

## Perceptual speed does not cause intelligence, and intelligence does not cause perceptual speed

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### Abstract

There is ongoing debate whether the efficiency of local cognitive processes leads to global cognitive ability or whether global ability feeds the efficiency of basic processes. A prominent example is the well-replicated association between inspection time (IT), a measure of perceptual discrimination speed, and intelligence (IQ), where it is not known whether increased speed is a cause or consequence of high IQ. We investigated the direction of causation between IT and IQ in 1212 genetically related subjects from Australia and The Netherlands. Models in which the reliable variance of each observed variable was specified as a latent trait showed IT correlations of  $-0.44$  and  $-0.33$  with respective Performance and Verbal IQ; heritabilities were 57% (IT), 83% (PIQ) and 77% (VIQ). Directional causation models provided poor fits to the data, with covariation best explained by pleiotropic genes (influencing variation in both IT and IQ). This finding of a common genetic factor provides a better target for identifying genes involved in cognition than genes which are unique to specific traits.

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Psychophysical approaches to the study of individual differences in intelligence (IQ) have spawned an era of debate on the causality between lower-level and higher-level cognitive processes, with reductionists upholding the view that complex cognition is driven by basic cognitive components (Brody, 2001; Jensen, 1998). But, as Deary (2001) reminds us “It is necessary to prove rather than assume that a cognitive variable is causal to intelligence differences, and it is necessary to test the reverse hypothesis and the possibility that some other variable or variables is causal to both” (p. 168). In this study, the use of genetically informative data enables the unique specification of these alternative models so that their likelihood can be evaluated. We apply this genetic modeling to the association between a basic cognitive process, inspection time, and IQ.

The inspection time (IT) task provides an index of sensory discrimination speed, usually requiring participants to distinguish between two vertical lines that vary noticeably in length. The shortest duration of the stimulus display necessary to make a correct decision at a desired level of accuracy (e.g., 85%) is the IT estimate. A quarter century ago, IT was shown to correlate with IQ; as perceptual speed increases, performance on complex cognitive tasks improves. The most recent meta-analysis of 92 studies ( $N = 4197$ ) reported an uncorrected correlation of  $-0.30$  and a corrected (for sampling error, error of measurement, restriction of IQ range) correlation of  $-0.51$  (Grudnik and Kranzler, 2001). The association between IT and IQ is generally independent of the type of visual stimulus used in the task (Alexander and Mackenzie, 1992; Longstreth et al., 1986), is consistent across age (Anderson, 1986; Nettelbeck and Rabbitt, 1992; Nettelbeck, 1989), and invariant across visual and auditory modalities (Deary et al., 1989).

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Reductionists have appealed to the notion that IT reflects some fundamental property of the brain, such as neuronal transmission speed, which directly affects information processing speed which in turn influences complex cognition through an intermediate cognitive process such as working memory (Jensen, 1993). Critics of the bottom-up processing view suggest that more intelligent individuals are better able to optimize their information processes, employ cognitive strategies, or attend to a repetitive task, thus improving their ITs (Mackintosh, 1986). Some research on cognitive strategies and motivational effects has been undertaken. The most commonly reported strategy is the use of apparent motion cues (i.e., a flicker radiating from the bottom of the shorter line on appearance of the mask), but while cue users were found to have shorter ITs than non-cue users, they did not have accompanying higher IQs (Egan, 1994). Motivational influences have been studied by Bates and Eysenck (1993) and Stough (1996) who demonstrated that personality factors, suggestive of a motivational temperament, did not mediate the IT–IQ relationship.

Bottom-up theorists have also relied on developmental research to advocate their view. For instance, they cite the correlation between infant habituation (i.e., the rate at which an internal representation of a stimulus is formed) and childhood IQ as support for their argument that IT determines IQ, claiming that IT precedes the formation of IQ (Bornstein and Sigman, 1986; Fagan, 1984; Fagan and McGrath, 1981). However, this is not an explicit test of IT as infant habituation is only a proxy for IT. One of the best longitudinal designs to determine direction of causation is the cross-lagged panel design. Using this methodology, Nettelbeck and Young (1990) repeated the measurement of IT and IQ in children after 1 year; finding no differences in the cross-lagged correlations, they concluded that a third factor, akin to general ability, influenced both IT and IQ. Conversely, another longitudinal study in children showed that the first assessment of auditory IT predicted subsequent IQ test scores attained 2 years later, rather than the reverse (Deary, 1995).

