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STRUCTURAL EQUATION MODELS IN HUMAN BEHAVIOR GENETICS

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Introduction

That IQ is a highly heritable trait has been widely reported. Rather less well-known are such recent reports in major scientific journals that the heritability of controllable life events is 53% among women and 14% among men (Saudino et al., 1997), while the heritabilities of inhibition of aggression, openness to experience, and right-wing authoritarianism are respectively 12%, 40%, and 50% (Pedersen et al., 1989; Bergeman et al., 1993; McCourt et al., 1999). It seems that milk and soda intake are in part heritable, but not the intake of fruit juice or diet soda (de Castro, 1993).

These numbers are parameter estimates obtained in structural modeling of measures taken on pairs of siblings -- prototypically identical (monozygotic) and fraternal (dizygotic) twins, reared together and reared apart. The models are of the random effects type, in which variances and covariances of an observed trait -- a phenotype -- are specified in terms of latent factors -- genetic and environmental -- whose prespecified cross-twin correlations differ by zygosity and rearing status. Estimation is by maximum likelihood, with chi-square testing, and confidence intervals (occasionally based on empirical likelihood). Heritability, the key parameter of interest, is the proportion of phenotypic variance that is attributable to genetic variance.

For these studies, various issues arise. Those to be touched on here include: identification by theoretical restrictions, nonnegativity constraints, pretest estimation, conditioning of the design matrix,

multivariate analyses, and the objectives of structural modeling.

For present purposes, I focus on the SATSA project -- the Swedish Adoption/Twin Study of Aging which, beginning about 1984, assembled a sample of adult twin pairs -- approximately 200 MZT (identical twins reared together), 200 DZT (fraternal twins rered together), 100 MZA (fraternal twins reared apart), and 150 DZA (fraternal twins reared apart). Only same-sex pairs are included. The subjects have been assessed in person and via mail questionnaires on repeated occasions, with varying sample sizes, on a wide range of traits, some cognitive, others concerning personality, temperament, and recollections of childhood upbringing. Serious concerns about the representativeness of the samples and of the reliability and validity of the measures are expressed in Goldberger & Kamin (1998) and Kamin & Goldberger (2001). Here those concerns are suppressed in order to focus on the modeling.

Primary Model

The specification of the primary SATSA model is captured as follows. Consider a typical individual, whose phenotype (observable trait value) Y is determined as

(1) $$Y = \alpha_1 G + \alpha_2 D + \alpha_3 S + \alpha_0 U.$$

Here G is the additive genetic factor, D is the nonadditive (dominance) genetic factor, S is the shared environment factor, and U is the nonshared environment factor. (The distinction between the two genetic factors will be clarified later). Assume the factors are uncorrelated, and standardize all variables to have zero means and unit variances, so the phenotypic variance is

(2) $V(Y) = \alpha_1^2 + \alpha_2^2 + \alpha_3^2 + \alpha_0^2 = 1$.

The individual is paired with his/her sibling, whose phenotype is determined as

(3) $Y' = \alpha_1 G' + \alpha_2 D' + \alpha_3 S' + \alpha_0 U'$.

Across the sibling pair, all factor covariances are assumed to be zero except perhaps for those that link the sibs' additive genetic, nonadditive genetic, and shared environment factors. So the phenotypic covariance for sibs is

(4) $C(Y,Y') = C(G,G')\ \alpha_1^2 + C(D,D')\ \alpha_2^2 + C(S,S')\ \alpha_3^2$.

Referring to identical and fraternal twins (MZs and DZs), reared together and apart (T and A), those factor covariances are assumed to be

$C(G,G') = 1$ for MZs, $= 1/2$ for DZs

(5) $C(D,D') = 1$ for MZs, $= 1/4$ for DZs

$C(S,S') = 1$ for MZTs and DZTs, $= 0$ for MZAs and DZAs

$C(U,U') = 0$,

see Figure 1. With all variables standardized, covariances are also correlations, and will be labelled as such.

