prior distribution, players share a common selection process for choosing among multiple Bayesian Nash equilibria. The resulting structure of expectations avoids the need for the absolute-continuity assumption, albeit at the expense of losing the convergence of best-response strategies in the case of mixed equilibria.

Alternatively, the sophisticated Bayesian expectations could be viewed as having been derived by a game theorist who does not know the players' payoff functions. If the players rely on the theorist's predictions, their faith will be vindicated by the convergence of the expectations to Nash equilibria of the true game, at least in the case of myopic behavior and the theorists' use of a prior distribution satisfying the smoothness and independence conditions. Thus the sophisticated Bayesian learning model can be viewed simply as providing a class of expectation functions that can be used by myopic players in any game to learn Nash equilibrium expectations.

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Behavior, Genetic Influences on

Intermediate article

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CONTENTS

Studies of twins have established that individual differences in intelligence, personality, and psychopathology are strongly associated with genetic variations. Environmental variations are also important.

INTRODUCTION

The program of traditional (i.e. nonmolecular) behavioral genetics is to use correlations of relatives (especially twins) to make inferences about genetic and environmental contributions to behavioral variation. There are two major conclusions from such studies. First, a substantial percentage of behavioral variation seems to be due to genetic variation. Second, although environmental variation is also important, it appears that unique, idiosyncratic, experiences are more important for behavioral variation than experiences shared by twins.

Both findings represent challenges to traditional social science, the first because social science does not emphasize biological factors, and the second because the environmental factors that are the focus of social science research (parents, homes, socioeconomic status, neighborhoods, and schools) are shared by twins and are thus seen by behavioral genetics as being in a sense less potent than social science supposes. The 'in a sense' qualification is necessary since twins obviously have unique experiences with common environmental factors. A 'strict' or 'cultured' home environment may be experienced in different ways by different children (Maccoby, 2000).

TWIN STUDIES

The following text provides a moderately detailed example of a behavioral genetic analysis that supports the first and second conclusions outlined above; it exposes the weaknesses as well as the strengths of such analyses. The analysis applies to studies that include twins reared apart as well as twins reared together. Thus these studies involve twins separated early in life by adoption. Analysis of twin studies without adopted subjects is considered later.

Fundamental Equations

Behavioral genetic analyses typically lead to equations expressing correlations in terms of genetic and environmental parameters. Consideration of 'identical' (monozygotic, MZ) and 'fraternal' (dizygotic, DZ) twins reared apart (A) and together (T) leads to four equations, the character of which varies somewhat with the precise assumptions that are in force. The following set is representative:

$$r_{\mathrm{MZA}} = h^2 + d^2 + i^2 \quad (1)$$

$$r_{\mathrm{DZA}} = 0.5h^2 + 0.25d^2 \quad (2)$$

$$r_{\mathrm{MZT}} = h^2 + d^2 + i^2 + c^2_{\mathrm{MZ}} \quad (3)$$

$$r_{\mathrm{DZT}} = 0.5h^2 + 0.25d^2 + c^2_{\mathrm{DZ}} \quad (4)$$

where h^2 is the additive genetic variance, d^2 is the dominance genetic variance, i^2 is the epistatic genetic variance, c^2_{MZ} is the common or shared environmental variance for MZ twins raised together, and c^2_{DZ} is the common or shared environmental variance for DZ twins raised together. These equations instantiate the model that is the focus of this section. In the present interpretation there are four population correlations (r) on the left, identified by the subscript abbreviations defined above, and five population parameters on the right. Our objective is to use these equations to develop estimators of the parameters based on sample correlations. Before doing this, however, we must make a long digression to define the parameters and discuss the kinds of assumptions that lead to the equations.

All of these variances are relative to the total phenotypic variance of the trait under discussion. Additive genetic variance is associated, fundamentally, with the sum of effects of the gene derived from the mother and the gene derived from the father at a single genetic locus. *Dominance* is the interaction of these effects. However, it is clear that most if not all of the traits discussed here are influenced by many genetic loci, and our h^2 and d^2 correspond to the sums of additive and dominance effects over all contributory loci. Variance i^2 associated with multilocus interaction is epistatic. Dominance and epistasis are termed 'nonadditive effects'. Remarkably, it is possible to see the signature of such low-level effects in molar correlational data.

Additive genetic variance, h^2, is called 'heritability' or 'narrow heritability'. The sum of all three genetic variances is termed 'broad heritability' and denoted $h_b{}^2$. According to eqn [1], this quantity equals r_{MZA}.