An alternative method to establish direction of causation is to use data from genetically related individuals such as monozygotic (MZ; genetically identical) and dizygotic (fraternal) twins (DZ; sharing roughly half of their genes). When two correlated traits display relatively different sources of variation, e.g., variation in one trait is mainly genetic and in the second trait mainly non-genetic, it is possible to resolve the direction of causation between them (Heath et al., 1993). For example, if Trait A is mostly influenced by genes, whereas Trait B is mostly influenced by the common environment, then the direction of causation from A to B predicts the cross-covariance between Twin 1 Trait A and Twin 2 Trait B to be predominantly genetic (i.e., larger MZ than DZ covariance), whereas if B causes A, the cross-covariance will be largely environmental. Different expectations for the co-twin cross-covariances apply as long as the genetic influence on each variable is sufficiently

different in magnitude and if the sample of twins is sufficiently large (Duffy and Martin, 1994).

In this paper, we investigate the direction of causation between IT and IQ by combining the data from studies of IT and IQ in Australia (Luciano et al., 2001) and The Netherlands (Posthuma et al., 2001). Data are available on 2012 subjects, which give reasonable statistical power to address the question of causation.

## 1. Methods

### 1.1. Participants

The Australian sample was recruited from the ongoing Brisbane Memory, Attention and Problem-Solving (MAPS) twin study (Wright et al., 2001). Thirteen families comprised twins with two singleton siblings, but only data from one sibling was included to simplify statistical modeling. Zygosity was determined by ABO, MN and Rh blood groups and by nine independent polymorphic DNA markers. Dutch families were recruited from The Netherlands Twin Registry to participate in a large ongoing project on the genetics of cognition and adult brain function (Boomsma, 1998). A maximum of four singleton siblings per family was imposed to simplify analyses, so the data of five individuals were excluded. Zygosity was determined by DNA fingerprinting.

Exclusion criteria included: history of significant head injury, neurological or psychiatric illness, substance dependence, and long-term medications with central nervous system effects. Participants reported normal or corrected-to-normal vision, and this was better than 6/12 Snellen equivalent in the Australian sample whose vision was checked at the time of testing. Written informed consent was obtained before testing.

### 1.2. Sample characteristics

The study is an extended twin family design, i.e., it uses MZ and DZ twins and one or more of their singleton siblings (see Posthuma and Boomsma (2000) for a detailed description of the extended twin design and its statistical properties). The sample size and family configuration of participants is shown in Table 1, with the total number of families equaling 877 and comprising 2012 individuals. In the Australian sample, twins were mostly in their penultimate year of secondary school and aged between 15 and 18 years ( $16.2 \pm 0.3$  years), while siblings were aged between 15 and 22 years ( $17.2 \pm 1.1$  years). The Dutch sample comprised two cohorts based on the age of the twins (young cohort <36 years; old cohort  $\geq 36$  years): the mean age of the young cohort was 25.8 ( $\pm 2.9$ ) years and the mean age of the old cohort was 49.4 ( $\pm 6.8$ ) years. There was a slight overlap in age of the non-twin siblings between the two cohorts.

Table 1

Family configuration—number of families (fam) and individuals (ss)—by number of non-twin siblings in family ( $N$  ranging 0–4) in the Australian and Dutch samples according to zygosity and age cohort (young vs. old)

