

# Genetics and general cognitive ability (*g*)

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**Two recent articles in this journal made the case for the existence and importance of *g* and reviewed research on cognitive and psychophysical correlates of psychometric *g*. This review considers *g* from a genetic perspective. Multivariate genetic research indicates that *g* accounts for nearly all of the genetic variance of diverse psychometric cognitive tests (genetic *g*). Recent research suggests not only that elementary cognitive tasks are genetically linked to psychometric *g* but also that genetic *g* pervades these tasks. Contrary to the assumption of modularity that dominates cognitive science, genetic *g* exists in the mind as well as in psychometric tests.**

Inter-individual differences in performance on diverse psychometric tests of cognitive abilities – such as verbal, spatial, memory and processing speed – intercorrelate about 0.30 on average and a general factor (an unrotated first principal component) accounts for about 40% of the total variance of these psychometric tests [1]. *g* is not the whole story of cognitive abilities – group factors representing specific abilities also represent an important level of analysis – but trying to tell the story of cognitive abilities without *g* loses the plot entirely. A recent article in *TICS* made the case for the existence and importance of *g*, which is sometimes called ‘intelligence’ [2]. A companion paper described the cognitive and psychophysical correlates of psychometric *g* [3]. In this review, we consider *g* and its cognitive correlates from a genetic perspective. By ‘genetic’, we mean ‘heritable’ (inherited DNA differences among individuals) rather than ‘innate’ (evolutionarily constrained differences among species).

A stronger case has been made for substantial genetic influence on *g* than for any other human characteristic. Dozens of studies including more than 8 000 parent-offspring pairs, 25 000 pairs of siblings, 10 000 twin pairs, and hundreds of adoptive families all converge towards the conclusion that genetic factors contribute substantially to *g* [4]. Estimates of the effect size, called heritability (see Ref. 4 for explanation), vary from 40 to 80% but estimates based on the entire body of data are about 50%, indicating that genetic variation accounts for about half of the variance in *g*. Breaking down the research by age, heritability can be seen to increase almost linearly from infancy (about 20%) to childhood (about 40%) to adulthood (about 60%) [5]. Genetic research has moved beyond merely estimating heritability. One new direction that is the focus of the present review is multivariate genetic analysis, which provides important new insights into the structure of cognitive processes linked to *g*.

## Multivariate genetic analysis of psychometric tests

A technique called multivariate genetic analysis examines the extent to which genetic and environmental factors mediate the phenotypic covariance between variables. It also yields a statistic called the ‘genetic correlation’, which is the extent to which genetic effects on one trait correlate with genetic effects on another trait independent of the heritability of the two traits. That is, although all psychometrically assessed cognitive abilities are moderately heritable, the genetic correlations between cognitive abilities could be anywhere from 0.0, indicating complete independence, to 1.0, indicating that the same genes influence different cognitive abilities. Multivariate genetic analysis is described in Box 1.

Multivariate genetic analyses of psychometric tests consistently find that genetic correlations are very high – close to 1.0 – in adolescence and adulthood [6,7]. That is, if a gene were identified that is associated with a particular psychometrically assessed cognitive ability, the same gene would be expected to be associated with other cognitive abilities as well. In other words, although psychometric *g* accounts for about 40% of the total phenotypic variance of diverse cognitive tests, genetic *g* accounts for nearly all of the genetic variance.

