A Brief Introduction to Quantitative Methods in Human Behavior Genetics and a Sampler of Findings

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Resumé:

V minulosti bylo hlavním důvodem pro provádění genetických studií chování jakýchkoliv lidských psychologických znaků stanovit, zda je správné nebo nesprávné ignorovat genetické faktory při studiu vlivu prostředí na daný psychologický znak. Nyní máme dostatečné empirické doklady, které jsou schopny přesvědčit všechny, s výjimkou extrémních skeptiků, že prakticky každý psychologický znak je do určité míry ovlivňován genetickými faktory. Turkheimer (2000) tvrdí, že prvním zákonem behaviorální genetiky je premisa: „Všechny znaky lidského chování jsou dědičné". Ve skutečnosti je toto tvrzení jednoduše manifestací obecnějších objevů kvantitativní genetiky, zvláště pak zjištění, že jakýkoliv měřitelný charakter organismu by měl být dědičný. Ve své učebnici kvantitativních genetických metod Lynch a Wlech (1998), která se týká především neetologických dat, demonstrují širokou škálu dědičných znaků. Znamená to, že téměř každý zkoumaný znak u většiny druhů má nenulovou dědičnou hodnotu.

Introduction

In the past a major reason for carrying out a behavior genetic study of any human psychological trait was to establish whether or not it was legitimate to ignore the influence of genetic factors when studying environmental influences on that trait. There is now sufficient empirical evidence to convince all but the most extreme skeptic that virtually every reliably measured psychological trait is influenced by genetic factors to some degree. Turkheimer (2000) has asserted that the first law of behavior genetics is that "All human behavioral traits are heritable". Actually, this claim is simply a manifestation of a broader finding in quantitative genetics, namely that any character of an organism one chooses to measure will be heritable. Lynch and Walsh (1998, p. 174), in the definitive textbook on quantitative genetic methods and referring primarily to non-behavioral traits, claim that "if ones sole interest in performing a quantitative-genetic analysis is to demonstrate that the character of interest is heritable, there is probably little point in expending the effort. The outcome is virtually certain. Almost very character in almost every species that has been studied intensively exhibits nonzero heritability".

There are two major consequences of the first law of behavior genetics. First, much contemporary social science research is uninterpretable because it assumes genetic factors are of no importance and its influence is ignored in the research design (Harris, 1998; Scarr, 1997). Secondly, theories constructed on the basis of these findings must be clearly wrong or at best misleading regarding causal mechanisms (Bouchard, 2004).

The argument, made above, regarding the uninterpretability of many social science findings is contingent on genetic influences being of sufficiently magnitude that they make a diference – they are large enough. How large is large enough? This question can be answered in a straightforward manner. All we need do is compare behavior genetic findings to typical findings in the social sciences. Hemphill (2003) has reported the distribution of correlations (effect sizes) for two large meta-analyses of the psychological literature (Psychological Assessment and Treatment). The results were quite similar in both domains so they were combined. The lower third of the distribution ranged from -.08 to .17. The middle third of the distribution ranged from .18 to .29 and the upper third of the distribution ranged from .29 to .78. These three ranges might well be called Small (<.20), Medium (.20 to .30) and Large effects (>.30). Quite independently Lubinski and Humphreys (1997) have pointed out that Cohen's (1988) effect sizes ((SD) differences (or ds)) of .20 – small, .50 – medium and .80 – large. Correspond to correlations of .10, .24 and .37 respectively. Hemphill's empirical results and Cohen's largely intuitive criteria converge nicely. By these criteria it will be obvious that the behavior genetic findings fall mostly in the medium to large categories.

Twin and Adoption Methodology

Genetic and environmental influence on a trait can be estimated empirically by fitting models to the observed variances and covariances between different kinds of kin such as monozygotic (MZ) and dizygotic (DZ) twins, parents and children, etc. Figure 1 illustrates one of the simplest models of this type. This is the path diagram for the correlation between two parallel psychological tests. It is called the Hoyt reliability (Hayes, 1973; Hoyt, 1941). This correlation is computed via analysis of variance and is called an intraclass correlation as opposed to the ordinary Pearson correlation. The intraclass correlation is simply a Pearson correlation computed with every pair of individuals taken in every possible order. If one were to compute a Pearson correlation for a group of monozygotic twin pairs (say 50 pairs) one would get a slightly different correlation every time the pairs were ordered differently. One could solve this problem by entering each pair in each order. This is the so-called double-entry correlation. The intraclass correlation is a better solution.

