

IQ heritability estimation: Analyzing genetically-informative data with structural equation models

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When analyzing genetic data, Structural Equations Modeling (SEM) provides a straightforward methodology to decompose phenotypic variance using a model-based approach. Furthermore, several models can be easily implemented, tested, and compared using SEM, allowing the researcher to obtain valuable information about the sources of variability. This methodology is briefly described and applied to re-analyze a Spanish set of IQ data using the biometric ACE model. In summary, we report heritability estimates that are consistent with those of previous studies and support substantial genetic contribution to phenotypic IQ; around 40% of the variance can be attributable to it. With regard to the environmental contribution, shared environment accounts for 50% of the variance, and non-shared environment accounts for the remaining 10%. These results are discussed in the text.

Estimación de la heredabilidad del CI: analizando datos genéticamente informativos con Modelos de Ecuaciones Estructurales. Cuando se analizan datos genéticos, los Modelos de Ecuaciones Estructurales (SEM) proporcionan una metodología sencilla y directa para descomponer la varianza fenotípica utilizando una aproximación basada en diferentes modelos. Además, se pueden implementar, probar y comparar diversos modelos fácilmente utilizando SEM, permitiendo al investigador obtener información muy valiosa acerca de las fuentes de variabilidad. En este trabajo, se describe brevemente esta metodología y se reanalizan unos datos de CI españoles utilizando el modelo biométrico ACE. En resumen, aportamos estimaciones de la heredabilidad que son consistentes con las de estudios anteriores y que dan soporte a una contribución genética sustancial al CI fenotípico, alrededor del 40% de la varianza puede ser atribuida a la genética. Respecto a la contribución ambiental, el ambiente compartido da cuenta de un 50% de la varianza, y el ambiente no compartido explica el remanente 10%. Los resultados se discuten en el texto.

Although the old debate over nature versus nurture appears interminable (Bouchard & McGue, 2002), researchers in the field of behavioural genetics are reaching a relative consensus about the influence of nature, nurture, and especially of nature via nurture (Plomin, 1994). This consensus has been reached after a number of studies spanning more than one hundred years.

It was Sir Francis Galton who, in his book *Hereditary genius: an inquiry into its laws and consequences* (Galton, 1892), first sought to gather scientific knowledge of environmental and genetic sources of human individual differences. His basic proposition was that if a given trait is genetically determined, then the closer the degree of kinship between two relatives the higher the correlation for that given trait between them. Some years after Galton's pioneering insights, Ronald Fisher provided the first formal and general account of how the correlations between relatives could be explained on the supposition of Mendelian inheritance (Neale & Maes, in press). His main contribution was

the development of the concept of likelihood, which would become essential for understanding future developments in behavioural genetics.

More recently, in the early 1970s, David W. Fulker introduced the maximum likelihood estimation into human quantitative genetics (Jinks & Fulker, 1970). This new estimation and a method of covariance structure analysis proposed by Jöreskog (1970) led Martin and Eaves to develop specific FORTRAN software in order to implement this type of analysis in behavioural genetics research (Martin & Eaves, 1977). The widespread use of LISREL (Jöreskog & Sörborm, 1989) in the 1980s meant that this approach was readily available to applied researchers and it became a common feature of such work (Boomsma, Martin, & Neale, 1989; Fulker, 1988).

This methodology has prevailed throughout the 1990s so far. However, the extensive use of *Mx* (Neale, Boker, Xie, & Maes, 2003), an alternative software to LISREL, has simplified the specification of the behavioural genetics variance decomposition models.

As the existence of a number of twin registers and studies in both Europe and the rest of the world show, this methodology has been widely adopted (Boomsma, Busjahn, & Peltonen, 2002). The aim of these types of study is to decompose the sources of variance (see Methods) that determine the variability of a given phenotypic

trait. This approach leads to a better understanding of how these sources interact with each other to produce the observable individual differences.

Although some research has been conducted in behavioural genetics (see Bartrés-Faz et al., 2001), unfortunately in Spain there are no twin and adoption registries of public access, at least not for research purposes. Consequently, only a few studies have been conducted and published with regard to twin or adoption designs. Of these, some are related to general medicine or neurology (Narberhaus et al., 2004; Ramos-Arroyo, 1991; Rozas et al., 2000) while others explore the phenomenon of twinning (Hernández, García-Moro, Toja, Esparza, & González-José, 2004). Only the group led by Lourdes Fañanás at the University of Barcelona has addressed the research on psychiatric disorders using twins. Specifically, they have investigated the relationship between dermatoglyphic abnormalities and psychosis (Rosa, Fañanás, Bracha, Torrey, & van Os, 2000; Rosa et al., 2002; van Os, Fañanás, Cannon, Macdonald, & Murray, 1997) comparing concordant and discordant twin pairs by means of percentages and odds ratios.