It is convenient to set out this display of variables:

(6)

		x_1	x_2	x_3
1.	MZT	1	1	1
2.	DZT	0.5	0.25	1
3.	MZA	1	1	0
4.	DZA	0.5	0.25	0

Here x_1, x_2, x_3 refer to the additive genetic, nonadditive genetic, and shared environment factors. The values assigned to those variables correspond to the correlations assumed above: The additive genetic factor

correlates 1 for MZs and 0.5 for DZs, the nonadditive genetic factor correlates 1 for MZs and 0.25 for DZs, the shared environment factor correlates 1 for twins reared together and 0 for those reared apart, and the nonshared environment factor correlates not at all.

So, the primary SATSA model has this linear specification for the population phenotypic correlations:

(7) $$\rho = X\,\beta$$

where $\rho = (\rho_1\ \rho_2\ \rho_3\ \rho_4)'$, $X = (x_1\ x_2\ x_3)$, $\beta = (\beta_1\ \beta_2\ \beta_3)'$, the βs being the squares of the corresponding αs -- that is the components of phenotypic variance. More explicitly,

(8) $$\begin{bmatrix} \rho_1 \\ \rho_2 \\ \rho_3 \\ \rho_4 \end{bmatrix} = \begin{bmatrix} 1 & 1 & 1 \\ 0.5 & 0.25 & 1 \\ 1 & 1 & 0 \\ 0.5 & 0.25 & 0 \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \\ \beta_3 \end{bmatrix}$$

In this model, the sum $\beta_1 + \beta_2$ gives heritability. The nonshared environment component of variance follows as $\alpha_0^2 = \beta_0 = 1 - (\beta_1 + \beta_2 + \beta_3)$.

With 4 moments expressed in terms of 3 parameters, there is 1 equality restriction, namely

$$\rho_1 - \rho_3 = \rho_2 - \rho_4,$$

which says that the difference between MZ and DZ correlations is the same whether the twins are reared together or apart. Also with all 4 βs assumed nonnegative, there is an inequality restriction

$$\rho_3/2 \geq \rho_4 \geq \rho_3/4,$$

the DZA correlation should lie between one-fourth and one-half of the MZA correlation. To the extent that β_3 is small, the same might hold also for the DZT & MZT correlations.

Given random samples from each of the 4 groups, one might take

observed phenotypic correlations $r = (r_1\ r_2\ r_3\ r_4)'$, interpret them as estimates of the population correlations, and estimate $\beta = (\beta_1, \beta_2, \beta_3)'$ by running the LS linear regression of the 4 x 1 vector r on the 4 x 3 matrix X, thus minimizing

$$\sum_{i=1}^{4} (r_i - \rho_i)^2.$$

A more appropriate procedure, feasible WLS, would take account of the fact that the variance of a sample correlation coefficient depends on the population correlation coefficient and the sample size, and thus choose values for the β-estimates to minimize

$$\sum_{i=1}^{4} w_i (r_i - \rho_i)^2,$$

where $w_i = (1 - r_i^2)^2/n_i$, with n_i being the number of observations in the i-th twin group.

Most convenient is to work with Fisher's z-transforms of correlations coefficients,

$$z = (1/2)\ \log[(1 + r)/(1 - r)],$$

$$\zeta = (1/2)\ \log[(1 + \rho)/(1 - \rho)],$$

relying on the fact that z is approximately $N(\zeta, 1/n)$. This method, which I'll label ZLS, chooses values for the β-estimates to minimize

$$\sum_{i=1}^{4} n_i (z_i - \zeta_i)^2,$$

which is a simple, albeit nonlinear, calculation. With 4 observations and 3 parameters, the minimized criterion provides an asymptotic $\chi^2(1)$ statistic of model fit, which tests the equality restriction $\rho_1 - \rho_3 = \rho_2 - \rho_4$.