	Number of additional non-twin siblings									
	$N = 0$		$N = 1$		$N = 2$		$N = 3$		$N = 4$	
	fam	ss	fam	ss	fam	ss	fam	ss	fam	ss
Australia										
MZ—twin pair + $N$ siblings	208	416	46	138	–	–	–	–	–	–
DZ—twin pair + $N$ siblings	112	224	38	114	–	–	–	–	–	–
DZOS—twin pair + $N$ siblings	128	256	29	87	–	–	–	–	–	–
Total Australia	448	896	113	339	–	–	–	–	–	–
The Netherlands										
Young cohort										
MZ—twin pair + $N$ siblings	37	74	22	66	2	8	1	5	–	–
MZ—singles + $N$ siblings	1	1	3	6	–	–	–	–	–	–
DZ—twin pair + $N$ siblings	19	38	24	72	7	28	–	–	–	–
DZ—singles + $N$ siblings	1	1	4	8	1	3	–	–	–	–
DZOS—twin pair + $N$ siblings	11	22	12	36	2	8	1	5	–	–
DZOS—singles + $N$ siblings	2	2	4	8	1	3	1	4	–	–
No twins	–	–	2	2	2	4	–	–	–	–
Total Dutch—young	71	138	71	198	15	54	3	14	–	–
Old cohort										
MZ—twin pair + $N$ siblings	43	86	18	54	6	24	2	10	–	–
MZ—singles + $N$ siblings	2	2	3	6	–	–	–	–	–	–
DZ—twin pair + $N$ siblings	28	56	16	48	1	4	–	–	–	–
DZ—singles + $N$ siblings	4	4	2	4	1	3	–	–	–	–
DZOS—twin pair + $N$ siblings	10	20	10	30	2	8	–	–	1	6
DZOS—singles + $N$ siblings	3	3	1	2	–	–	–	–	–	–
No twins	–	–	2	2	–	–	1	3	–	–
Total Dutch—old	90	171	52	146	10	39	3	13	1	6
Total Dutch	161	309	123	344	25	91	6	27	1	6
Total combined	609	1205	236	683	25	91	6	27	1	6

Notes: Australian sample includes 12 incomplete sets of IT and IQ data (no IT for 9 pairs, no IQ for 3 pairs and 3 co-twins). Dutch sample is missing IT data for 37 participants.

### 1.3. Measures

#### 1.3.1. Inspection time

An identical IT task was administered for both cohorts (see Luciano et al., 2001). Briefly, it consisted of a line discrimination task requiring the participant to identify the longer of two lines differing in length by pressing the corresponding left or right arrow key on the keyboard (see Fig. 1a). The stimulus duration was variable, ranging between 14.2 and 2000 ms. A flash mask, consisting of two

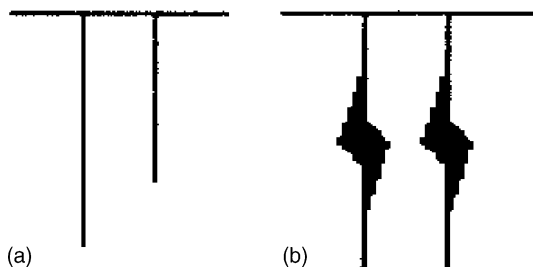


Fig. 1. The inspection time task stimulus (a) is presented briefly, and then hidden by a mask (b).

vertical lines (37 mm) shaped as lightning bolts (see Fig. 1b), immediately followed the stimulus and was presented for a period of 300 ms to limit further stimulus processing (Evans and Nettelbeck, 1993).

The stimulus duration on each trial was altered using a parameter estimation by sequential testing procedure, which allows an efficient estimation from short to long ITs (Findlay, 1978; Pentland, 1980). To minimize bias from random responses and lapses in attention, IT was estimated by fitting post hoc a cumulative normal curve (mean = 0) to accuracy as a function of stimulus onset asynchrony (SOA). The statistic of interest is the standard deviation of the curve, which in this study is the SOA at which 84% accuracy is achieved. Participants with a high proportion of guesses could be identified by their poor fit ( $R^2 < 0.95$ ) to the cumulative normal function. In the Australian sample, 57 participants (4.6%) were excluded on this basis, while none were excluded from the Dutch sample. Inspection time was transformed by a logarithmic function in both samples (note that Posthuma et al. (2001) reported a reciprocal transformation); outliers were removed from the Australian (2.7% of sample) and Dutch (1.4%) datasets. As the procedure for

administering the IT task was the same for both Australian and Dutch cohorts, the much higher proportion of guessing and longer ITs observed for the Australian sample may be a reflection of their younger age and lessened maturity to conscientiously approach the IT task, which is very sensitive to lapses in attention or random responding.

### 1.3.2. Psychometric intelligence

In the Australian sample, a shortened version of the Multidimensional Aptitude Battery (MAB; Jackson, 1998) was used which included three verbal subtests (Information, Arithmetic and Vocabulary) and two performance subtests (Spatial and Object Assembly). In the Dutch sample, IQ was measured with the Dutch adaptation of the Wechsler Adult Intelligence Scale-III (WAIS-III, 1997). The shortened version included four verbal subtests (Information, Similarities, Vocabulary and Arithmetic) and three performance subtests (Picture Completion, Block Design and Matrix Reasoning). While the IQ tests differed between cohorts, there is good evidence to suggest that the MAB and WAIS-R (predecessor to the WAIS-III) tap highly related constructs. The factor structure of the MAB and WAIS-R (excluding the Digit Span subtest) has been compared statistically, with the verbal and performance factors demonstrating correlation coefficients of 0.97 and 0.96, respectively; correlations on the verbal and performance scaled scores were 0.94 and 0.79 (Jackson, 1984).