To our knowledge only three articles have been published in Spain regarding the study of individual differences from this point of view (Carrillo-Gistain & Pigem, 1982; Carrillo-Gistain & Sánchez-Turet, 1990, 1992), and all of them report the results of an unpublished dissertation (Carrillo-Gistain, 1987). The authors analysed their results in a narrow sense, that is, they reported series of differential correlations between monozygotic (MZ) and dizygotic (DZ) twins in different environmental conditions, along with some contingency tables, in order to show a possible interaction between genetic pool and environment in the development of intelligence. However, they did not report any heritability estimate, nor apply Falconer's formula (Falconer, 1960), which is the simplest, albeit an inaccurate, way of calculating it.

With regard to the heritability of intelligence, several studies are available elsewhere (for a review, see Bouchard and McGue, 2002). Some studies have focused on the heritability of this phenomenon during adulthood (Bouchard & McGue, 1981, 2002; Plomin & Spinath, 2004; Posthuma, de-Geus, & Boomsma, 2002), concluding that genetic factors account for a substantial variance of IQ (around 60%). However, in childhood and adolescence these results are somewhat lower. Hence, some studies have investigated the reasons for these discrepancies, and it seems that heritability of IQ changes during the lifespan, increasing over time (Palfrey-Davis, Arden, & Plomin, submitted for publication; Plomin & Spinath, 2004; Spinath, Ronald, Harlaar, Price, & Plomin, 2003).

In order to obtain the maximum amount of information from Carrillo-Gistain's data (1987) we re-analysed it using structural equation modelling (SEM) in order to illustrate how SEM can be used to analyse genetic data. We focus primarily on the steps to follow in analysing such data and, secondly, aim to check whether the obtained results are similar to previous literature or not.

Methods

Participants

The sample comprised 140 subjects, distributed into two groups: one group of 39 MZ twin pairs and another of 31 DZ twin pairs. The sample was recruited in Lleida and its province, as well as in the cities of Igualada and Ciudad Real (Spain). Thirty-three

pairs of twins came from a rural environment (47%) and 37 from an urban environment (53%). With regard to socio-economic status (SES), 15 pairs (22%) had a high SES, 43 pairs (61%) a medium SES and 12 pairs (17%) a low SES. The original source (Carrillo-Gistain & Sanchez-Turet, 1990) provides further details about the sample.

Procedure

The original authors applied Wechsler scales to the participants in accordance with their age. Thus, the WPPSI (Wechsler, 1986), WISC (Wechsler, 1988) and WAIS (Wechsler, 1976) were applied to obtain the IQ score.

Analyses

A general view of model fitting can be found in various sources (Plomin, DeFries, Craig, & McGuffin, 2002), as can a detailed description of model fitting in behavioural genetics (Boomsma, Martin, & Neale, 1989; Neale & Maes, in press).

It will be useful to begin with some remarks regarding the terminology used. Heritability (see equation 1), which is also known as h^2 , is a descriptive statistic that can be defined as the proportion of phenotypic variation of a population that is due to genetic variance. This genetic variance can be decomposed into additive genetic variance (A), in which genotypic values provoke linear effects on the phenotype, and non-additive genetic variance (D), which represents genetic influences as dominance (interactions between alleles at the same locus) or interactions between different loci, that is, epistasis,

$$h^2 = \sigma_A + \sigma_D \tag{1}$$

On the other hand, environmental differences between individuals can also lead to phenotypical differences between them. Indeed, behavioural genetics often offers researchers the best evidence for the relevance of environmental influences on development. As in the case of genetic variance, environmental variance can be separated into two types (see equation 2): common environmental (or shared environment) variance (C), which contributes to the phenotypical resemblance of family members, and specific environmental (or non-shared environment) variance (E), which is defined as all environmental influences that make a certain individual within a family different from the rest. It also includes the measurement error.

$$\sigma_{ENV} = \sigma_C + \sigma_E \tag{2}$$

In general, phenotypic variance (IQ score in our case) can be decomposed in terms of heritability, environment and interaction between the two terms, as is shown in equation 3.

$$\sigma_P = (\sigma_A + \sigma_D) + (\sigma_C + \sigma_E) + ((\sigma_A + \sigma_D) \times (\sigma_C + \sigma_E)) \tag{3}$$

However, in the approach adopted here, which is also known as the biometric model (Neale & Maes, in press), we assume that all genetic variance is additive and that there is no interaction between genetic and environmental effects. Therefore, in our model there are three unknown variables: A, C and E. Thus, the model is known as the ACE model, as described in equation 4.

$$\sigma_P = \sigma_A + \sigma_C + \sigma_E \quad (4)$$

In addition, in order to test the importance of genetic and environmental components, three alternative models (AE, CE, and E) are also tested. The equations for these models can be easily derived from equation 4.