Both genetic variation and environmental variation contribute to behavioral variation. Behavioral genetics usually treats environmental variation as the sum of two uncorrelated components. The first reflects parts of the environment such as parents, home, neighborhood, and schools that are shared by twins reared together. The second represents such things as unique friends and unique experiences that are not shared. The extent of the latter is indexed by yet other parameters, u^2_{MZ} and u^2_{DZ}. It is assumed that only shared experiences contribute to psychological similarity. That is why only shared environmental variances appear on the right in eqns [3] and [4].

Obviously, different twins may have unshared experiences with common parents, houses, neighborhoods, and schools, and such experiences would be recorded in the unshared column, if it were really necessary to tally them up. Fortunately, it is not. Although effects of particular environmental variables can be studied (e.g. Caspi *et al*., 2000), behavioral genetics can and usually does make inferences about aggregate shared and unshared environmental effects without explicitly measuring their constituents.

Measurement error (m^2) is a source of differences between twins' test scores. It is sometimes

implicitly included in unshared environmental variance, but it can be separated from other sources of uniqueness if the reliability of the test is known.

Newcomers to this subject may be puzzled by the 'squares' in these parameters. The unsquared genetic and common environmental parameters are correlations of a single individual's test score with an underlying genetic or environmental component. The squares arise in correlations between relatives because the 'path' between the relatives has two links: from one relative to a common genetic or environmental factor and then on to the other relative.

Now that we have defined the parameters that appear in eqns [1] to [4], we can begin to discuss the equations themselves. The 0.5 coefficient of h^2 in eqns [2] and [4] reflects the fact that DZ twins share half their genes, on average. The 0.25 coefficient of d^2 is explained by the observation that, at a single locus with only two alleles, 0.25 is the probability that two twins receive copies of exactly the same gene from both parents, and thus inherit precisely the same genotype at this locus.

Absence of covariances on the right-hand sides reflects the assumption that all sources of variation are uncorrelated. Also absent by assumption are genotype–environment interaction and effects of assortative mating. It is easy to think of concrete environmental factors (such as parental education, in the case of cognitive ability) that are definitely correlated with children's genotype. This brings out the point that the environmental factors in our equations should not be thought of as composites of concrete, directly measurable, environmental factors, but rather as akin to regression residuals, so that they are, by construction, uncorrelated with genetic 'main effects'. It is possible that the oversimplified model instantiated in our equations may allow interactions and correlations involving concrete environmental factors to be disproportionately absorbed into genetic terms.

Substantial assortative mating is known to occur for some of the traits to which we will apply the model (e.g. cognitive ability). The main defense that can be offered for omitting it, and for our other questionable assumptions, is that the model gives an adequate fit to most of the data to which we will apply it. This is not a strong justification, since MZA twin correlations are based on relatively small numbers of twin pairs, so the corresponding tests of goodness of fit are not very powerful. Thus, successful fits can be regarded only as showing that our assumptions are probably not flagrantly inappropriate.

Fitting the Model

One's first impulse is to replace population correlations in the equations by sample correlations, and solve the resulting linear equations for the quantities on the right. The solutions would then be estimators of the corresponding population parameters. However, this is not feasible because there are five unknowns but only four equations. Fortunately, it is possible to eliminate one unknown by reparameterization, in such a way that our ability to test interesting hypotheses is not seriously compromised. This involves appending half of d^2 to h^2 and the other half to i^2, yielding the new parameters $h_d^2 = h^2 + 0.5d^2$ and $i_d^2 = i^2 + 0.5d^2$. These parameters can respectively be called 'intermediate heritability' (since it lies between broad and narrow heritability) and 'nonadditivity'. In terms of these quantities, eqns [1] to [4] take the following attractive form:

$$r_{\mathrm{MZA}} = h_d^2 + i_d^2 \tag{5}$$

$$r_{\mathrm{DZA}} = 0.5h_d^2 \tag{6}$$

$$r_{\mathrm{MZT}} = h_d^2 + i_d^2 + c_{\mathrm{MZ}}^2 \tag{7}$$

$$r_{\mathrm{DZT}} = 0.5h_d^2 + c_{\mathrm{DZ}}^2 \tag{8}$$

These equations are easily solved for the four parameters on the right in terms of the correlations on the left, and it is somewhat amusing to apply the resulting formulas to sample correlations to obtain parameter estimates. However, this 'equation-solving' approach is a sterile exercise, since it does not yield a test of the framework within which the estimation takes place. Consequently, one does not know whether the estimates are of any interest. Thus we will proceed directly to a more modern model-fitting approach, in which we explicitly test various hypotheses about the parameters, and implicitly test the underlying framework. The specific hypotheses to be tested are:

- equal environments assumption (EEA): $c_{\mathrm{MZ}}^2 = c_{\mathrm{DZ}}^2$
- no common environmental effects: $c_{\mathrm{MZ}}^2 = c_{\mathrm{DZ}}^2 = 0$
- no nonadditive effects (NNE): $i_d^2 = 0$
- no additive or dominance effects: $h_d^2 = 0$
- no genetic effects: $h_b^2 = 0$

Note that $h_b^2 = h_d^2 + i_d^2$, so the last hypothesis is equivalent to the conjunction of the two preceding it. Note also that, if EEA fails, one expects $c_{\mathrm{MZ}}^2 > c_{\mathrm{DZ}}^2$.