I have oversimplified the procedure of the SATSA group in several respects. They do not standardize the observed variables, but rather work with variances and covariances, taking β_0 as a free parameter. (In that case, the parameter estimates are rescaled ex post to report the

proportional allocation of variance). They do not use ZLS but rather (Gaussian) ML . Often, they take as data 8 phenotypic variances -- the between-family and within-family components for each of the 4 twin groups. This gives 3 additional degrees of freedom for model fit, which are essentially allocated to the hypothesis that the four phenotypic variances are the same, an interesting hypothesis, but one that has little to do with the behavior-genetic theory. Sometimes they work with 12 observed phenotypic variances and covariances -- for each twin group a variance for twin A, a variance for twin B, and the covariance. This gives 4 additional degrees of freedom for model fit, which are effectively allocated to equating the phenotypic variances for twins A and B in each twin group. That labelling of the twins was arbitrary, so those 4 additional degrees of freedom are in effect testing whether their own random assignment of the labels was in fact random, a hardly interesting hypothesis and one that has nothing to do with the behavior-genetic theory. In economics, Ashenfelter & Krueger (1994), working with twins but not with behavior genetics, also act as if an arbitrary labelling of Twin A and Twin B were meaningful.

Typically, the observed traits have been residualized on age and gender before the modelling exercise begins. But occasionally age is introduced into the model itself as a covariate. This adds 2 parameters (the population age variance and the trait-on-age slope), and adds 12 observed moments -- the covariance of twin A's trait with age, the covariance of twin B's trait with age, and the variance of age -- for each of the 4 twin groups. (Not only is the A:B labelling arbitrary, but the twins have the same age). So Pedersen et al. (1992) could report a total of 18 degrees of freedom for model fit, while the core model, in correlational

terms, had only 1. Perhaps an analogous situation -- success in fitting features of the data that have no particular relevance to the core theory -- occurs on occasion in economics?

Genetical Theory

At this point, it should be apparent that the molecular content of this line of research is, to put it mildly, minimal. It might be said that all the genetical theory exploited is comprised of the integers 0, 1, 2, 4. The genetic content of the model, after all, consists of the ratios 1/2 and 1/4 for DZ twins relative to MZ twins. The distinction between the two genetic factors arises from the distinction between conditional expectation functions and best linear predictors. Consider a gene with two alleles, m and M. At this locus, an individual may be mm, mM/Mm, or MM. Score these as Z = 0, 1, 2, and consider the distribution of phenotypes for persons of each score. (See Figure 0). If $E(Y|Z)$ is linear -- the expected trait for heterozygotes (Z = 1) is halfway between those for homozygotes (Z = 0 and Z = 2) -- then only the additive genetic factor is present. If $E(Y|Z)$ is nonlinear -- for example if the expected trait for Z = 2 is the same as for Z = 0 -- then a nonadditive genetic factor is present. In that case, the $BLP(Y|Z)$ reflects the additive factor, and the deviations $E(Y|Z) - BLP(Y|Z)$ reflect the nonadditive factor. Only the additive factor contributes to similarity between parent and child, while both factors contribute to similarity between siblings. The Appendix sketches why DZs correlate 1/2 and 1/4 on the two genetic factors. Remarkably, the argument for a single locus extends directly to multiple loci.

Observe how many possibilities have been ruled out a priori in the

primary SATSA model to obtain identification. Covariance between an individual's genotype and environment is ruled out, which runs counter to conventional wisdom. There is no allowance for the possibility that the separated twins were placed into similar environments. The specified correlations 1/2 and 1/4 for C(G,G') are valid under random mating; they will be different if there is assortative mating for the trait. Most critically, there is no allowance for MZTs to have more similar environments than DZTs, that is no allowance for C(S,S') to differ by zygosity. This neglect is crucial, as can be seen by imagining that C(S,S') = 0.5 for DZTs, in which case the shared environment factor would be partly tracking the additive genetic factor. SATSA attributes all excess phenotypic similarity of MZs over DZs to their excessive genetic similarity.

It is quite ironic that the assumptions of the SATSA model are primarily about social behavior rather than about biological processes. From a skeptical point of view, the nonadditive genetic factor -- a statistical construct -- is simply a device that gives more flexibility to the genetic side of the story, producing a combined genetic factor that can correlate somewhere between 0.50 and 0.25 for DZs as compared with MZs.