To test the models described above, and shown in Figure 1, we used the Reticular Action Metamodel (RAM) approach to structural equation modelling. These rules were developed by John J. McArdle between 1978 and 1981 (see, for instance, McArdle, 1979, 2005). This approach links the traditional graphic representation of path analysis to a matrix model of covariance structures using a series of rules that provide the means to translate the graphic depiction into matrices.

In general, RAM rules convert the graphic relationships between the p observed and q latent variables and allow the development of three matrices: S , A and F . The relations between variables are contained into matrices S and A .

Matrix S has dimensions $t \times t$, its elements represent all the symmetric paths (i.e. double headed arrows in figure 1) included in the model, where $t = p+q$ is the total number of variables involved in the analysis. S contains the symmetric paths (i.e., double headed arrows) represented in the graphic model, thus containing variances and covariances.

Matrix A of dimensions $t \times t$ is also defined to represent directed or asymmetric relationships (i.e., single headed arrows in Figure 1) between variables. Elements of matrix A model the causal relationships between variables driven by theory or working hypotheses. Consequently, the elements of S and A are structural parameters of the covariance structure model, and their elements can be fixed, freed or constrained as convenient to constructing the model.

Finally, F is a fixed matrix of dimensions $p \times t$. Elements of F are zeros and ones designed to extract the observed variables out of the full set of manifest and observed variables. That is, to filter the observed variables in the model and isolate them. The purpose

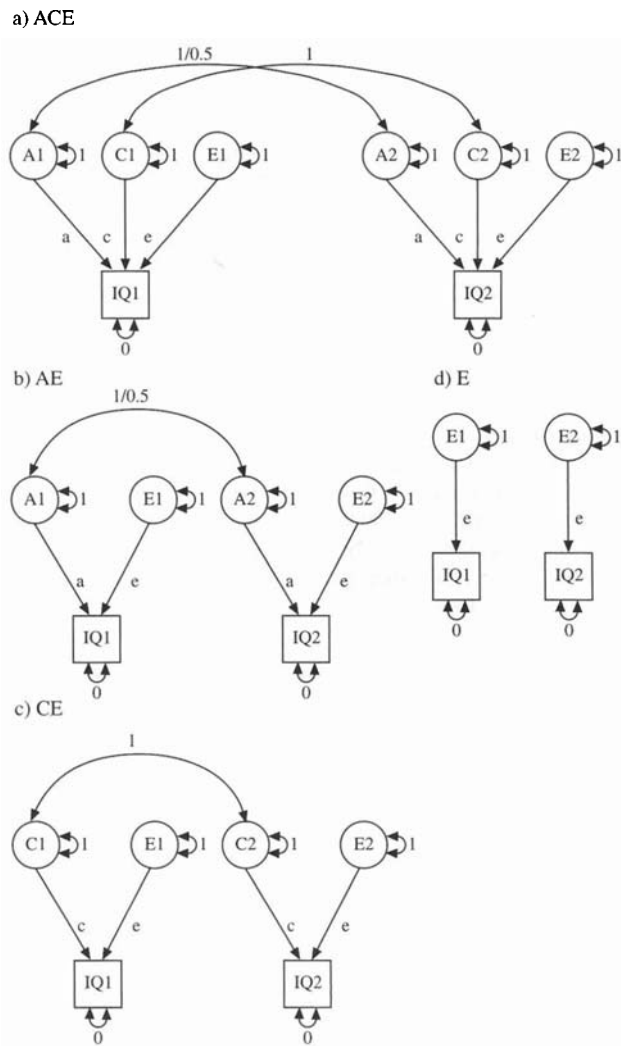


Figure 1. Diagram representing the ACE, AE, CE and E models. Note: Latent variables (circles) and observed values (squares) are duplicated to show both members of a twin pair. A= Additive genetic variance; C= shared environment variance; E= non-shared environment variance; IQ= phenotypic IQ of each twin. Double headed arrows represent correlations between latent factors or variance. Single headed arrows represent factor loadings. Lowercase letters represent the parameter to estimate

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of this matrix is simply to implement into the model the difference between indicators (observed variables) and factors (latent variables).

