

Genetic influence on the relationship between choice reaction time and IQ: a twin study

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Using the classical twin design, this study investigates the influence of genetic factors on the large phenotypic variance in 8-choice reaction time (CRT), and whether the well-established CRT-IQ association can be explained by a common genetic factor. Subjects were 390 pairs of twins (184 MZ; 206 DZ) with a mean age of 16 years, of whom 49 pairs returned approximately 3 months later for retesting. Mean CRT of correct responses ranged from 279 to 900 ms ($589 \text{ ms} \pm 99$) and a retest reliability coefficient of 0.68 was estimated. Full scale IQ was assessed by the Multidimensional Aptitude Battery (MAB) and ranged from 79 to 145 (111 ± 13). The phenotypic association between CRT and IQ was confirmed (-0.35) and could be attributed entirely to a common genetic factor that accounted for 64% of the variance in CRT and 18% of the variance in IQ. A genetic correlation between CRT and IQ of -0.47 was estimated. A smaller part of the CRT variance (36%) was accounted for by a unique environmental factor, and a specific genetic factor accounted for a further 63% of the variance in IQ with the remainder of the variance (19%) attributed to unique environment. Findings agreed with those of Rijdsdijk, Vernon and Boomsma (1998) in support of a genetic factor mediating the relationship between CRT and IQ.

The relationship between information processing ability and intelligence has been extensively studied for the past 30 years (Hunt, 1980; Jensen & Munro, 1979; Vernon, 1983). The general finding is that performance on reaction time (RT) tasks that make minimal demands on higher order cognitive functions and primarily probe how fast one can carry out simple cognitive operations is negatively correlated with IQ. A range of RT tasks which measure such processes as short term memory scanning, retrieval of information from long term memory and stimulus comparison have been used as cognitive correlates of IQ, but a favoured information processing task which consistently demonstrates significant correlations (around -0.30) with IQ is the choice reaction time (CRT) task - popularised by Jensen (1982, 1987; Jensen & Munro, 1979). This task requires a rapid response to a single stimulus (e.g., appearance of a light) from an array of others. An attempt to identify the specific cognitive process driving the CRT-IQ association has placed the locus at the response choice stage, which directly follows the discrimination of the stimulus and involves selection of the response (Neubauer & Knorr, 1997).

Some findings have shown that the CRT correlation with IQ increases in conditions of higher choice, for instance, 8-choice compared with 2- or 4-choice (Jensen, 1987; Neubauer, 1991; Neubauer & Knorr, 1997; Smith & Stanley, 1983). In Jensen's meta-analysis (1987) of CRT studies, he reports unweighted mean correlation coefficients between intelligence and median CRT of -0.19 , -0.21 , -0.24 , and -0.26 for 1-, 2-, 4-, and 8-choice, respectively. This was based on 15 independent groups comprising 1129 participants. A linear trend analysis of the correlation coefficients across set size was significant, and although this was not consistent when the studies were viewed

individually, it provides some indication that the CRT relationship with IQ is stronger in conditions of higher choice.

The heritability of RT using elementary cognitive tasks was first examined by Vernon (1989) who reported heritabilities ranging from 0.24 to 0.90. In addition, this study showed that the heritability estimates of each RT measure were positively correlated with the mean RT of each measure ($r = .68$). This was interpreted as a complexity effect, such that tasks with slower RTs were more difficult and more heritable than tasks with faster RTs. The phenotypic correlation between RT and IQ was also positively associated with heritability ($r = 0.60$), so that measures which correlated more strongly with IQ were more heritable. Boomsma and Somsen (1991) pursued Vernon's hypothesis that heritability increased with task complexity. Heritabilities were established for RTs from an auditory 2-choice response task with and without a dual task component (mental arithmetic) and with varying interstimulus intervals. They found that conditions using shorter interstimulus intervals (and especially with the dual task) showed increasing genetic influence (heritabilities ranging from 0.18 to 0.48), thus supporting Vernon's (1989) complexity hypothesis.

Only a few studies (Baker, Vernon, & Ho, 1991; Petrill, Luo, Thompson, & Detterman, 1996; Rijdsdijk et al., 1998) have explored the genetic contribution to the variance shared by RT and IQ in an effort to establish a biological link between the processes. The first genetic study to measure the genetic covariance between RT indices (e.g., from memory search, category matching, and dual storage and processing tasks) and IQ found that a common genetic factor explained 49% of the variance in the general RT factor and 64% of the variance in verbal and performance IQs (Baker et al., 1991).