Secondary Model

Occasionally, the SATSA group adopts an alternative model, one which drops the nonadditive genetic factor, and introduces a "selective placement" or "correlated environment" factor that correlates perfectly across twins of all types. In terms of the tabular display above, replace x_2 with a new variable x_4 whose value is 1 for all 4 twin groups, and

correspondingly replace β_2 with β_4. In this secondary model

$$(9) \qquad \begin{bmatrix} \rho_1 \\ \rho_2 \\ \rho_3 \\ \rho_4 \end{bmatrix} = \begin{bmatrix} 1 & 1 & 1 \\ 0.5 & 1 & 1 \\ 1 & 1 & 0 \\ 0.5 & 1 & 0 \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_4 \\ \beta_3 \end{bmatrix}$$

Here allowance is made for environmental similarity to differ between reared-together and reared-apart twins, thus meeting the objection that the separated twins may not have been fully separated. (This secondary model is formally the same as one in which the variable $x_4^* = 1$ for twins reared apart, 0 for twins reared together, replaces x_4, and $\beta_4^* = \beta_4 + \beta_3$ replaces β_4).

As the SATSA group points out, it is not possible to allow for both nonadditive genetic variance and distinct environmental similarities by rearing type because of the perfect collinearity that would result: $x_4 = 3\ x_1 - 2\ x_2$. (Of course, the requirement that β_3 and β_4^* be nonnegative is not the same as a requirement that β_3 and β_4 be nonnegative).

Evidently, the design matrix in (9) spans the same space as that in (8), so the secondary model again implies the single equality constraint,

$$\rho_1 - \rho_3 = \rho_2 - \rho_4 .$$

However, with all its βs assumed nonnegative, the relevant inequality is now

$$(10) \qquad \rho_3/2 \leq \rho_4 \leq \rho_3 ,$$

the DZA correlation should lie between 1/2 and 1 times the MZA correlation. So this model attracts SATSA when observed DZ correlations run high relative to the MZ correlations. Or they may choose retroactively after observing that one of the formulations would have some more negative

parameter estimates.

We might describe SATSA's approach as following a specific-to-specific strategy. Can examples of that also be found in the economic literature?

Agnostic Model

A specification that formally subsumes both the primary and secondary models is

$$(11) \qquad \begin{bmatrix} \rho_1 \\ \rho_2 \\ \rho_3 \\ \rho_4 \end{bmatrix} = \begin{bmatrix} 1 & 0 & 1 \\ 0 & 1 & 1 \\ 1 & 0 & 0 \\ 0 & 1 & 0 \end{bmatrix} \begin{bmatrix} \theta_1 \\ \theta_2 \\ \theta_3 \end{bmatrix}$$

Here $\theta_1 = \beta_1 + \beta_2$, $\theta_2 = 0.5\ \beta_1 + 0.25\ \beta_2$, $\theta_3 = \beta_3$. This design matrix spans the same space as the previous ones did, and therefore implies the same single equality constraint $\rho_1 - \rho_3 = \rho_2 - \rho_4$. But even with all θ's required to be positive, it allows the DZA correlation to be free relative to the MZA correlation. Adopting it would avoid the need to follow the model choice procedure. This reparameterization would also take away some of the pretense of profound genetic-theoretical underpinnings to the SATSA analyses.

Skeptical Model

As mentioned above, a crucial feature of the SATSA models is that they make no allowance for environmental resemblance to differ for MZTs and DZTs, as a result say of more similar treatment by parents and peers. A simple alternative, that still has only 3 parameters, would allow for an additive genetic factor, an MZT shared environment factor, and a DZT shared

environment factor. Thus

$$(12) \qquad \begin{bmatrix} \rho_1 \\ \rho_2 \\ \rho_3 \\ \rho_4 \end{bmatrix} = \begin{bmatrix} 1 & 1 & 0 \\ 0.5 & 0 & 1 \\ 1 & 0 & 0 \\ 0.5 & 0 & 0 \end{bmatrix} \begin{bmatrix} \delta_1 \\ \delta_2 \\ \delta_3 \end{bmatrix}$$

The single equality restriction is now

$$(13) \qquad \rho_4 = \rho_3/2.$$

We may suppose that all 3 δs are nonnegative, and presumably also $\delta_2 > \delta_3$ (environmental similarity greater for MZTs than DZTs). This specification, as far as I know, has not been used by behavior geneticists.

Estimation

It is rare in practice for SATSA to actually report estimates of either the full primary or secondary models set out above. Almost inevitably, one or another of the three factors will be dropped and a reduced model fitted and reported. Either one of the estimated βs was "nonsignificant" or their algorithm -- which evidently precludes negative estimates -- found the nonnegativity constraint to be binding, and so set the estimate at zero. That is, only reduced models are estimated and reported. This is the general-to-specific phase of their strategy.