Any of these hypotheses reduces the number of parameters to less than four, so eqns [5] to [8] cannot be solved exactly. Instead, one accepts

approximate equality in place of exact equality, and seeks parameter values that yield the best approximate solution. Different estimation methods correspond to different overall measures of the approximation error. We will use the error function and associated tests described by Loehlin (1989) in a slightly different context. The computer programs MX and LISREL provide other approaches to model fitting (Neale and Cardon, 1992).

INTELLIGENCE AND PERSONALITY

The Swedish Adoption/Twin Study of Aging

The Swedish Adoption/Twin Study of Aging (SATSA) is a study of elderly twins. The basic design and many results are summarized by Pedersen *et al*. (1991), and twin correlations and analyses of different types of variables appear in separate papers (Pedersen *et al*., 1992; Bergeman *et al*., 1993). Table 1 summarizes the results of a reanalysis of a few variables using the approach described above.

Cognitive ability is the first principal component of a number of tests of special abilities, and is thus a variant of intelligence quotient (IQ). The next seven variables in the table are standard dimensions of personality. The type A variable is derived from the famous Framingham type A scale, which measures the degree to which an individual is hard-driving, ambitious, and feels as if he or she is under pressure. Variables 'F-Cohesion' and 'F-Control' relate to the twins' recollections of the warmth and strictness of the families in which they were raised. Bear in mind that, for twins reared apart, these were different families, so these variables explore the possibility that twins' perceptions of family warmth and strictness may, to some extent, derive from the twin instead of from the family. Variable BMI is the body mass index, a measure of fatness. This is not a personality variable, but relates to eating habits, which are of great psychological interest in connection with eating disorders.

The χ^2 and p-values in the last two columns correspond to tests of the equal environments assumption. This assumption is rejected only for the last variable, 'F-Control'. This rejection confirms that the tests of EEA are not hopelessly insensitive. The parameter estimates given in the table are optimal assuming EEA, and are thus meaningful for all variables except 'F-Control', which will not be considered further. The c^2 parameter in Table 1 is the common value of c^2_{MZ} and c^2_{DZ} under EEA. Asterisks refer to tests of hypotheses that the corresponding parameters are zero, with one, two, and three asterisks indicating $p < 0.05$, 0.01, and 0.001, respectively. These tests are based on increments in the χ^2 goodness of fit index, beyond its value assuming just EEA. The $u^2 + m^2$ values cannot be tested for significance within this framework, but these values are large for all variables except cognitive ability and BMI.

The table confirms the overall conclusions presented in the introduction, which are, in turn, consistent with those of the original SATSA papers. In 10 out of 12 cases, broad heritability is statistically significant whereas common environmentality is statistically insignificant. The reversal of this pattern for 'Agreeableness' indicates that this pattern is not forced by an artefact of the method. The very high estimate of broad heritability for cognitive

Table 1. Components of variation for variables in the Swedish Adoption/Twin Study of Aging

Variable	h^2_d	i^2_d	h^2_b	c^2	$u^2 + m^2$	χ^2	*p*
Cognitive ability	0.55***	0.24	0.79***	0.00	0.21	0.54	0.46
Extraversion	−0.02	0.40***	0.38***	0.12	0.50	2.27	0.13
Neuroticism	0.49***	−0.16	0.33***	0.04	0.63	1.81	0.18
Openness	0.38**	0.11	0.49***	−0.01	0.52	1.24	0.26
Agreeableness	−0.06	0.21	0.15	0.26**	0.59	0.00	1.00
Conscientiousness	0.08	0.21	0.30**	0.12	0.58	2.65	0.10
Impulsivity	0.25*	0.18	0.44***	−0.01	0.57	0.56	0.46
Monotony avoidance	0.27*	−0.05	0.22*	0.03	0.75	0.06	0.80
Type A	0.34**	−0.07	0.27**	0.08	0.65	0.40	0.53
BMI (male)	0.41*	0.26	0.67***	0.08	0.25	0.63	0.43
BMI (female)	0.51***	0.14	0.65***	0.01	0.34	0.02	0.90
F-Cohesion	0.61***	−0.16	0.44***	0.14*	0.41	0.31	0.58
F-Control						7.89	0.01**

BMI, body mass index. Probability: *, $p = 0.05$; **, $p = 0.01$; ***, $p = 0.001$. See text for details of variables and parameters.

ability is consonant with values obtained for IQ in other studies involving adult MZAs (Neisser *et al*., 1996).