As the IQ subtests varied between cohorts and because Dutch normative data were not available, Verbal and Performance IQs were calculated as an average percentage correct across the subtests within each scale. Sex and age corrections were factored into the genetic modeling through adjustments to the mean. In the Australian sample, there were four univariate outliers (three verbal, one performance).

## 1.4. Statistical procedure

### 1.4.1. Preliminary analyses

Empirical models examined the equality of means and variances across twin birth order in the Australian sample, and across zygosity and family member type (twin versus sibling) within each country cohort. Co-twin correlations were tested for homogeneity between DZ twin and twin sibling pairings, and between males and females within MZ and DZ same-sex groups. Mean sex and age effects were also tested for significance. Following empirical testing within countries, IT means and variances, and IT and IQ co-twin correlations were tested for equality between countries. Models were estimated by maximum likelihood using the statistical program, Mx 1.51 (Neale et al., 1999), with goodness-of-fit assessed by the likelihood ratio chi-square test ( $\chi^2$ ). Multivariate outliers (four in the Australian sample, none in the Dutch) were identified using the mx%P function, which calculates likelihood statistics for each family conditional on the model (the z-score is based on the

Mahalanobis distance of the data vectors, i.e., twin/sibling pairs with scores on IT and IQ), and were excluded from further analysis.

### 1.4.2. Genetic model fitting

The expected variance–covariance matrix was parameterized in terms of additive genetic (A), common environmental (C) and unique environmental (E) components. Genetic theory predicts the MZ covariance as  $A + C$ , and the DZ covariance as  $(1/2)A + C$ . The covariance between twins and their non-twin siblings was fixed to that of DZ twins. Using Mx 1.51, models were fitted to raw data rather than to variance–covariance matrices since the varying number of singletons in each family resulted in incomplete data. Means and variances were modeled according to the best-fitting empirical models (described earlier). Prior to investigating directional causation models, the heritabilities of IT and IQ (Performance, Verbal) and their genetic covariance were tested for homogeneity between Australian and Dutch cohorts. This involved modeling the covariance between IT and IQ (separately for Verbal and Performance IQ) in terms of a bivariate Cholesky decomposition (see Neale and Cardon, 1992) and estimating A, C and E parameters for each country (note that A, C and E parameters have shown to be equivalent between Dutch young and old cohorts; Posthuma et al., 2001). A submodel which constrained the A, C, and E parameters to equal between the cohorts was assessed by the likelihood ratio chi-square test on nine degrees of freedom. Acceptable fit of this model then allowed direction of causation modeling using the combined Australian and Dutch data.

In the direction of causation (DOC) models, a unidirectional phenotypic pathway was included between IT and IQ, separately for Performance and Verbal IQ (Duffy and Martin, 1994; Neale et al., 1994). Each causal model is nested within the full bivariate Cholesky decomposition of IT and IQ (Verbal/Performance) enabling model comparison using the likelihood ratio chi-square test. Measurement error in the directional causation model will increase estimates of unique environment and therefore bias all other parameter estimates since the expectation for the phenotypic covariance between traits includes the multiplication of the causal parameter by the unique environmental variance in the causal variable (Heath et al., 1993). To avoid this bias, IT and IQ were modeled as latent variables with the standardized pathway leading to the observed variable fixed to the square root of the test–retest correlation ( $r$ ) of each respective measure. Hence, in the DOC model, the causative pathway is included between the latent IT and IQ factors, which are free of measurement error (see Fig. 2). Test–retest correlations had been estimated from a sample of 50 Australian twin pairs who were retested 3 months after their initial visit and are as follows: IT, 0.69; Performance IQ, 0.87; Verbal IQ, 0.89 (Luciano et al., 2001). While these estimates were derived from a subsample of participants, they are consistent with test–retest reliabilities reported in other studies of IT