In particular, throughout the SATSA publications, one rarely -- perhaps 5% of the time -- finds traits for which both additive and nonadditive genetic variance components are estimated. It is not hard to see why. While introducing separated twins formally identifies β_1, β_2, and β_3, the identification is tenuous. Treating the 4 x 3 X matrix of (8) as if it were 4 observations, the correlation (about zero) between x1 and x2 is 0.97. The high collinearity carries over to ZLS and ML estimation,

producing unreliable and negatively correlated estimates of β_1 and β_2. The implied estimate of $\beta_1 + \beta_2$ -- heritability -- might well be reliable. Pedersen, Plomin, Nesselroade, & McClearn (1992) cast their lot with the additive side; Plomin, Pedersen, Lichtenstein, & McClearn (1994), analyzing the same cognitive traits, cast their lot with the nonadditive side.

Had the Agnostic model been used instead, in almost all cases the SATSA group would have stayed with a full 3-parameter model for the correlation coefficients. The condition $\theta_1 > \theta_2 > 0$ is equivalent to $\beta_1 + \beta_2 > 0.5\ \beta_1 + 0.25\ \beta_2 > 0$, that is to $0.5\ \beta_1 + 0.75\ \beta_2 > 0$, and almost invariably, even when unconstrained estimates of one of those βs would be negative, the corresponding estimates of the θs would have been properly ordered.

One virtue of the ZLS method is that it does not constrain the parameter estimates to be nonnegative, and indeed for many of the SATSA articles, produces negative estimates where SATSA would report zeros. That permits tests of the nonnegativity constraints, which are never reported by SATSA. A curiosity of the SATSA analyses is that they almost always formulate the model in terms of path coefficients (our αs) rather than the variance components (our βs, which are squared αs). As a result, they report ML standard errors for the estimated path coefficients, which do not translate into standard errors for the parameters of primary interest, namely the proportions of variance explained. The ZLS procedure routinely produces standard errors for the estimated β's.

For a few traits selected from SATSA publications, Table 1 gives estimates of the several models.

More recently, behavior geneticists occasionally report confidence

intervals using the (profile) likelihood function. Their source article is Neale & Miller (1997), which among other things, recommends discarding any negative portion of the interval, that is, left-truncating the interval at zero.

Empirical implementation of the SATSA models is not a straightforward task, but involves a sequence of choices and stopping rules. Nothing about the path that led to their final variant is accounted for in the statistical inference they engage in. Thus the standard errors and confidence intervals they report are merely nominal, ignoring the chain of tried and discarded models that were encountered en route. The pretesting issues associated with such model selection are not raised in the behavior-genetic reports. My impression from the econometric and statistical literature is that under pretesting nominal standard errors are misleadingly low, so actual precision is overstated. Is the present context special in that there is no split between focus and secondary parameters, and the constraints are inequalities? Some Monte Carlo runs will be useful.

If the SATSA group insist on the requirement that all βs be nonnegative, it is because of their insistence on interpreting them as components of variance. But there is nothing in principle that precludes factors that contribute to dissimilarity rather than similarity of twins. Perhaps negative coefficient estimates should not serve to reject a particular variant, or perhaps the frequent occurrence of negative estimates should serve as an indication that their general behavior-genetic approach is wrong?

Multivariate Models

Having analyzed dozens of observed traits separately in the same manner, the SATSA group has moved on to multivariate analyses, in which several phenotypes are modeled jointly in terms of latent factors. So now one is concerned with accounting for observed covariance, as well as variance, of traits. For example, Lichtenstein & Pedersen (1995) analyze five observable traits jointly -- life events, loneliness, perceived support, quantity of relationships, and health -- for which they have measures on their twin pairs. (See Figure 2).