Negative estimates arise because the χ^2 minimization routine varied $a = h_d^2$, $b = i_d^2$, etc., without restricting these quantities to positive values. In no case would the fit have been significantly worse if the negative quantity had been assumed to be zero, so the negative values should simply be regarded as negligible.

Of the 11 variables with significant h_b^2, 9 had h_d^2 but not i_d^2 significant, suggesting that genetic variation is mainly additive. One, 'Conscientiousness', had neither h_d^2 nor i_d^2 significant, leaving us little basis for inference about the distribution of broad heritability among its three components. Finally, 'Extraversion' had i_d^2 but not h_d^2 significant, suggesting that genetic variation is mainly epistatic. (Dominance and epistasis do not contribute to parent–child correlation, so they contradict the common misconception that genetic effects are always revealed by parent–child resemblance.)

We close this section with the results of analysis of a variable from the Minnesota study of twins reared apart (Bouchard *et al*., 1990), by the methods used in Table 1. There is a personality scale called 'well-being' that is, roughly speaking, a measure of happiness. For this scale, EEA was not rejected, $c^2 = -0.04$ is negligible, and $h_b^2 = 0.48$. Happiness in adults thus appears to be highly heritable (Lykken and Tellegen, 1996).

Studies Involving Only Twins Reared Together

Monozygotic twins reared apart are uniquely informative, but they are scarce. The analyses reported in Table 1 involved between 44 and 95 pairs of such twins. This leads to high variability of estimates and low power of tests. On the other hand, dizygotic and monozygotic twins reared together are plentiful, so it is not surprising that studies involving only twins reared together are the mainstay of traditional behavioral genetics. Unfortunately, the gain in precision from large samples is matched by a loss of generality due to the necessity of extra assumptions. For a feeling for the difficulties involved, consider

$$h_{\text{est}}^2 = 2(r_{\text{MZT}} - r_{\text{DZT}}) \tag{9}$$

the traditional rough-and-ready estimator of heritability using sample correlations of twins reared together. For population correlations,

$$2(r_{\text{MZT}} - r_{\text{DZT}}) = h_b^2 + i_d^2 + 2\left(c_{\text{MZ}}^2 - c_{\text{DZ}}^2\right) \tag{10}$$

as a consequence of eqns [7] and [8], so h_{est}^2 will tend to overestimate h_b^2 when EEA fails or nonadditive effects are present.

There is a companion formula, $2r_{\text{DZT}} - r_{\text{MZT}}$, that is traditionally used to estimate common environmental variance. According to eqns [7] and [8],

$$2r_{\text{DZT}} - r_{\text{MZT}} = c_{\text{DZ}}^2 - i_d^2 - \left(c_{\text{MZ}}^2 - c_{\text{DZ}}^2\right) \tag{11}$$

so the companion formula has a tendency to underestimate c_{DZ}^2 when EEA or NNE fails. Sizeable negative values of the companion formula strongly suggest that use of h_{est}^2 is inappropriate, but one sees many instances in the literature where this warning has not been heeded.

Modern behavioral genetics uses model-fitting techniques in place of h_{est}^2 (see, for example, Loehlin, 1992), but model-fitting analyses, like their less sophisticated predecessors, typically assume EEA and sometimes also NNE.

MENTAL ILLNESS

A characteristic feature of many studies of mental illness is a categorical 'sick versus well' classification of each patient's condition. In place of twin correlation for (say) IQ, we have concordance for (say) schizophrenia, estimating the likelihood that a second twin is affected given that the first twin is affected. Concordances carry some of the same intuitions as correlations, but they are not the kinds of correlations to which behavioral genetic theory can be directly applied.

The liability threshold model provides a simple bridge from concordances to behavioral genetics. According to this model, there is a normally distributed liability, L, to the condition under consideration, and the condition is manifested if and only if L exceeds a threshold parameter T. Assuming that the distribution of liability has a mean of zero and a standard deviation of 1, one can estimate T from the prevalence of the condition.

