.
[8] McGue M, Bouchard TJ. Genetic and environmental determinants of information processing and special mental abilities: a twin analysis. In: Sternberg RJ, editor, Advances in the Psychology of Human Intelligence, vol. 5. Hillsdale (NJ): Lawrence Erlbaum, 1989:7–45.
[9] Vernon PA. The heritability of measures of speed of information processing. Pers Indiv Diff 1989;10:573–6.
[10] Petrill SA, Thompson LA, Detterman DK. The genetic and environmental variance underlying elementary cognitive tasks. Behav Genet 1995;25:199–209.
[11] Ho HZ, Baker LA, Decker SN. Covariation between intelligence and speed of cognitive processing: Genetic and environmental influences. Behav Genet 1988;18:247–61.
[12] Baker LA, Vernon PA, Ho HZ. The genetic correlation between intelligence and speed of information processing. Behav Genet 1991;21:351–67.
[13] Petrill SA, Luo D, Thompson LA, Detterman DK. The independent prediction of general intelligence by elementary cognitive tasks: Genetic and environmental influences. Behav Genet 1996;26:135–47.
[14] Rijsdijk FV, Boomsma DI, Vernon PA. Genetic analysis of peripheral nerve conduction velocity in twins. Behav Genet 1995;25:341–8.
[15] Rijsdijk FV, Boomsma DI. Genetic mediation of the correlation between peripheral nerve conduction velocity and IQ. Behav Genet 1997;27:87–98.
[16] Van Beijsterveldt CEM, Molenaar PCM, de Geus EJC, Boomsma DI. Heritability of human brain functioning as assessed by Electroencephalography (EEG). Am J Hum Genet 1996;58:562–73.
[17] Raven JC. Standard Progressive Matrices: Sets A, B, C, D, and E. University Printing House, Cambridge. London: M.K. Lewis & Co, 1985.
[18] Stinissen J, Willems PJ, Coetsier P, Hulsman WLL. Manual for the Dutch Translated and Adapted Version of The Wechsler Adult Intelligence Scale (WAIS). Lisse: Swets and Zeitlinger, 1970.
[19] Neale MC. Statistical Modelling with Mx. Department of Human Genetics, Box 3, MCV, Richmond, VA 23298, 1995.
[20] Neale MC, Cardon L. Methodology for Genetic Studies of Twins and Families, NATO ASI Series: Behavioral and Social Sciences. Dordrecht: Kluwer, 1992.
[21] Bouma A, Mulder JL, Lindeboom J. Neuropsychologische Diagnostiek: Handboek. Lisse: Swets and Zeitlinger, 1996.
[22] Vernon PA, Kantor L. Reaction times correlations with intelligence test scores obtained under either timed or untimed conditions. Intelligence 1986;10:315–30.
[23] Heath AC, Kessler RC, Neale MC, Hewitt JK, Eaves LJ, Kendler KS. Testing hypotheses about dierection of causation using cross-sectional family data. Behav Genet 1993;23:29–50.
[24] Jensen AR, Sinha SN. Cerebral glucose metabolism and intelligence. In: Vernon PA, editor. Biological Approaches to the Study of Human Intelligence. Norwood (NJ): Ablex, 1993.