The structure here may be captured as follows (dropping the nonadditive genetic factor). For an individual,

(14) $$y = A_1\, g + A_3\, s + A_0\, u,$$

where the observed vector y is 5 x 1, and the latent factors g, s, u are 5 x 1 (with identity variance matrices), while the parameter matrices A_1, A_3, and A_0 are at most lower triangular. The individual is paired with his/her twin (identified by primes) for whom

(16) $$y' = A_1\, g' + A_3\, s' + A_0\, u'.$$

The familiar assumptions are made about cross-twin correlations among the latent factors. ML estimation of the parameter matrices then produces a decomposition of the 5 x 5 V(y) matrix into its genetic and environmental constituents. This leads Lichtenstein & Pedersen to conclude, e.g., that of the 0.17 correlation between perceived support and health among women, 0.15 is due to genetic factors, and 0.02 to nonshared environment.

Macroeconomists will recognize the Cholesky structure of the model. The ordering of the elements of y is apparently arbitrary. The behavior geneticists credit Martin & Eaves (1977) for the start of multivariate twin

modeling. But Behrman, Taubman, & Wales (1977) empirically implemented such a model, one with a natural recursive ordering: education to initial occupation to current occupation to earnings.

Objectives

This stream of research represents structural modeling in the sense that the equations represent causal links rather than (mere) empirical associations. The regressions among observable variables are derived in terms of more fundamental parameters. The parameters of interest are not those of the conditional expectation of one observed variable given others. However, the requirement that one of the structural parameters may change while others remain unchanged has not been invoked by the behavior geneticists.

It is fair to ask what the objectives of these human behavior genetics exercises are. What does one learn by learning that genetic factors account for say 50% of the variance of a certain trait? There may be a popular impression that to the extent that a trait is heritable, it is not malleable, that is not subject to change by environmental intervention. That impression is incorrect: Goldberger (1979), Maccoby (2001). Nor do the SATSA group make such a claim; they appear content to view the variance allocation as an end in itself. I recall only one article in which a policy recommendation was offered. Because perceptions of job climate have a high genetic component, Hershberger et al. (1994) recommend that firms should "place some value on selecting employees on the basis of [their] reported perceptions of climate from past jobs." The authors hasten to add that firms may already be doing just that.

Note: This paper derives from joint work with Leon J. Kamin.

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Appendix

Consider a single locus at which there are two possible alleles m and M, so at individuals are either mm, mM/Mm, or MM. Let Z = "the score" denote the number of capital Ms an individual has at that locus, so Z = 0, 1, 2. For simplicity suppose that the two alleles are equally likely, so that in equilibrium, Prob(Z=0) = 1/4, Prob(Z=1) = 1/2, Prob(Z=2) = 1/4. Assuming that all phenotypic variance is genetic, for each Z there is a phenotype Y, which we can code as

$Y_0 = -a \quad Y_1 = b \quad Y_2 = a$.

Then $E(Y) = b/2$ and $V(Y) = a^2/2 + b^2/4$. The two pieces of V(Y) are the additive and nonadditive genetic variances respectively. If b = 0, Y is linear in Z, the heterozygote's phenotype is halfway between those of the homozygotes, that is all genetic variance is additive. If a = 0, there is no linear component in Y(Z), the two homozygotes' phenotypes are the same, that is all genetic variance is nonadditive.

Denote the scores of husband, wife, and child by H, W, S respectively. It's easy to verify the tabulations of $Pr(S|H,W)$ below, and then $E(Y|H,W)$ for the two extreme cases. The final column gives the probabilities for each H,W combination assuming random mating.

		Conditional probabilities			Expected phenotypes		
H	W	S = 0	S = 1	S = 2	If b = 0	If a = 0	Pr(H,W)
0	0	1	0	0	-a	0	1/16
0	1	1/2	1/2	0	-a/2	b/2	2/16
0	2	0	1	0	0	b	1/16
1	0	1/2	1/2	0	-a/2	b/2	2/16
1	1	1/4	1/2	1/4	0	b/2	4/16
1	2	0	1/2	1/2	a/2	b/2	2/16
2	0	0	1	0	0	b	1/16
2	1	0	1/2	1/2	a/2	b/2	2/16
2	2	0	0	1	a	0	1/16

Conditional on H,W, any two (non-MZ) sibs are drawn independently, so across all families, C(Y,Y'), the covariance of their phenotypes is the same as the variance of the sibship means.