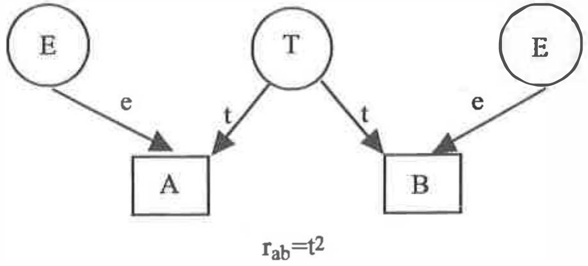

Figure 1. Path Diagram of the Hoyt Reliability Coefficient

The test scores (measured phenotypes shown in boxes) are represented by A and B. The latent psychological construct (unmeasured constructs are always shown in circles) is T and represents the individual's true score. The cause of the correlation between the observed scores is the true score and its influence is manifest through the paths t and t. Note if the paths are standardized we speak of correlations, if the paths are unstandardized we speak of covariances. To keep things simple we will deal with correlations in this paper. Models fitting of covariances is, however, the more standard procedure (Neale, Boker, Xie, & Maes, 1999). The rules of path analysis specify that the correlation between the phenotypes is computed by multiplying the paths that link them. In this instance $(t \times t) = t^2$. Thus the correlation between the two forms of the test is directly interpreted as a variance (t^2). This is an important point. The correlations represented by the path diagrams below are variance estimators, variance due to underlying latent constructs, and are not squared. The widely cited rule that correlations must be squared to estimate variance applies only to the correlation between two measured variables (phenotypes) and is called the coefficient of determination.

The path diagram in Figure 2 represents the correlation between monozygotic twins reared apart (MZA). This is precisely the same model as Figure 1 only we show two sources of variance (causes), the genetic influence for MZA twin 1 and MZA twin 2, as correlated 1.00. This correlation represents our knowledge from biological theory that monozygotic twins share all their genes. The twins are thus treated as parallel forms of a test. Thus the correlation is (h x h) x 1.00 = h^2.

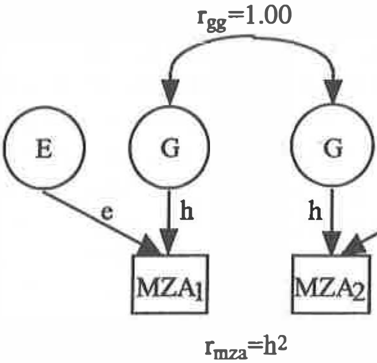

Figure 2. Path Model for Monozygotic Twins Reared Apart

This model makes the explicit assumption that the trait relevant environments of the twins are not correlated. This idea is easily represented in a model as well, but first let us turn to the path model for monozygotic twins reared together (MZT) shown in Figure 3.

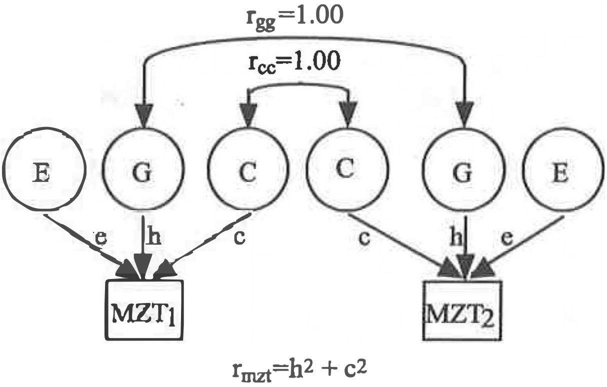

Figure 3. Path Model for Monozygotic Twins Reared Together

This model differs from Figure 2 in that a new latent construct has been added, specifically common or shared environmental influence (C). The environmental influence that makes twins (both monozygotic and dizygotic) similar is defined as common environmental influence. Environmental influence that is not shared is called unshared or idiosyncratic. The correlation between dizygotic twin reared together would be represented by setting the correlation between the G's in Figure 3 to rgg=.5. The equation would then read rdzt=.5h^2 + c^2.

Note that (rmzt – rdzt)=.5h^2. This is the essence of the simplest form of the twin method—the "so-called" Falconer heritability. It assumes that all genetic influence is additive and that shared trait relevant environmental influence is the same for MZ and DZ twins (the equal trait relevant environment assumption). The models make these assumptions explicit. Critics of quantitative behavior genetics have argued that the only genetics involved here is the assignment of the values 1.00 and .50. Actually this is correct, although I might add that more complex models can set values for dominance, and other values for more distant kin (e.g., .125 for cousins, etc.). I don't understand why this is a criticism as any environmental model, if it is to be testable, must also set path values. Consider the environmental path diagram in Figure 4. This diagram represents the similarity between Unrelated Individual Reared Together (UT).

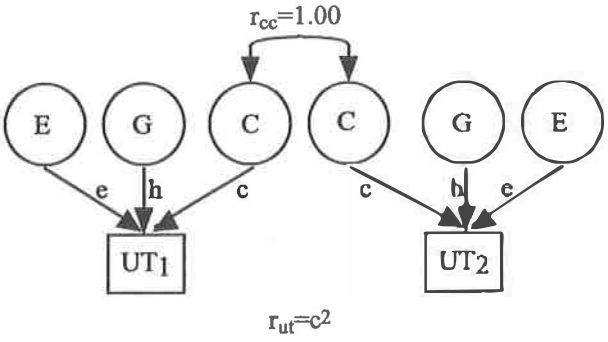

Figure 4. Path Diagram for Unrelated Individuals Reared Together

Just as with MZT and DZT twins it assumes that there is common or shared environmental influence that makes the individuals similar. Just as the MZA correlation estimates genetic influence directly (assumes the G's are correlated 1.00) the UT correlation assumes the C's are correlated 1.00, consequently (c x 1.00 x c) = c^2. Modeling simply makes these assumptions about genes and/or environments explicit. As mentioned earlier, for a long time psychologists simply assumed that the similarity between siblings was due to the fact that they shared a common environment and that the correlation between the G's was zero (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). That this set of assumptions was rarely tested was shown clearly by Scarr (1985; 1997) who provided a number of striking examples of how behavior genetic models explain data much better than simple environmental models that fail to take genetic factors into account.