Once we have defined these matrices we can proceed to calculate the expected covariance matrix between observed variables as:

$$\Sigma_{pp} = F(I-A)^{-1}S(I-A)^{-1}F' \tag{5}$$

where **I** represents a $t \times t$ identity matrix.

In our particular case, the observed variables are the IQ scores for each twin in a twin pair. The latent variables are the A, C and E factors. Thus, we have $p=2$ and $q=6$. Symmetric relationships are contained in matrix **S**, whereas causal relationships are contained into matrix **A**. The **S** matrix is 8×8 , representing all symmetric paths (variances and covariances) in the model. **A** is also 8×8 and contains only causal paths. Finally, **F** is a 2×8 design matrix.

Let's see with more detail the matrices that represent the complete ACE model. Matrix **A** is as follows:

$$A = \begin{pmatrix} 0 & 0 & a & c & e & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & a & c & e \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{6}$$

The matrix **S** differs depending on which twin group is being analysed. The element (6, 3) of **S** represents the genetic correlation between two relatives. Thus, in the MZ group the correlation has a value of 1, because MZ twins share the 100% of their genes. In contrast, in the DZ group this element is 0.5, because DZ twins share half of their genetic pool.

$$S = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 1 \text{ or } 0.5 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} \tag{7}$$

Finally, the configuration of the **F** matrix is the following:

$$F = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{8}$$

In order to fit the model (i.e. to compare the expected covariance matrix with the observed covariance matrix); we used the MPLUS software (Muthén & Muthén, 1998). Additionally, for the sake of exemplifying purposes, we also analysed it with *Mx* software (Neale, Boker, Xie, & Maes, 2003), which is specifically

intended for the analysis of genetic data (input syntaxes are available upon request).

The models were estimated via maximum likelihood (Ximénez & García, 2005), using sample covariances as input. As the different models are not nested, the selection of the best fitting model should be made using the Akaike's Information Criterion (AIC: Akaike, 1987). However, we will use the Bayesian Information Criterion (BIC: Schwarz, 1978) as it is more robust to distributional misspecification under certain conditions and outperforms AIC in large samples and complex models (Markon & Krueger, 2004).

Results

Table 1 shows the intraclass correlations between the members of each pair of twins and means and standard deviations for IQ scores, for both monozygotic and dizygotic twins. We used these statistics as sample statistics to perform the analysis.

For the IQ score, four models were fitted to the data. As Table 2 shows, the model that allowed for additive genetic (A), common environmental (C) and specific environmental (E) parameters provided the best fit to the data. The model that allowed only for specific environmental (E) parameters provided the worst fit to the data. Table 2 provides the complete results of the model fitting.

Table 3 indicates the proportion of variance due to each latent factor and the parameter estimates. Additive genetic variance accounts for a significant additive genetic influence (40%); however, common or shared environment influences account for the largest portion of variance (50%). In addition, specific or non-shared environment influences account for the residual 10%.

Table 1
Intraclass correlations, means and standard deviations between MZ and DZ twins for the IQ score

	MZ ^a		DZ ^b	
	Twin 1	Twin 2	Twin 1	Twin 2
ICC	1	1	1	1
	.89**		.68**	
Mean	102.08	102.03	105.48	107.58
S.D.	15.74	15.02	14.58	14.73

^aN= 39
^bN= 31
Notes: MZ= Monozygotic twins; DZ= Dizygotic twins; **p<0.01

Table 2
Results for the fitting different models including A, C or E

Model	χ^2	df	p-value	BIC	RMSEA	90% CI of RMSEA
1. ACE _a	0.56	3	0.91	1081.78	0	0.000 - 0.116
2. AE	5.09	4	0.28	1082.06	0.09	0.000 - 0.283
3. CE	10.08	4	0.03	1087.06	0.21	0.043 - 0.373
4. E	84.23	5	0	1156.95	0.67	0.551 - 0.803

^a Best fitting model. Notes: A= Additive genetic variance; C= Common environmental variance; E= Specific environmental variance; df= Degrees of Freedom; BIC= Bayesian Information Criteria; RMSEA= Root Mean Square Error of Approximation

	Parameter	S.E.	% of variance
A	9.31	1.89	40
C	10.46	2.14	50
E	4.83	0.54	10

Notes: A= Additive genetic variance path; C= Common environment variance path; E= Specific environment variance path; S.E.= Standard error of the parameter estimate

Discussion

The aim of this article was to illustrate the use of structural equation modelling in the behavioural genetics field. We have exemplified, by means of a simple approach (the RAM one) and two different software programs, how this can be done in an easy, accurate and straightforward way.

The main strength of this approach, as shown in the paper, is that when changing the model and comparing different models it is easy to check which one fits best. This can be done in MPLUS simply by removing a couple of code lines and in *Mx* by specifying alternatively the matrices. This allows fast and simple comparisons to be made between models.