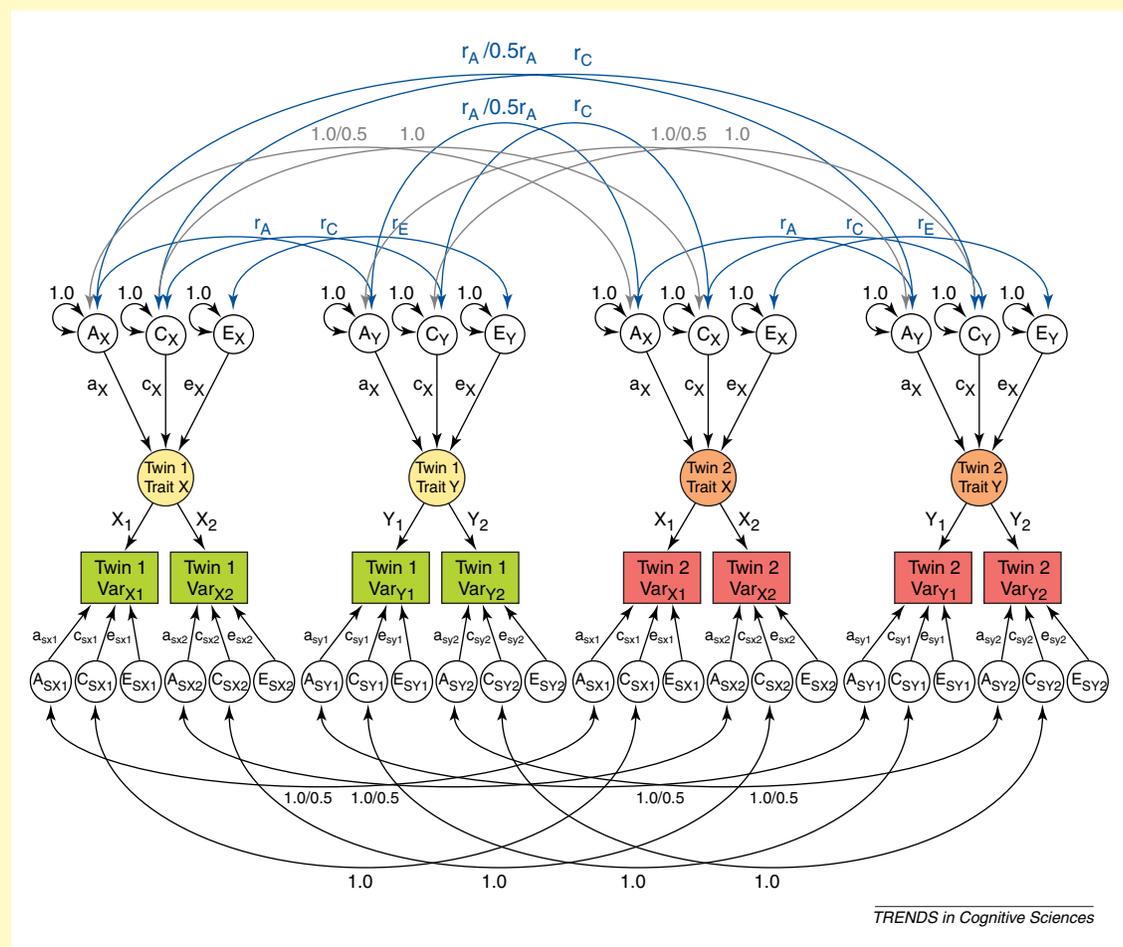
## Models of cognitive mechanisms and genetic *g*

Can genetic *g* as inferred from psychometric tests be explained in terms of more basic cognitive mechanisms? Although it might seem obvious to cognitive scientists that the answer is yes, there is a problem. In cognitive science, it is generally assumed that cognitive processes are independent or modular. Although Fodor’s original concept of modules as innate and invariant information-processing units [8] has been watered down to the notion of domain specificity, it remains a pervasive view in cognitive science from older lesion studies and newer neuroimaging research that cognition consists of many discrete and independent processes [9–11]. How can modular cognitive processes explain such a molar outcome as genetic *g*?

In cognitive science, modularity is considered from a normative perspective, focusing on species-typical processes as assessed for example by neuroimaging or by disrupting normal processes with drugs, lesions or knockout genes. The provenance of *g*, however, is individual differences – why people differ in their

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## Box 1. Multivariate genetic research



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performance on cognitive tasks. The normative and individual differences perspectives represent different levels of analysis (i.e. means and variances) and there is no necessary connection between them [12]. Thus, there is no paradox if the normative theory of modularity does not apply to individual differences. However, if the construct of modularity were to be extended to consider individual differences, it would predict that genetic correlations among cognitive processes should be low, which would conflict with the high genetic correlations found among psychometric tests of cognitive abilities.

The simplest solution to this apparent paradox is to posit that a single module, uncorrelated with other modules, is responsible for psychometric  $g$  (Model 1 in Fig. 1). As discussed below, the candidate most often studied is processing speed. Although at first glance, Model 1 appears reasonable, upon reflection it seems highly unlikely that a fundamental process such as processing speed could be responsible for  $g$  and yet be uncorrelated with all other cognitive processes.

A second solution lies in the hypothesis that many independent elementary cognitive processes contribute to performance on complex psychometric tests of cognitive abilities (Model 2 in Fig. 1). That is,

psychometric tests may be so complex that they enlist many independent elementary cognitive processes, thus creating phenotypic and genetic correlations among the psychometric tests, even though the elementary cognitive processes are themselves uncorrelated. This model, which could be called the standard model of cognitive psychology, has led to research on presumably independent elementary cognitive processes and their relationship to  $g$  [13–17].

A third possibility is that many different elementary cognitive processes contribute to psychometric tests but that these elementary processes are not independent (Model 3 in Fig. 1). The implication of this model is that psychometric  $g$  reflects  $g$  that exists among elementary cognitive processes. In other words,  $g$  exists in the mind as well as in psychometric tests. Multivariate genetic analysis is a crucible for testing these models at the level of genetic and environmental etiologies of individual differences.