Petrill and colleagues (1996) included CRT (mean RT derived from five CRT conditions: 1, 2, 4, 6, 8) in their multivariate analysis of elementary cognitive tasks and IQ in a sample aged 6 to 13 years. They reported a model which incorporated a genetic speed factor (CRT loading of 0.50), a genetic general factor (CRT loading of 0.19), and a common environment general factor (CRT loading of 0.28) which tapped the IQ subtests better than did the genetic general factor. A univariate analysis of CRT reported earlier by Petrill, Thompson, and Detterman (1995) had also demonstrated the significance of common environment, there being no contribution to the variance from additive genes. These results correspond with findings of increased common environmental effects on specific cognitive abilities and IQ in childhood compared with adulthood (McCartney, Harris, & Bernieri, 1990; McClearn et al., 1997; Wilson, 1986). A more recent study by Rijdsdijk and colleagues (1998) examined the genetic relationship between simple RT, visual 2-choice RT and IQ subtests in a sample of 213 adolescent twin pairs. The correlation between CRT and IQ (-0.22) was completely genetically mediated, with genetic correlations ranging from 0.18 to 0.40. CRT loaded on a general genetic factor composed of IQ subtests (11%), and on a genetic speed factor (20%) which also loaded on simple RT; a specific genetic factor was also apparent (23%).

The present study further examines whether the relationship between CRT and IQ is influenced by a common genetic factor. This study uses an 8-choice RT as part of a psychometric battery to probe information processing speed within an ongoing study of the genetics of cognition in adolescent Australian twins. The principal aim of this larger study is to identify genetic variants that influence individual differences in processing

speed, and working memory, both of which play a significant role in higher level functioning (Wright et al., submitted).

In this paper we use the classical twin method to partition the variance in CRT among individuals into genetic and environmental components, and to measure the extent to which CRT and psychometric intelligence are influenced by the same underlying factors. In this design, the similarity of monozygotic (MZ) twin pairs who share 100% of their genes is compared to similarity of dizygotic (DZ) twins who share roughly 50% of their segregating genes. If the causes of familial similarity are additive genes (transmissible from parent to child), the correlation in performance of MZ co-twins is expected to be twice that of DZ co-twins. An underlying assumption is that environmental influences on cognition are equivalent in MZ and DZ twins (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993).

Method

Participants

Data are collected in the context of the ongoing Brisbane Memory, Attention and Problem-Solving (MAPS) twin study. Here we report data from the first 390 twin pairs tested: 97 MZ female, 87 MZ male, 52 DZ female, 48 DZ male, and 106 DZ opposite sex pairs. Most twins had participated in a melanocytic naevi study two years earlier (Zhu et al., 1999) and others were ascertained through mail-outs to secondary schools in the Brisbane region. Zygosity was determined by ABO, MN and Rh blood groups and by 9 independent polymorphic DNA markers. Twin pairs were excluded if either one had a

history of significant head injury, neurological or psychiatric illness, substance dependence or if they were currently taking long-term medications with central nervous system effects. Participants had normal or corrected-to-normal vision (better than 6/12 Snellen equivalent). The twins were mostly in their penultimate year of secondary school and aged between 15 and 18 years (16.17 years; SD = 0.34). Written informed consent was obtained from the participant, as well as their parent/guardian, prior to testing.

Experimental Protocol

The CRT task and IQ test were part of a psychometric battery, which also included an inspection time task and two reading tests. The session approximated 1.5 hours in length, and was either preceded or followed by a testing session of similar duration which involved the measurement of event-related potentials during a delayed response task. Tests were computer administered in the presence of one of three experimenters. One twin completed the psychometric session while the other completed the alternate session. The order of session testing was counterbalanced between twin pairs based on the birth order of the twins. A full description of the protocol is given in Wright et al (Wright et al., submitted).

A sub-sample of twins (49 pairs) returned for retesting approximately 3 months (1 - 5 months) after their initial test session. All participants approached for retesting agreed. This sample comprised 23 MZ and 26 DZ pairs (57 females, 43 males). An identical battery of tests was administered on both occasions. To minimise confounding effects the participants performed the sessions in the same order on retest.