For the b = 0 case, where $E(Y) = 0$ and $V(Y) = a^2/2$, we calculate

$$V[E(Y|H,W)] = (a^2/16)\ (1 + 4/2 + 1) = a^2/4 ,$$

which is one-half of the additive variance. For the a = 0 case, where $E(Y) = b/2$ and $V(Y) = b^2/4$, we calculate

$$E[E^2(Y|H,W)] = (b^2/16)(1 + 4/2 + 1 + 1) = b^2(5/16),$$

so

$$V[E(Y|H,W)] = b^2(5/16) - (b/2)^2 = b^2/16,$$

which is one-fourth of the nonadditive variance.

The same conclusion follows when Y(Z) has both additive and nonadditive components, when allele probabilities are unequal, when there is random variation in Y for given Z, when multiple loci are introduced. See Falconer & Mackay (1996, Chapter 9). When Y(Z) is not deterministic, then one extreme case has $E(Y|Z)$ linear so $BLP(Y|Z) = E(Y|Z)$, and the other has $BLP(Y|Z)$ horizontal with $E(Y|Z)$ not constant.

Fitting SATSA Models & Variants to Cognitive Abilities

	Observed Correlations				Chi-	Primary			Secondary			Agnostic			Skeptical		
	r1	r2	r3	r4	squ	b1	b2	b3	b1	b4	b3	t1	t2	t3	d1	d2	d3
First Principal Component																	
Observed	**.80**	**.22**	**.78**	**.32**	-												
SATSA model: from their ML					1.94	0	.81	0									
: from my ZLS					1.77	0	.80	0									
Primary/Secondary/Agnostic					0.60	.31	.48	-.01	1.04	-.24	-.01	.80	.28	-.01			
Skeptical					0.56										.77	.03	-.16
Synonyms																	
Observed	**.81**	**.24**	**.58**	**.29**	-												
SATSA model: from their ML					2.99	0	.63	.14									
: from my ZLS					2.92	0	.65	.15									
Primary/Secondary/Agnostic					2.69	.14	.52	.13	.91	-.26	.13	.66	.20	.13			
Skeptical					0.00										.58	.23	-.05
Names and Faces Immediate																	
Observed	**.31**	**.24**	**.15**	**.18**	-												
SATSA model: from their ML					2.15	.32	0	0									
: from my ZLS					1.99	.29	0	0									
Primary/Secondary/Agnostic					0.19	.46	-.27	.10	.05	.14	.10	.19	.16	.10			
Skeptical					0.74										.22	.09	.13
Names and Faces Delayed																	
Observed	**.39**	**.25**	**.42**	**.15**	-												
SATSA model: from their ML					0.61	.42	0	0									
: from my ZLS					0.53	.40	0	0									
Primary/Secondary/Agnostic					0.37	.33	.04	.04	.40	-.02	.04	.38	.18	.04			
Skeptical					0.27										.39	.00	.06

Guide to Table 1.

Pedersen et al. (1992), for 14 cognitive ability traits, report correlations in Table 2, and ML parameter estimates in Table 3 (obtained from variance-covariance matrices) for their preferred reduced version of the primary model. The observed correlations given above are, along with the sample sizes, all the input data available to me.

I apply their ML estimates to get fitted correlations, and an associated chi-square statistic using the ZLS criterion. Then I re-estimate by ZLS to provide a baseline. The numbers are reassuringly close.

Then I fit the full Primary, Secondary, and Agnostic models, by ZLS. All give the same chi-square; they differ in violations of the nonnegativity constraints. Finally I fit the Skeptical model, by ZLS.

Degrees of freedom for model fit are the number of correlations (4) minus the number of parameters estimated (3 for full models, 2 or 1 for reduced versions).

Fitted correlations can be calculated from the parameter estimates.