#### Multivariate genetic analyses of cognitive mechanisms and their relationship to genetic $g$

The *TICS* review mentioned earlier [3] considered phenotypic associations between psychometric  $g$  and

Any behavioral genetic design such as the twin method that can be used to decompose the variance of a trait into genetic and environmental components can also be used to decompose the covariance between two or more traits [a]. Although model-fitting multivariate genetic analyses look complicated (see Fig. 1), they are in essence simple [b]. In the standard univariate twin analysis, one twin's score on X is correlated with the other twin's score on the same trait X. Genetic effects on the variance of X (heritabilities) are estimated by the extent to which the twin correlation for identical twins (monozygotic, MZ) exceeds the correlation for fraternal twins (dizygotic, DZ). For multivariate twin analysis, instead of correlating one twin's score on X with the other twin's score on X, one twin's score on X is correlated with the other twin's score on Y. Genetic mediation of the phenotypic covariance between X and Y is estimated by the extent to which this cross-twin correlation is greater for MZ than for DZ twins. Estimates of heritability have wide confidence intervals unless samples are large. Estimates of genetic mediation of the

**Fig. 1.** Multivariate genetic analysis. Path diagram describing a latent multivariate genetic ACE model (see text). The paths between twin siblings for the same trait are depicted in grey; paths for different traits are shown in blue. The measured variables  $X_1$  and  $X_2$ , and  $Y_1$  and  $Y_2$  are shown as rectangles for twin 1 and twin 2. These are assigned individual weights ( $x_{1/2}$ ,  $Y_{1/2}$ ) and combined into a latent variable for each twin, labelled Trait X and Y, respectively. (Note that multivariate models do not necessarily have to include latent variables. We have chosen to present a latent model for reasons of comparability with the analyses reported in Box 2.)

phenotypic covariance have even wider confidence intervals because these multivariate analyses decompose phenotypic covariance rather than total variance.

In multivariate genetic models the variance of the latent traits X and Y is dissected into additive genetic (A) variance, common or shared environmental (C) variance, and non-shared environmental (E) variance by comparing MZ and DZ twins – called the ACE model. MZ twins correlate 1.0 genetically because they are genetically identical. DZ twins, assuming a model of additive genetic effects, correlate 0.50 genetically as do their first-degree relatives. This is shown in the path diagram by '1.0/0.5' connecting A for the two twins. The model also indicates that both MZ and DZ twins completely share C because they are reared together. This is shown as '1.0' connecting C for the two twins. Remaining variance of the latent traits X and Y is attributed to non-shared environment (E). Because E does not make the two twins similar, no connection is shown for E between the two twins. Expected correlations for MZ and DZ twins can be constructed from this path model and used in maximum-likelihood structural equation analysis to estimate phenotypic variance due to genetic factors (heritability) and variance due to shared and non-shared environment. The path coefficients ( $a_x$ ,  $c_x$ ,  $e_x$ ) are standardized partial regressions and the variance explained by these components is the square of the path coefficients. Thus, heritability of trait X is represented as  $a_x^2$ . The variance of the latent trait X reflects common variance among the measured variables  $X_1$  and  $X_2$ . The specific (residual) variance of each measure is further dissected

into specific genetic ( $A_{Sj}$ ) and specific shared ( $C_{Sj}$ ) as well as non-shared ( $E_{Sj}$ ) environmental influences and the respective standardized path coefficients are denoted as  $a_{x_{Sj}}$ ,  $c_{x_{Sj}}$ , and  $e_{x_{Sj}}$ . Hence, the heritability of measure  $X_1$  can be expressed as  $(a_{x_1}^2) \times (x_1^2) + (a_{x_2}^2)$ .

The main strength of the multivariate model involves the decomposition of the covariance between latent traits X and Y, indicated by blue paths in the figure. The connection between  $A_x$  and  $A_y$  is a function of the 'genetic correlation' ( $r_A$ ) between X and Y. The genetic correlation represents the extent to which genetic effects on X correlate with genetic effects on Y. The genetic contribution to the phenotypic correlation between latent X and Y within one twin sibling is the product of the paths  $a_x r_A a_y$  and is often referred to as 'bivariate heritability'. Similarly, shared and non-shared environmental connections between X of one twin and Y of the other twin are weighted by  $r_C$  and  $r_E$ , respectively. Across twin siblings the genetic contribution to the phenotypic correlation between latent X and Y depends on zygosity: for MZ twins it is the product of  $a_x r_A a_y$  whereas for DZ twins it is the product of  $a_x 0.5r_A a_y$ .

Structural equation analysis of the entire model represented in the path diagram indicates the overall fit of the model to a particular set of data and provides maximum-likelihood estimates of these parameters and their standard errors.