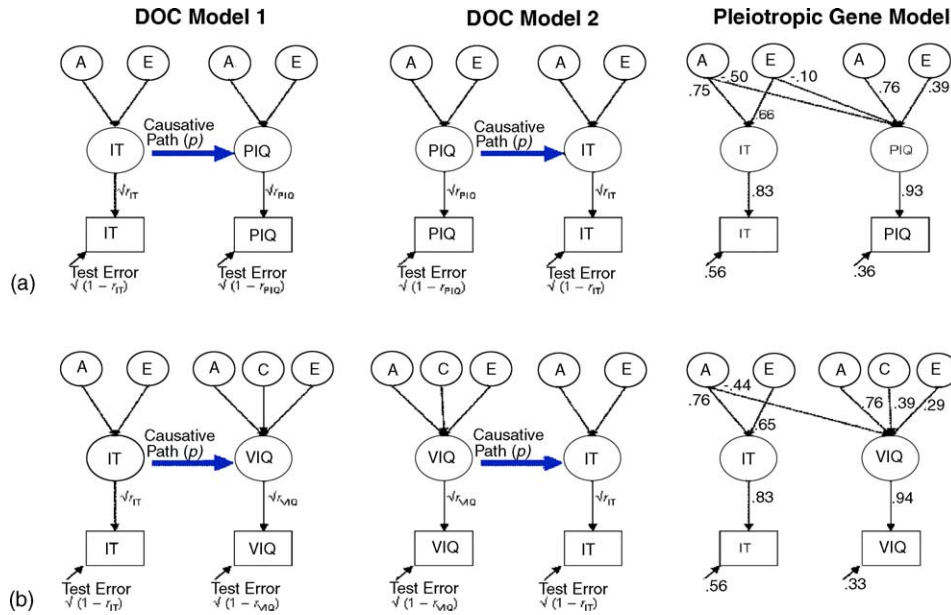


Fig. 2. Path diagrams showing the alternate direction of causation (DOC) models (1 and 2) and the best-fitting genetic model (pleiotropy) for covariation between (a) IT and Performance IQ (PIQ), and (b) IT and Verbal IQ (VIQ). In the standardized DOC models, the relationship between the observed variables, IT and IQ, is modeled in terms of each measure’s reliable variance, i.e., the latent IT and IQ factors are constrained to unit variance, while the pathways leading from the latent factors to the observed variables are fixed to the square root of the test–retest correlation ( $r_{IT}$  and  $r_{IQ}$ ) of each respective measure. Neither DOC models provided good fit to the data ( $p$ -values  $< 0.0001$ ): (a) DOC Model 1,  $\chi^2 = 55.8$  (1 d.f.), DOC Model 2,  $\chi^2 = 54.5$  (1 d.f.); (b) DOC Model 1,  $\chi^2 = 44.9$  (3 d.f.), DOC Model 2,  $\chi^2 = 29.3$  (3 d.f.). Standardized path coefficients are presented for the pleiotropic gene model.

(e.g., Nettelbeck and Rabbitt, 1992) and for the MAB and WAIS-R (Jackson, 1984; Wechsler, 1981), and therefore should be generalisable to the full sample.

**2. Results**

*2.1. Preliminary analyses*

Means and variances were generally found to be equal across twin birth order and zygosity, and between twins and singletons for all measures. Any significant age and sex effects were factored into the genetic modeling as fixed mean effects. As different IQ tests were administered across countries, a between country comparison of means and variances is only relevant for IT. Mean adjustments for age

and sex showed the observed Australian raw score mean IT (71 ms) to be aligned with the Dutch young cohort estimate (74.13 ms), while the age adjusted mean for the Dutch old cohort appeared larger, calculated as 78.45 ms.

In both Australian and Dutch (young and old) cohorts, MZ female and MZ male co-twin correlations were equal for all variables, and likewise, the DZ same-sex, DZ opposite-sex, and twin–singleton correlations could be equated. Furthermore, co-twin correlations within MZ and DZ groups were homogenous between countries. MZ and DZ co-twin correlations and cross-trait correlations for the combined sample are shown in Table 2 and are estimated from a model in which no adjustment for test measure unreliability was made. MZ co-twin correlations were larger than DZ co-twin correlations for all measures and cross-trait associations, indicative of genetic effects. The

Table 2

Test–retest correlations and phenotypic correlations ( $r$ ) between inspection time and IQ are shown; with co-twin correlations and cross-variable correlations presented separately for MZ and DZ groups