CRT Task

This task was presented to the participants in the pseudo-game form of dripping taps. The participant's task was to quickly press the appropriate computer key to stop a tap from dripping. The amount of water saved was indicated to the bottom left of the screen. Participants aligned and rested their fingers on the keys Z, X, C, and V (left hand, index finger on V) and the keys M, comma (,), period (.), and slash (/) (right hand, index finger on M) of a standard QWERTY keyboard. Different coloured taps corresponded to the same fingers on both hands to aid tap and finger alliance, for example, the taps matching the index fingers were both red. To familiarise the participant with the response keys, they were initially presented with the eight taps and were required to respond to each tap in a left to right sequence. To minimise between-participant practice and order effects the sequence of choice conditions was fixed in the order of 4, 2, and 8 (Smith & Stanley, 1983). The number of trials presented in each of the two, four, eight choice conditions was 96, 48, and 96, respectively. For all conditions, eight taps appeared on the monitor; those taps in use for the 2- and 4-choice conditions were made salient by brightening their colour. Output measures for each of the choice conditions included the number of correct and incorrect responses, and mean RTs (ms) and standard deviation of correct responses. Individual trials were excluded if RT was less than 150ms or greater than 2000ms. As this is a preliminary analysis of our CRT task, we report only mean RT for the 8-choice condition.

Multidimensional Aptitude Battery (MAB)

A shortened version of the MAB was used which included three verbal sub-tests, (Information, Arithmetic, Vocabulary) and two Performance sub-tests (Spatial and Object Assembly). All sub-tests had a multiple-choice format and were timed at seven minutes each. Participants were not penalised for guessing and were encouraged to answer every item within the time period. Administration and scoring were computerised. Although three composite IQ scores were calculated (verbal, performance, full scale), only full scale IQ will be considered in the current study. The MAB was patterned after the WAIS-R and as a result it possesses good psychometric properties (Jackson, 1984, 1998).

Statistical Analyses

Simultaneous equations, based on the genetic and environmental relationships of MZ and DZ co-twins reared together, were applied to the raw data. Ignoring nonadditive genetic effects, The equations represented in a classical twin design are: $r_{MZ} = A + C$ and $r_{DZ} = \frac{1}{2}A + C$, where A represents additive genetic effects and C represents common environment effects. Nonshared environmental (E) effects that include measurement error are not shared by co-twins but contribute to the total phenotypic variance which may be written $A + C + E$. Such a model assumes that genetic, common environment and unique environment components combine in an additive fashion and that there is random mating in the population. Common environment is distributed independently of zygosity, and of both genetic and unique environment factors (Hopper, 1993).

Initially, means and variances were tested across birth order and zygosity to assess sampling error using the raw data maximum likelihood procedure in Mx 1.50 (Neale,

1997). Sex differences were also tested by specifying a female mean and male deviation from that mean. Firstly, a saturated model is fitted, then progressively simplified models are compared to the full model. The fit of each submodel is compared to the one within which it is nested, by a likelihood ratio chi-square test (Neale & Cardon, 1992). The difference in minus two log likelihood (-2LL) between the models is compared to the critical value ($\alpha = .05$) of the chi-square distribution for the degrees of freedom difference. A non-significant chi-square indicates that there is no difference between the full model and the reduced model.

Maximum likelihood estimates of correlations for each zygosity group were computed with means constrained to be equal across zygosity groups but different between sexes.

The phenotypic correlation between RT and IQ was derived through maximum likelihood prior to fitting a bivariate genetic model, which would partition the sources (A, C and E) and pattern of covariation between RT and IQ. A Cholesky decomposition was applied to the first test data only. This model assumed two factors for each source of variance (A, C and E), where the first factor loaded on both variables, while the second factor was specific to the second variable. Nested AE and CE models were compared to the saturated ACE model using the chi-square difference test. Significance of individual factor loadings was also tested in this manner.

Results

Preliminary analyses

Computer or experimenter error resulted in the loss of IQ data from six participants (0.76%) and CRT data from two participants (0.26%). CRT and IQ were normally distributed. Outliers were identified in the bivariate analysis using the Mx %P function, which calculates likelihood statistics for each family conditional on the model. The output variable of interest was the z-score, based on the Mahalanobis distance of the data vectors (each vector represented a twin pair with scores on CRT and IQ). The z-score was assessed retaining the same criterion of ± 3 standard deviations as a basis for exclusion. Six pairs were outlying and excluded from further analysis.