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measures from experimental cognitive psychology that typically assess reaction time (RT) in response to manipulation of the stimulus–response relationship as well as psychophysical measures that assess processing of sensory information without a reaction-time component. In this section, we follow up on this review of candidate cognitive mechanisms from a genetic perspective. Specifically, we focus on issues of heritability, genetic correlations with psychometric  $g$ , and genetic correlations between the cognitive processes themselves. It should be mentioned at the outset that there is little evidence that attempts to devise a test that assesses a single elementary process have been successful, perhaps because of the pervasiveness of  $g$ .

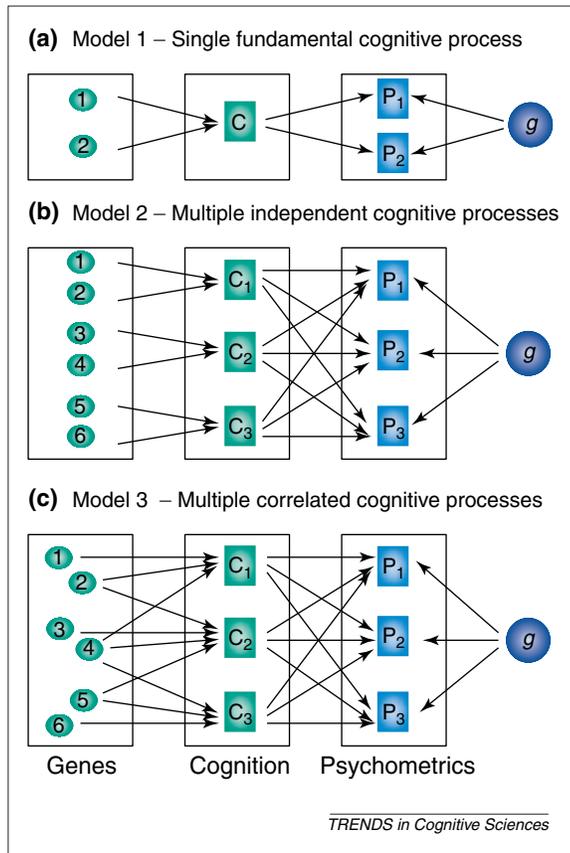
#### *Reaction-time measures: speed of information processing*

Reaction-time measures of processing speed have been of special interest because such measures appear to be elementary and quite different from psychometric tasks. Although psychometric tests include tests that involve speed of processing such as coding digits for symbols, comparing lists of letters

for differences, and simple arithmetic, reaction-time measures make it possible to decompose cognitive tasks into specific components of processing such as decision time and movement time. From dozens of studies using various reaction-time measures, it is clear that faster and less variable reaction times are correlated with  $g$  [3]. Two problems have emerged from this research. First, correlations between reaction time and  $g$  are modest, typically in the range of  $-0.20$  to  $-0.30$  [18], although a recent study of 900 middle-aged individuals yielded somewhat higher correlations [19]. (The negative correlation indicates that faster reaction times are related to higher  $g$  scores.) Second, the theoretically most interesting components of the tasks, such as the increase in decision time as a function of increasing bits of information, do not correlate any better with  $g$  than do simple reaction time and variability of reaction time.

As an example of research in this area, the German Observational Study of Adult Twins (GOSAT; [20]) included 169 monozygotic (MZ) and 131 dizygotic (DZ) adult twin pairs who were assessed on a battery of diverse psychometric tests of verbal, spatial and reasoning abilities as well as

**Fig. 1.** Models of the relationship between genes (1, 2, 3, ...), cognition ( $C_1, C_2, \dots$ ), psychometric tests ( $P_1, P_2, \dots$ ) and psychometric  $g$ .



reaction-time measures of speed of processing [21]. The GOSAT twin study included two widely used elementary cognitive tasks (ECTs), Sternberg's memory scanning task and Posner's letter matching task, which were designed to assess speed of accessing short-term and long-term memory, respectively (see Fig. 2). Several scores from the two measures were derived that were highly reliable and yielded age- and sex-adjusted correlations of about  $-0.30$  with  $g$ . Similar to the literature mentioned above, the most theoretically interesting component of each task (slope for the Sternberg task and the difference score for the Posner task) correlated least with  $g$  (correlations of  $-0.03$  and  $-0.19$ , respectively). Therefore, the analyses concentrated on RT measures. If the authors had used the theoretically more interesting Sternberg slope and Posner difference scores, the absence of a phenotypic correlation with  $g$  would have vitiated a multivariate genetic analysis. Moreover, the focus of these analyses is on  $g$ -related cognitive processes and these theoretically more interesting scores simply are not related to  $g$ .

Heritability estimates increased with task complexity [21], which replicates results from an earlier study that used different reaction-time measures [22]. For the Sternberg task, heritability estimates from full ACE models for set sizes of 1, 3 and 5 are, respectively, 0%, 35% and 47%. For the Posner task, heritability was 24% for matching physically identical letters and 60% for the more

difficult condition of matching letters based on their name identity despite the letters' physical differences. These heritability estimates are typical of those found for ECTs – it is noteworthy that these so-called 'biological' variables show no greater heritability than psychometric tests of cognitive abilities [23].