Assuming a bivariate normal distribution of twins' liabilities, computer programs like PRELIS can estimate the liability correlations corresponding to concordances. These are sometimes referred to as *tetrachoric* correlations, and their variances are different from those of ordinary, Pearson, correlations. Taking account of this, behavior genetic analyses of liability correlations can be done in a manner analogous to behavior genetic analyses of Pearson correlations. In particular, there are older studies that calculate h_{est}^2 from liability correlations.

Modern studies apply model-fitting techniques via LISREL or MX to twins reared together, usually assuming EEA and, often, NNE. Substantial

differences between MZ and DZ liability correlations are invariably associated with substantial heritability in these analyses. Such differences have been found for autism, attention deficit hyperactivity disorder, depression, bipolar disorder, and schizophrenia (McGuffin and Martin, 1999).

There is great uncertainty concerning the extent of possible genetic contributions to bulimia and anorexia (Fairburn *et al*., 1999). Though it is not a mental illness, it is interesting to note that McGue and Lykken (1992) have reported a heritability estimate of 0.525 for liability to divorce.

THE SEARCH FOR DEFINITE GENES

Traditional behavioral genetics operates at a tremendous level of abstraction (some might call it vagueness). It provides information only about aggregate quantities, though these quantities are of considerable interest. Some results are available showing behavioral effects of specific genes. In the future, one expects increasing emphasis on studies seeking to demonstrate such effects.

Specific gene effects on behavior vary greatly in size. One imagines that many genes affect each of the personality and ability dimensions, as well as most of the psychopathologies, considered above. If many genes contribute to a dimension, most of these contributions will be small and thus relatively hard to detect.

There are a number of cases where variation at a single genetic locus has drastic effects on the organism, including mental retardation. An especially interesting case of such a single major gene effect is phenylketonuria, in which neither of the alleles at a certain locus on chromosome 12 supports production of an enzyme that breaks down phenylalanine. The consequent build-up of the latter substance in the brain causes mental retardation. Such build-up and retardation can be prevented by a special diet. Since the genetic deficiency leads to a behavioral deficiency only in the presence of a certain environment (normal diet), this is an example of a genotype–environment interaction. At a more basic level, phenylketonuria illustrates that genetic involvement in a behavioral deficiency does not imply that the deficiency is immutable. One of the major thrusts of modern medical science is to discover environmental compensations (medicines) for genetic conditions.

Definite genes, or small chromosomal regions, have been implicated with various degrees of certainty in the following conditions: early-onset and late-onset Alzheimer disease, autism, bipolar disorder, dyslexia, and schizophrenia (McGuffin and Martin, 1999; Owen and Cardno, 1999). For example, the apolipoprotein E ε4 allele appears to be associated with late-onset Alzheimer disease. Information from the human genome project will doubtless make great contributions to this rapidly developing area.

EVOLUTION

Although we have considered genetic variability in a number of psychologically relevant dimensions, we have not considered genetic variability in fitness or reproductive success, the additive component of which directly controls evolution. In so far as certain traits are prevalent today, it is natural to think that they might somehow have conferred enhanced fitness long ago. Such evolutionary speculation is among the most powerful heuristics in biological science in general and evolutionary psychology in particular. Although the disciplines that incorporate it are flourishing, it is important to be aware that inference from present predominance to past superior fitness is fallible. This can be shown by examples involving variation controlled by a single genetic locus with only two alleles, A_1 and A_2. Assuming random mating, the heterozygotic genotype A_1A_2 has relative frequency $2p(1-p)$, where p is the relative frequency of the A_1 allele. Thus, regardless of its fitness, the relative frequency of A_1A_2 cannot exceed 1/2. In fact, it is not difficult to construct examples where the homozygote A_1A_1 predominates after many generations of evolution, even though the heterozygote is the fittest genotype.

Variants of such simple genetic examples can be constructed within the frameworks of W. D. Hamilton's kin selection theory and J. Maynard Smith's (1982) evolutionary game theory, two of the pillars of evolutionary psychology (Norman, 1981). A frequently cited part of Hamilton's theory suggests that one can predict the evolutionary fate of altruistic and selfish genotypes just by examining their inclusive fitnesses, but it turns out that superior inclusive fitness is not sufficient for asymptotic predominance of, say, an altruistic genotype, if that genotype is heterozygotic. Similarly, the standard version of Maynard Smith's criterion for an evolutionarily stable strategy (ESS) depends entirely on fitness 'payoffs', irrespective of genetic structure. However, a behavioral strategy exhibited by a heterozygote cannot be evolutionarily stable, regardless of associated payoffs, since a population composed entirely of heterozygotes will give rise to a mixed population in the next generation.

These examples represent relatively minor blemishes on these theories, but the examples do suggest that the theories should be applied with caution.

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