After removal of outliers, results from the testing of means and variances across birth order and zygosity were non significant in all models. Sex effects were significant for IQ only. The IQ range for females was 79 – 141 (109 ± 12) and for males was 81 – 145 (114 ± 13). In all subsequent analyses sex differences in mean IQ were incorporated in the models. RT ranged from 279 to 900 ms (589 ± 99), with a mean accuracy level of 79%. The maximum likelihood test-retest correlation for CRT was 0.68 and for IQ was 0.90. There were no practice effects in CRT, but a 7 point increase in IQ occurred on retest.

Estimating twin correlations and bivariate model fitting

Maximum likelihood correlations for MZ and DZ groups are presented in Table 1. Co-twin correlations for MZs and DZs were, respectively, 0.62 and 0.42 for RT, and 0.81

and 0.50 for IQ, indicating additive genetic effects for both variables, and the possibility of some shared environmental influences (or assortative mating).

Insert Table 1 here

The maximum likelihood estimate of the phenotypic correlation between RT and IQ was -0.35, indicating that a faster RT was associated with a higher IQ score. The 95% confidence interval extended from -0.28 to -0.42. In accordance with the hypothesis of RT determining IQ and not the reverse, RT was entered first into the bivariate model. Common environment and additive genetic models were compared by dropping all common environment paths for an AE model and similarly all additive genetic paths for a CE model. The change in chi-square was not significant for the AE model (delta chi-square [symbols] (3) = 6.40, $p > .05$), but was significant for the CE model (delta chi-square [symbols] (3) = 49.48, $p < .05$), suggesting that additive genes are an important source of variation and covariation between measures and that shared environment is not. Low factor loadings in the full AE model were tested for significance and dropped where appropriate; only the unique environment path between RT and IQ could be dropped. The standardised parameter estimates of this best-fitting model are depicted in figure 1 as a path diagram.

Insert Figure 1 here

The first additive genetic factor accounted for 64% of variance in RT and 18% of variance in IQ, such that genetic influences which decreased RT increased IQ. In other words, genetic factors almost completely accounted for the phenotypic correlation of -0.35 (i.e., $0.80 \times -0.42 = -0.336$). The genetic correlation between CRT and IQ (the extent to which the genetic deviations in CRT were associated with the genetic deviations in IQ) was -0.47. IQ was also influenced by a specific genetic factor that explained a further 63% of its variance. RT was more affected by unique environment (36% variance) than was IQ (19%). There were no correlated effects of nonshared environment.

Discussion

The significant correlation between CRT and psychometric intelligence is well established and replicated in the current study. We investigated further whether the relationship between CRT and IQ is influenced by a common genetic factor as first reported by Rijdsdijk and colleagues (1998). This was in contrast to an earlier study by Petrill and colleagues (1996) in young children indicating a role of common environment.

We found variances to be equal across birth order and zygosity, with MZ and DZ twin pairs performing similarly for the RT and IQ measures. There were no sex differences in CRT and the range and mean were typical of those ('decision time plus movement time' in CRT tasks using a home key) reported for conditions of higher choice (Jensen, 1987). There was a mean sex difference between males and females in IQ, with males out-performing females. However, because a shortened version of the MAB was

used, this finding may be artifactual since no sex effects have previously been reported for MAB IQ. A future analysis of the IQ subtest scores may elucidate the nature of the sex differences found in our sample. There was an improvement in IQ but not on CRT on retest. Retest studies of the computerised MAB are scarce, but verbal IQ has been shown to increase on repeated administrations, although full scale IQ was not reported (Harrell, Honaker, Hetu, & Oberwager, 1987).

Twin correlations for CRT demonstrated significant familial aggregation. MZ co-twins showed greater similarity ($r = 0.62$) than DZs ($r = 0.42$), and this correlation approached the test-retest correlation (0.68) indicating that the long-term effects of unique environment were not particularly important. In behavioural genetic research the test-retest correlation acts provides an upper limit to the estimate of heritability because the MZ co-twin correlation theoretically cannot exceed the test-retest correlation. For CRT the heritability of 0.64 was almost the same as the test-retest correlation, thus, practically all of the reliable variance in CRT was genetic. The test-retest reliability of CRT was comparable to those reported (e.g., 0.60 and 0.52) in studies which use a retest interval longer than 2 weeks (Barrett, Alexander, Doverspike, Cellar, & Thomas, 1982; Smith & Stanley, 1983).