Additionally, we were interested in obtaining the heritability estimates for the IQ score and check whether they were consistent with previous literature. According to Bouchard and McGue (2002), broad heritability of IQ is about 50% and our results are not far from this result. In a classic review by Bouchard and McGue (1981), they report the median IQ correlation for 4,672 pairs of identical (MZ) twins and for 5,546 pairs of fraternal (DZ) twins: 0.86 and 0.60, respectively. The correlations obtained in the present analysis are quite close to those reported by Bouchard and McGue.

As pointed out in the introduction, heritability is the proportion of total variance explained by additive genetic variance. Thus, in our sample additive genetic variance explains 40% of the variance. This means that a large proportion of IQ variance is explained by genes. In this regard, the hunting for genes for cognitive ability has just begun (Bouchard & McGue, 2002) and some candidate genes have been identified, for example, the *Plp* gene (Xq22.3) which codes for two membrane proteins important for myelination (Posthuma, de Geus, & Boomsma, 2002).

However, other authors have found broad heritabilities much higher than those presented above (Posthuma, de Geus, & Boomsma, 2002). This discrepancy can be explained in the following terms. Firstly, IQ heritability estimates are known to change during the life span (Plomin & Spinath, 2004), increasing over time. Here, we reanalysed a sample gathered under heterogeneous conditions, which thus contains a mixture of ages and urban and rural environments, etc. This age mix could explain the median IQ heritability estimates and thus, with regard to the results, we can conclude that they are consistent with previous literature.

It should also be noted that the influence of shared environment is more important than additive genetic variance: it explains half of the variance. This estimate is much higher than in other reports, especially with respect to studies of twins reared apart (Bouchard & McGue, 2002). Such a difference could be explained as follows:

in our sample, being reared together boosted the resemblance between both members of each twin pair. In addition, it is widely known that the different estimates of heritability and shared and non-shared environment estimates change across ages (Bouchard & McGue, 2002). Frank Spinath and co-workers (Spinath, Ronald, Harlaar, Price, & Plomin, 2003) investigated the genetic and environmental influences on *g* in early childhood and found a large proportion of variance explained by shared and non-shared environment influences, rather than genetic effects. This work is being continued at the Institute of Psychiatry in London by the team led by Robert Plomin, who are studying children aged 7, 9 and 10 years (Palfrey-Davis, Arden, & Plomin, submitted for publication); the results are quite consistent with previous literature, so heritability and non-shared environment estimates tend to increase over time while shared environment estimates tend to decrease. As our sample contains individuals from all age ranges, we consider this might be confounding the parameter estimates.

Although there is a large number of caveats in this study, the design is useful for the purposes of exemplification. Firstly, it is well known that structural equation modelling needs large samples in order to obtain powerful enough results when working with human beings, although not when working with inbred strains (Preacher & MacCallum, 2002). Secondly, the models can include covariates, such as sex, age, cultural transmission, different degrees of correlated environment, and so on. Despite the advantages, the inclusion of these variables in the model makes their implementation complicated. However, the building of complex models goes beyond the aim of the present paper and some examples can be found elsewhere (Neale & Maes, in press).

Thirdly, it can be argued that our sample is affected by a restriction of the range of SES variables (see Stoolmiller, 1999), and range restriction is known to attenuate the correlation between two variables. Given that we used correlations as input statistics, they could be affected by these restrictions. However, as Stoolmiller points out (p. 402) «the range restriction is an inherent part of the adoption design but need not be so for twin designs», and here we are dealing with a classical twin design.

Fourthly, and as discussed above, we are unable to disaggregate the effects of age and rearing without losing statistical power. Our sample contains several ages and at every age the proportion variance explained by shared environment and non-shared environment changes, increasing or decreasing over time.

Fifthly, it can be argued that the use of three different tests may confound the results. However, the APA 1996 Intelligence Task Force Report (Neisser et al., 1996) recognizes that all IQ scores, measured by means of any intelligence test (that is well built, valid, reliable, etc.), are equivalent.

The model we have presented herein (Martin & Eaves, 1977) is merely an example that has been extensively used for almost thirty years. It is not necessarily the correct one, although the evidence suggests that we cannot reject it.

In sum, this methodology is without doubt a powerful tool for detecting genetic, environmental or interaction sources of variation for a large set of individual differences (Bouchard & McGue, 2002). It should also be noted that it is not restricted to the models presented here, but can be used to compare alternative or competing models. This instrument is therefore available to investigators to develop new theories and must not be restricted to existing models.

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