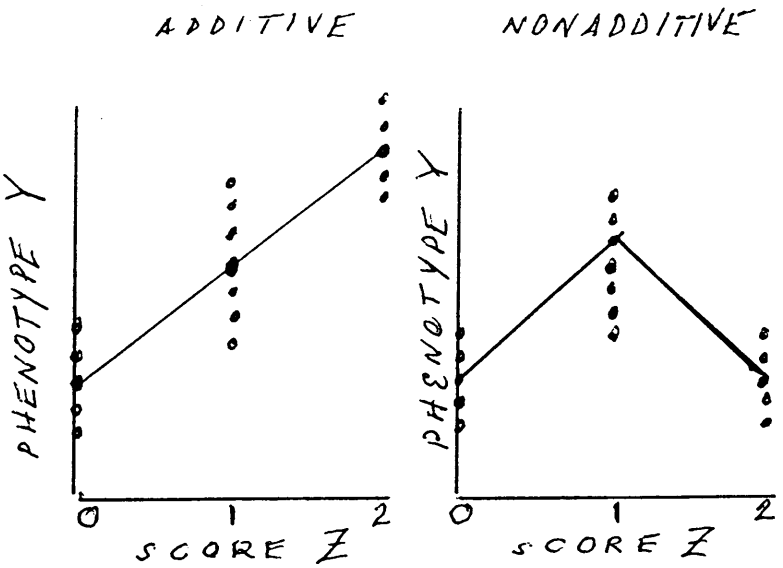
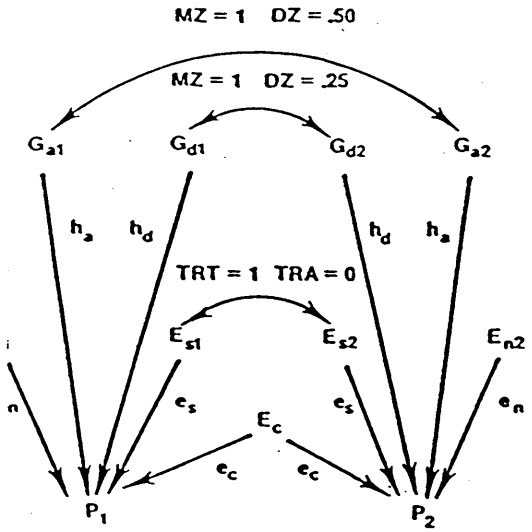


FIGURE 0.

FIGURE 1. PATH DIAGRAM DEPICTING GENETIC AND ENVIRONMENTAL RESEMBLANCE FOR TWINS



Note: G_a = additive genotypic deviation; G_d = nadditive (dominance) genotypic deviation; E_s = red rearing environmental deviation; E_c = corre-:d environmental deviation: E_n = nonshared

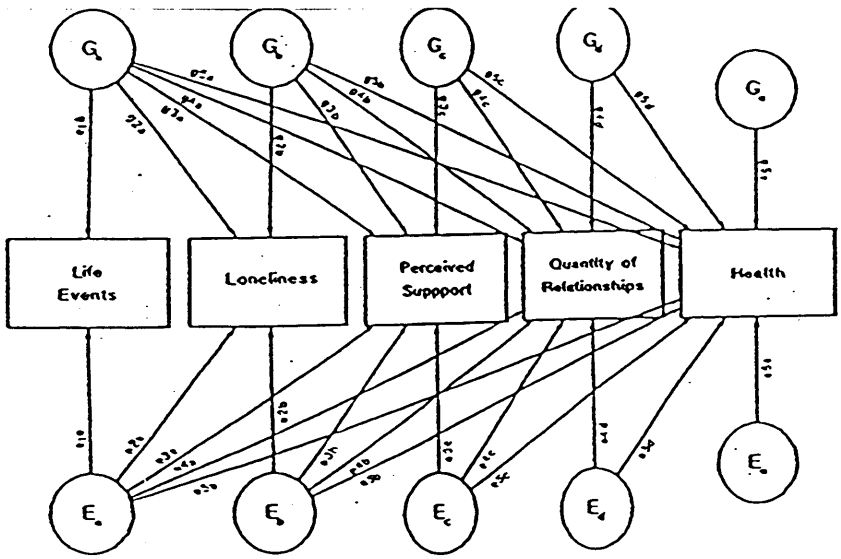


Figure 2 Cholesky path model depicting common and unique factors for genetic and environmental sources of variance and covariance for Life Events, measures of social relationships, and Health.
Note: The figure is simplified. It contains only one of the twins in the pair, and E denotes all possible environmental factors.
G=genetic factors; g=genetic loadings; E=environmental factors; e=environmental loadings.