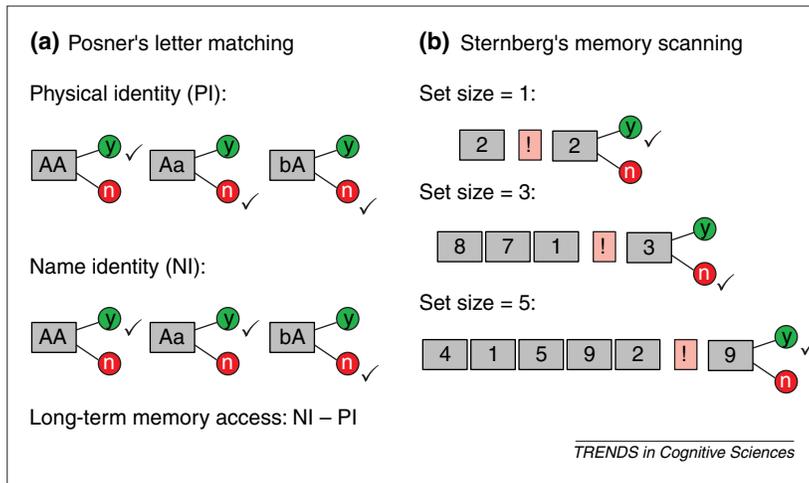
The key issue is their genetic correlation with psychometric  $g$ . For these analyses, a general ECT score was obtained by extracting the first unrotated factor from a principal axis factor analysis of the age- and sex-corrected RT measures for the three set sizes in the Sternberg task and for the physical and name identity procedures of the Posner task. This factor explained 58% of the total variance. Bivariate genetic analysis between this ECT composite and a psychometric  $g$  factor yielded a genetic correlation of  $-0.50$ , indicating substantial genetic overlap [21]. These results are similar to those of an earlier study of 82 MZ and 109 DZ twin pairs assessed at 16 and again at 18 years of age that yielded genetic correlations of about  $-0.40$  for the same two tasks as they relate to  $g$  [24].

#### Latent variable analysis

Box 2 presents results (previously unpublished) of a more powerful latent variable analysis in which the Sternberg and Posner tasks were used to index a general ECT latent variable and two intelligence tests were used to index a latent variable representing psychometric  $g$ . 83% of the association between the latent variables representing ECT and psychometric  $g$  is mediated genetically and the genetic correlation between the latent variables is  $-0.67$ . These results indicate substantial genetic overlap between processing speed from the two tasks and psychometric  $g$ .

These results are not consistent with Model 1 (Fig. 1) in that speed of processing from the two ECTs cannot completely account for genetic influence on psychometric  $g$ . However, the results are consistent with either Models 2 or 3. The latent bivariate genetic analysis (Box 1, Fig. 1) can distinguish between Models 2 and 3 by examining the association between the Sternberg and Posner tasks. These results clearly support Model 3 rather than Model 2 in that two-thirds of the genetic variance in the Sternberg task and all of the genetic variance of the Posner task is shared in common. A bivariate genetic analysis of the two tasks (not shown) yields a genetic correlation of 0.84, indicating substantial genetic overlap between these tasks.

Other twin studies have shown that choice RT overlaps genetically with psychometric  $g$ . Choice RT assesses speed of response to the appearance of a target stimulus from an array of other stimuli. For example, two-choice RT could involve responding when the left light of two lights is lit. The twin study mentioned earlier also included two-choice RT which was substantially heritable (about 50%) and its



**Fig. 2.** Elementary cognitive tasks. (a) In the Posner letter-matching (PLM) task, it is assumed that a correct response (indicated by a tick) in the physical identity (PI) condition requires visual discrimination only, whereas in the name identity (NI) condition additional access to long-term memory stores is required. Thus, a simple difference score, (mean reaction time NI) – (mean reaction time PI), has been suggested to index long-term memory retrieval rate [48]. (b) In the Sternberg memory-scanning (SMS) task, the slope of the regression of RT on the set size is assumed to reflect the time required to retrieve a single element from short-term memory.