The heritability we obtained for CRT ($h^2 = 0.64$) was of the same order as that reported by Rijdsdijk et al (1998) ($h^2 = 0.62$) and fell within the range of heritabilities presented by Vernon (1989) for other RT tasks. Confirming previous studies (Jensen & Munro, 1979; Neubauer, Riemann, Mayer, & Angleitner, 1997; Saccuzzo, Johnson, & Guertin, 1994) there was a significant correlation between CRT and IQ (-0.35). Bivariate model fitting indicated that the covariance between CRT and IQ was due to a common

genetic factor, which explained 64% of the variance in CRT and 18% of the variance in IQ. These proportions of variance were similar in magnitude to those reported by Risjdijk et al (1998) and our genetic correlation of -0.47 was also comparable to theirs (-0.37). There was no indication of a common environment factor influencing CRT nor the relationship between CRT and IQ as reported by Petrill and colleagues (1996) in a younger sample of twins (6 – 13 years). In fact, our point estimates indicate the presence of small shared environmental (or assortative mating) effects for both IQ and RT, but our study does not have power to detect them simultaneously with additive genetic effects. Further research within a longitudinal context may be needed to clarify the change in influence of common environment from childhood to adolescence.

Our results showed that genetic influences independent of CRT explain a further 49% of variance in IQ, suggesting that CRT is related to only one of the processes contributing to intelligence. In fact, it has been estimated that hundreds of genes contribute to the genetic variance of IQ (Jinks & Fulker, 1970) probably encompassing many different neuropsychological processes, and we are quite surprised that so much of the variance of such a complex trait can be explained by the same genes that influence the much simpler one of CRT. The remaining variance was composed of nonshared environment, but in the case of RT this was mostly measurement error since the MZ co-twin correlation was almost as high as the test-retest correlation.

The finding of a genetic factor mediating the relationship between RT and IQ supports the claim for a biological basis affecting both processing speed and higher order cognition. Jensen (1998) advocates a neural efficiency model where factors such as oscillation speed of neuronal excitatory potentials and myelination of neurons determine

the speed of information processes. A faster oscillation frequency causes the action potential to be nearer to the threshold of excitation, resulting in a faster response. Increased myelination of neurons might also promote a faster speed and efficiency of information processing since myelinated fibres are responsible for transmitting neural information to differing regions of the brain, and across the corpus callosum. Although these hypotheses have not been directly investigated the results from behavioural genetic studies of information processing indicate that biological avenues of inquiry are worthwhile.

Moreover the rapid advances in molecular genetics means that it may soon be possible to identify specific genes responsible for genetic influence on cognitive abilities. Recently there have been reports of QTLs associated with IQ (Chorney et al., 1998; Fisher et al., 1999) and a long term goal of our work is to extend this approach by using several phenotypes, such as CRT, and combine quantitative genetic information from a number of genetically related measures to substantially enhance the power to detect QTLs for cognition.

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quantitative-trait locus for mole density is linked to the familial melanoma gene CDKN2A: A maximum-likelihood combined linkage and association analysis in twins and their sibs. *American Journal of Human Genetics*, 65, 483-192.

Table 1

Maximum likelihood correlations and 95% confidence intervals for MZ (above diagonal) and DZ (below diagonal) pairs for CRT and IQ. Co-twin correlations for each measure are shown in bold.

<i>Monozygotic twins (n = 180 – 182 pairs)</i>			
	<i>Twin 1</i>		<i>Twin 2</i>
	<i>CRT-1</i>	<i>IQ-1</i>	
			<i>CRT-2</i>
			<i>IQ-2</i>
<i>CRT-1</i>		-.34	.62
		(-.20 to -.46)	(.54 to .70)
<i>IQ-1</i>	-.31		-.38
	(-.18 to -.42)		(.75 to .85)
<i>CRT-2</i>	.42	-.29	-.44
	(.30 to .53)	(-.15 to -.41)	(-.33 to -.54)
<i>IQ-2</i>	-.13	.50	-.32
	(.0 to -.26)	(.39 to .59)	(-.19 to -.44)
<i>Dizygotic twins (n = 201 – 202 pairs)</i>			

Figure 1

Path diagram showing latent genetic and environmental influences on CRT and IQ.