	$r$	MZ	DZ	A	C	E
Inspection time (IT) <sup>a</sup>	0.69	0.39	0.19	0.39 (0.57)	0 (0)	0.61 (0.43)
Performance IQ (PIQ)	0.87	0.73	0.36	0.72 (0.83)	0 (0)	0.28 (0.17)
Verbal IQ (VIQ)	0.89	0.81	0.47	0.68 (0.77)	0.14 (0.15)	0.18 (0.08)
IT–PIQ	–0.34 (–0.44)	–0.31	–0.15			
IT–VIQ	–0.26 (–0.33)	–0.31	–0.13			

Notes: Estimates in parentheses have been adjusted for test measurement error. Estimates of additive genetic (A), common environmental (C) and unique environmental (E) proportions of variance, obtained from the bivariate inspection time–IQ Cholesky decompositions, are also displayed.

<sup>a</sup> A, C and E estimates are derived from the bivariate IT–VIQ Cholesky decomposition.



phenotypic intercorrelations between IT and IQ (adjusted for test–retest reliability) were  $-0.44$  for Performance IQ and  $-0.33$  for Verbal IQ (unadjusted estimates were  $-0.34$  and  $-0.26$ ).

## 2.2. Causal genetic modeling

Bivariate additive genetic (A), common environmental (C) and unique environmental (E) Cholesky decompositions between IT and each IQ scale were fitted to Australian and Dutch data separately and then using the same parameters. Both analyses showed A, C and E estimates to be homogenous across countries (IT–Performance IQ,  $\chi^2_9 = 1.87$ ,  $p = 0.99$ ; IT–Verbal IQ,  $\chi^2_9 = 8.61$ ,  $p = 0.47$ ). The parameter estimates (see Fig. 2) show that additive genetic and unique environmental effects are important for each measure, while common environmental influences are substantial for Verbal IQ only. Estimates of heritability and proportions of environmental variance (non-adjusted and adjusted for test measurement error) are shown in Table 2. As the adjusted heritabilities for IQ (0.83 for Performance, 0.77 for Verbal) and IT (0.57) are different, bivariate direction of causation models between (1) IT and Performance IQ and (2) IT and Verbal IQ are tested (see Fig. 2). Their goodness-of-fit is compared to respective AE and ACE full bivariate models (incorporating test measurement error), in which each DOC model is nested. The results of each analysis showed that neither DOC model provided good fit to the data. Since it is conceivable that aspects of IT may influence IQ and vice versa, a reciprocal causation model that allowed causative pathways in both directions was tested. In both analyses, this model also failed to provide acceptable fit to the data ( $p < 0.0001$ ). Thus, the Cholesky decomposition in each analysis gave the most parsimonious account of the data (see Fig. 2 for parameter estimates adjusted for test measurement error). The relationship between IT and Performance IQ was explained primarily by genes with some influence from the unique environment, while for Verbal IQ, the relationship was mediated solely by genes. Genetic effects were correlated  $-0.55$  between IT and Performance IQ, and a genetic correlation of  $-0.49$  was observed between IT and Verbal IQ.

## 3. Discussion

The direction of causation between IT and IQ has been a source of debate amongst proponents of opposing bottom-up and top-down processing theories of cognition (Jensen, 1993; Brody, 2001; Mackintosh, 1986). Whilst bottom-up theorists argue that IT, as a basic processing skill reflecting neuronal functioning, influences variation in IQ, top-down advocates maintain that differences in IT stem from variation in IQ. Our study indicates that neither of the direction of causation models (nor one allowing reciprocal causation effects) sufficiently describe the covariation

between IT and IQ (Performance or Verbal). The Cholesky decompositions of covariance indicated pleiotropic genetic influences between IT and IQ, so that the same set of genes influenced variation in IT ( $\sim 57\%$ ) and IQ (Performance, 25%; Verbal, 19%). These results have been previously discussed (see Luciano et al., 2001; Posthuma et al., 2001). In short, there is no causal relationship between IT and IQ; instead, both processes/abilities are partially dependent on the same underlying cause, which analysis has shown to be genetic.