modest phenotypic correlation ( $-0.22$ ) with an IQ test (Raven's Progressive Matrices) was completely mediated genetically although the genetic correlation was only  $-0.36$  [24]. These findings were replicated in a study of choice RT that included 2, 4 and 8 choices in 390 pairs of young adult twins, although the phenotypic correlations with IQ were higher ( $-0.32$ ,  $-0.55$ , and  $-0.48$ , respectively) [25]. Genetic factors almost completely mediated phenotypic correlations with IQ and the genetic correlations with IQ were  $-0.41$ ,  $-0.71$  and  $-0.58$ , respectively, for two-, four-, and eight-choice RT. Multivariate genetic analysis of the three-choice RT conditions yielded genetic correlations of about 0.80, indicating substantial genetic overlap among the conditions. Two earlier twin studies used non-standard RT measures. One study of 287 twin pairs from 6 to 13 years old found negligible heritability (0.03) for a simple RT task and moderate heritabilities of 0.35 and 0.42 for more complex RT tasks [26]. A multivariate genetic analysis suggested some genetic overlap (a general genetic factor) between complex RT tasks and psychometric  $g$  [27]. A study of RT measures in adolescent twins (50 MZ and 52 DZ) yielded higher than usual genetic correlations near 1.0 between RT measures and psychometric  $g$  [28].

In summary, speed of information processing is highly heritable as assessed using the Sternberg and Posner tasks and choice RT. Multivariate genetic analyses indicate that their phenotypic relationship with  $g$  is largely mediated genetically. However, the results do not fit Model 1 because genetic correlations with  $g$  are considerably less than unity in most studies. Multivariate genetic analyses also indicate that these tasks are highly correlated genetically with each other, results that support Model 3 rather than Model 2.

#### *Psychophysical measures: inspection time*

Even more elementary than reaction-time measures are measures of speed of processing sensory information. The most widely used task is inspection time which involves a simple visual judgment of the length of two lines in which the target stimulus is followed by a mask at a varying interval from a few milliseconds to a few hundred milliseconds [3]. Although inspection time appears to be a more elementary process than RT measures, it correlates even higher with  $g$ , about  $-0.40$  in a recent meta-analysis [29]. In a study of 390 pairs of young adult twins, the phenotypic correlation between inspection time and  $g$  was largely mediated genetically and the genetic correlation was  $-0.63$  [30], results replicated in a study of 688 family members (twins and siblings) in 271 families [31].

Thus, as was the case for the Sternberg and Posner tasks and choice reaction time, inspection time correlates genetically with psychometric  $g$  but the results do not fit Model 1 because the genetic correlation is less than unity. A multivariate genetic analysis of the phenotypic correlation of about 0.30 between two-choice RT and inspection time yielded a genetic correlation of 0.35 [32], considerably lower than the genetic correlations found between the Sternberg and Posner tasks (0.84) and between conditions of choice RT (0.80). Although this finding of a modest genetic correlation between two-choice RT and inspection time needs to be replicated, it is possible that inspection time overlaps less with the other tasks which involve RT. Nonetheless, this finding provides some additional support for Model 3 rather than Model 2.

#### *Psychophysiological measures: EEG and ERP*

Although not included in the previous *TICS* review (but see Refs [33,34]), psychophysiological measures have also been the target of genetic analyses. Peripheral nerve conduction velocity [35,36], EEG alpha peak frequency [37] and EEG coherence (a measure of brain interconnectivity [38–40]) are all highly heritable but correlate only marginally with  $g$ . Thus,  $g$  does not seem to involve speedier brains, at least as assessed by these psychophysiological measures.

#### *Working memory*

At the other end of the continuum from elementary cognitive processes is working memory. The previous *TICS* review [3] began by mentioning the massive literature on working memory, which is assessed in diverse ways but with essentially psychometric types of tests. It is increasingly clear that various measures of working memory correlate with  $g$  near the reliability of the measures [41–44]. This could mean that working memory is the Factor X that explains  $g$  (Model 1 in Fig. 1). However, it seems more likely that working memory is just another name for  $g$  – tests of working memory look suspiciously like psychometric tests of  $g$  [3].

## Box 2. Genetic overlap between psychometric tests and elementary cognitive tasks (ECTs)

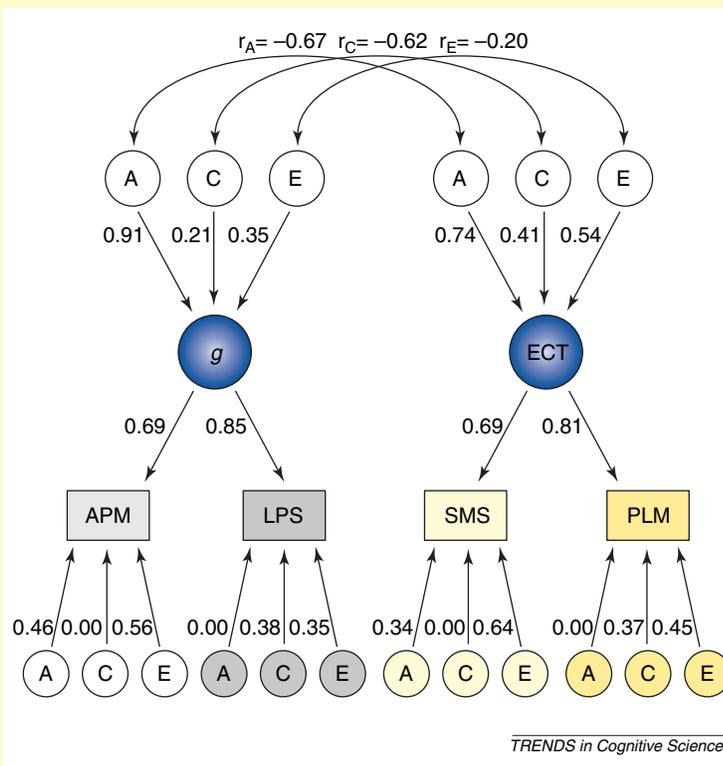
Fig. 1 depicts results (previously unpublished) from the GOSAT twin study. At the top of the figure are path coefficients representing additive genetic (A), common or shared environment (C) and non-shared environmental (E) contributions to the phenotypic correlation between the latent variables  $g$  and elementary cognitive tasks (ECT). (For reasons of space, this path diagram is shown for one individual rather than for both members of the twin pair as in Box 1.) The genetic correlation of  $-0.67$  indicates substantial genetic overlap

between  $g$  and ECT. Shared environmental influences displayed a similar degree of overlap ( $0.62$ ) whereas non-shared environmental influences on  $g$  and ECT correlated to a lesser extent ( $0.20$ ). Bivariate heritability, the genetic contribution to the estimated correlation between  $g$  and ECT, is calculated as  $0.91 \times (-0.67) \times 0.74 = -0.45$ . In a similar manner the contribution of shared environmental influences [ $0.21 \times (-0.62) \times 0.41 = -0.05$ ] and non-shared environmental influences [ $0.35 \times (-0.20) \times 0.54 = -0.04$ ] can be derived from the model. The

sum of these three mediating influences equals the estimated correlation between latent  $g$  and ECT which is  $(-0.45) + (-0.05) + (-0.04) = -0.54$  for the present analysis. This implies that 83% of the phenotypic correlation is mediated genetically ( $-0.45/-0.54$ ). Shared and non-shared environmental influences contribute substantially less to the phenotypic correlation between  $g$  and ECT despite the large overlap of shared environmental influences on  $g$  and ECT.

Thus,  $g$  and ECT are highly correlated genetically and the genetic overlap accounts for most of the phenotypic correlation between the two latent variables. What about the genetic correlation between the psychometric APM and LPS tests and, especially, between the ECTs (SMS and PLM)? These results are indicated at the bottom of the figure. The path of  $0.00$  from A to LPS indicates that there is no genetic variance on LPS that is independent of APM. Similarly, the path of  $0.00$  from A to PLM indicates that there is no genetic variance on PLM that is independent of SMS. Separate bivariate genetic analyses (not shown) estimate a genetic correlation of  $0.92$  between APM and LPS and a genetic correlation of  $0.84$  between SMS and PLM.

These results are representative of multivariate genetic analyses showing that psychometric tests are highly correlated genetically, that ECTs are highly correlated genetically, and that psychometric tests and ECTs are highly correlated genetically.



**Fig. 1.** Latent multivariate genetic analysis of two psychometric tests and two elementary cognitive tests (ECTs). Additive genetic (A), common or shared environment (C) and non-shared environmental (E) contributions to the phenotypic correlation between  $g$  and ECTs are shown. Raven's Advanced Progressive Matrices (APM; [a]) is a non-verbal psychometric test of intelligence. The Leistungs-Pruf-System (LPS; [b]) is a widely used psychometric test of intelligence based on Thurstone's primary mental abilities [c]. A shortened version of the LPS consisting of seven tests was administered [d]. The model used these two measures as an index of a general latent factor ( $g$ ) which is free of measurement error. Similarly, a general ECT latent factor (ECT) was indexed by two tasks, Sternberg's memory scanning (SMS) and Posner's letter matching (PLM).