This finding is in line with that of Nettelbeck and Young (1990), where an equality of cross-lagged panel correlations between IT and IQ in each predictive direction was found. The authors appealed to the notion of a common cognitive ability factor influencing IT and IQ. Within this framework, IT can be interpreted as simply another measure of general cognitive ability, similar to IQ subtests. Our study, which uses an alternative research design to that of Nettelbeck and Young, therefore confirms that IT neither causes variation in IQ nor is it a consequence of one's IQ. Nettelbeck (2001) suggests that the psychological processes underpinning IT involve focused attention and decision processes used in response monitoring. We speculate that these same processes are required for the successful completion of IQ subtests irrespective of their informational content, and hence the generality of effects. The possibility also exists that IT taps a broad perceptual speed group factor rather than a general ability factor, both of which have been supported in factor analytic studies of IT and IQ subtests (Crawford et al., 1998; Mackintosh, 1998). But a recent genetic analysis of IT, choice RT and five IQ subtests including Digit Symbol in the Australian twin dataset used here indicated that most of the genetic variance in IT was explained by genes influencing general ability and not the perceptual speed/organization factor (Luciano et al., 2004).

What is the nature of the individual differences in brain structure and function underlying both IT and IQ? A single fMRI study of IT exists to date (Deary et al., 2001), and this pilot study showed that the patterns of brain activation during IT task performance were aligned with those reported for complex cognitive tasks, such as the Raven's Matrices and Wisconsin Card Sorting Test. The areas of overlapping activation between the studies included the cingulate gyrus of the limbic lobe and the inferior, medial and superior frontal gyri of the frontal lobes. An imaging study in which both IT and higher-order cognitive tasks are sampled on the same individuals is required to confirm these preliminary findings. Neurotransmitters that have been implicated in IT via pharmacological studies that systematically manipulate neurochemical systems include acetylcholine, nicotinic receptors and muscarinic receptors, while serotonin, noradrenaline and dopamine have not yet received support (Stough et al., 2001). Stough et al. (2001) speculate that the cholinergic system contributes to the observed relationship between IT and IQ. The cholinergic corticopetal projection system (projecting from basal forebrain areas to cortical

areas and layers) has been hypothesized to affect detection, selection and discrimination of sensory input (Sarter and Bruno, 1997). Indeed, an association of the cholinergic muscarinic receptor 2 (CHRM2) to WAIS-R IQ scores and years of education has been reported by Comings et al. (2003), whose regression analysis uncovered 1% of overlapping variance between genotype and each cognitive phenotype.

As IT is less cognitively complex than IQ, it may prove simpler to model biologically, and hence present a more tractable framework for the selection of other candidate genes influencing cognitive ability. Part of the common genetic factor influencing IT and IQ may represent genes coding for basic structural aspects of neural wiring like connectivity, myelination, number of ion channels and speed of synaptic transmission (Jensen, 1998). Posthuma et al. (2001) suggested that genes related to central nervous system axonal conduction would be good candidates for genetic association studies of IT, as factors such as fibre diameter of the axon, the number and form of ion channels in the axon membrane, and the quality of the myelin sheath (generated by the oligodendrocytes) determine the optic nerve conduction velocity and conduction velocity from lateral geniculate nucleus neurones to cortical areas. While other aspects of visual processing are bound to affect the variation of IT, these may not necessarily overlap with IQ, which may explain the moderate genetic correlations observed between IT and IQ. Nonetheless, the results of the present study suggest that various candidate genes selected to play a role in IT will also be important for IQ. This may be more so for Performance than Verbal IQ, which demonstrated a slightly larger genetic correlation with IT, and hence an increased reliance on the same genes. Our own planned association studies of IT will benefit from the potential for replication studies between Australian and Dutch cohorts, in which the same candidate genes will be typed.

The finding of a moderate IT heritability and largely genetic mediation of its phenotypic covariation with IQ in cohorts from two different countries has positive implications for the gene finding strategy of linkage. Firstly, the equality of findings across countries suggests that further data pooling incorporating additional countries may be possible and this will greatly increase statistical power for linkage. Secondly, the multivariate linkage analysis of IT and IQ should reduce the number of sib pairs required to detect linkage (Boomsma and Dolan, 2000; Williams et al., 1999); although the gain in power by multivariate methods is greatest if the QTL is the only cause of covariation between the traits (Martin et al., 1997). While it is unlikely that a single QTL accounts for the total covariation between IT and IQ, multivariate analysis will nonetheless improve power relative to the univariate case. Future analyses could therefore be directed toward a molecular genetic analysis of IT and IQ in a combined sample of DZ twin and/or sibling pairs from diverse research groups.

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