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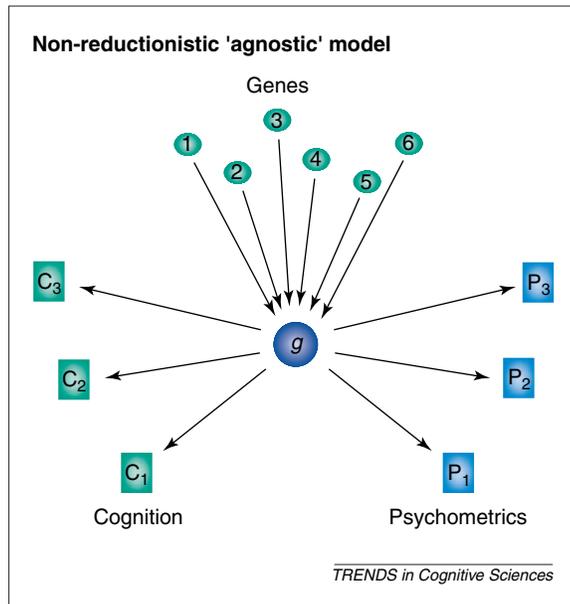
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No genetic studies of working memory have been reported. One multivariate genetic analysis refers to working memory but in fact assesses several timed tasks of motor performance [45]. Nonetheless, if working memory is perfectly correlated with  $g$ , it must be heritable to the same extent as  $g$  and its genetic correlation with  $g$  must be 1.0. Multivariate genetic research on the components of working memory (articulatory loop, phonological store and central executive) [46]

and their relationship to psychometric  $g$  is needed.

### Cognitive and psychometric correlates of genetic $g$

Multivariate genetic research on the cognitive correlates of psychometric  $g$  has identified several heritable elementary cognitive processes that are genetically correlated with psychometric  $g$ . Multivariate genetic analysis showing substantial genetic correlations among these cognitive



**Fig. 3.** A non-reductionistic 'agnostic' model of the relationship between genes, cognition and psychometric tests. Genes broadly determine psychometric  $g$ , which in turn determines cognition and the results of psychometric tests.

processes supports Model 3 over Model 2, suggesting that genetic  $g$  emerges from tests of elementary cognitive processes as well as from psychometric tests. Although much more multivariate genetic research is needed, we conclude that genetic  $g$  is in the mind as assessed by indices of elementary cognitive processes, not just in complex psychometric tests.  $g$  might also exist in the brain in the sense that  $g$ -related physiological and physical processes might overlap genetically. It may be that  $g$  goes all the way upstream to physiological measures such as synaptic plasticity

to physical measures such as qualities and quantities of neurons and even to gene expression. For example, a recent twin study suggests that gray matter density in several brain regions is highly heritable, substantially intercorrelated across brain regions, and moderately correlated with  $g$  [47].

An even more radical interpretation questions the reductionistic assumption of all the models in Fig. 1. The *TICS* review concluded that there is disillusionment with the reductionistic premise of research on the cognitive correlates of psychometric  $g$ : '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' [3]. We suggest that the 'other variable' is genetic  $g$ . That is, the same genetic  $g$  might be assessed not only by psychometric tests but also by reaction time, inspection time, and psychophysiological measures. Some of these measures, especially psychophysiological measures, are less good indices of genetic  $g$  and thus correlate less highly phenotypically and genetically with other indices of  $g$  (see Fig. 3). Although the reductionistic model of brain  $\rightarrow$  cognition  $\rightarrow$  behavior is deeply embedded in our thinking, an agnostic model in which brain, cognitive and psychometric measures are considered merely as correlates rather than causes of genetic  $g$  is all that is warranted from the data so far. Moreover, we hope that the non-reductionistic 'agnostic' model will help to keep our conceptual options open by considering possible top-down models rather than being automatically seduced by bottom-up reductionistic models.

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# The topography of high-order human object areas

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**Cortical topography is one of the most fundamental organizing principles of cortical areas. One such topography – eccentricity mapping – is present even in high-order, ventral stream visual areas. Within these areas, different object categories have specific eccentricity biases. In particular, faces, letters and words appear to be associated with central visual-field bias, whereas buildings are associated with a peripheral one. We propose that resolution needs are an important factor in organizing object representations: objects whose recognition depends on analysis of fine detail will be associated with central-biased representations, whereas objects whose recognition entails large-scale integration will be more peripherally biased.**

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Topographic mapping is a ubiquitous property of sensory and motor cortex: there is an orderly and gradual change in some functional property of cortical neurons laid along the cortical surface. However, the topographical map is never a simple copy of the sensory surface, rather it undergoes complex and precise transformations, along well-defined organizing principles. This striking phenomenon has prompted the suggestion that topographic transformations might serve to facilitate specific cortical computations (e.g. [1,2]).

This conjecture is compatible with a more general perspective in which cortical neuroanatomy and mapping principles are treated as computational devices [3].

From this perspective, the interest in visual cortex topography stems not only from its use as a principle that defines the layout of many visual areas, but rather in providing important insights concerning the actual computations and optimizations performed by cortical networks. In this sense, the information about 'where' things occur in the cortex is highly relevant to knowing 'how' cortical computations are performed.

## The basics of visual cortical topography

The topographic transformation from the retina to the cortical surface has been amply documented both in non-human primates (e.g. [4]) and, more recently, in human early visual areas, using functional magnetic resonance imaging (fMRI) [5–7]. A consistent finding in all these studies is that the mapping principle involves a topographic