It is important to note that any well specified (and therefore testable) theory must make assumptions. The trick is to create additional designs that make it possible to test the assumptions. If a variety of designs, which make different assumptions, converge on similar results then one can have more confidence in the results. As Scarr (1981) has put it, "There are flaws in all the studies reported....From my point of view, the most important fact is that the flaws of one study are not the same as those of another; there are nonoverlapping cracks in the evidence. ..Each study can be criticized for its lack of perfection, but laid on top of one anther, the holes do not go clear through" (p. 528).

The Scarr quotes makes it clear that in the domain of human behavior genetics there are no perfect experiments. All studies have flaws. We interpret the correlations in the above designs are "causal" because they are based on "quasi experiments". The MZ and DZ twins are experiments of nature. Nature has provided the MZ twins with all the same genes whereas the DZ twins have one-half of their genes identical by descent. The UT pairs are an experiment of society where they do not share genes but do share a full dose of common environment. Since the experiments are far from perfect, thus the term "quasi", it is necessary to evaluate the assumptions. This can be done directly or as indicated above by carrying out studies with complementary weaknesses and strengths. Consider the assumption underlying the study of MZA twins. Critics assert that adoption is not a random process and that it is unlikely that such twins have been placed in environments at randomly, a requirement of a well conducted experiment. This argument is only partly correct. In fact randomization is only necessary with regard to trait relevant factors. Placement with regard to environmental characteristics that do not influence the trait under study is irrelevant. Consider the hypothetical possibility that the MZA twin pairs have been placed and reared in homes that were painted the same color. One pair in green homes, another in red homes, etc. No one would argue that placement in homes painted the same color was a cause of MZA twin similarity in personality, interests or mental ability. The reason is simple. No one believes that the color of the home one groups up in is a relevant causal variable. Placement must occur for a plausible causal variable and the causal influence of the variable must be tested. The most powerful way of testing the influence of a purported set of causal variables is the UT design shown in Figure 4. If being placed in similar homes is the reason MZA twins are alike on a trait, genetic factors being irrelevant, then unrelated individuals reared together should be very similar as they experience a placement coefficient of 1.00. Keep in mind also that this treatment is multivariate (placement occurs on a very large number of features of the environment), powerful and long term, being experienced daily over the course of years. Notice that this experiment works because the study participants are unrelated (adopted). The average IQ correlation for unrelated individuals reared together and assessed as adults is .04 (Bouchard, 1998).

The UT design makes assumptions just like every other design. First it assumes that the individuals have not been placed. In this instance we are concerned with similarity in the background of the children. Perhaps, either inadvertently or by plan, children from parents with higher IQ's (or SES background) are placed in higher SES families and children from parents with lower IQ's (or SES background) are placed in lower SES families. If genes do influence IQ then correlation between parental SES and child's IQ would be inflated artificially. Secondly, we assume that the children have been exposed to the range of environments to which we wish to generalize. Stoolmiller (1998; 1999) has argued that adoption studies have failed to sample the full range of environments in which children are raised (restriction of range) and this may well be true. It may also be true that the range of genotypes put up for adoption is restricted. The degree to which range restriction is a problem is an open question (Loehlin & Horn, 2000). An excellent study which has implemented the UT design, with additional controls, and which involves an entire population is Teasdale and Owen (1984).

We can generalized the UT model in order to provide a test of the hypothesis that placement is an important bias in the study of MZA twins. On the assumption that genes are irrelevant we can consider MZA twins as unrelated individual reared in correlated environments. This model is shown in Figure 5. We simply remove the correlation between the genotypes and insert a correlation between their environments. We must now estimate the correlation ree and the magnitude of the e path.

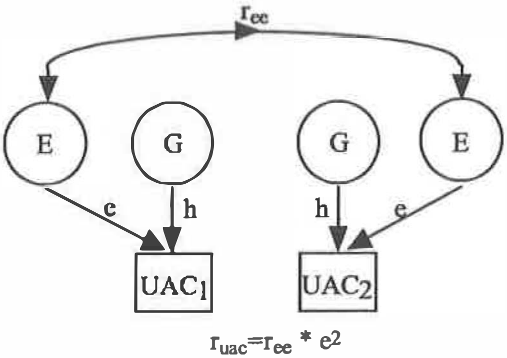

Figure 5. Path diagram for Unrelated Individuals Reared Apart in Correlated Environments (MZA twins with the assumption that genes are irrelevant)

An approximation of ree can be obtained by asking the twins to report on their child rearing. Examples of the use of this model are provided for intelligence in (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990) and religiousness in (Bouchard, McGue, Lykken, & Tellegen, 1999).

Behavior genetic models have not escaped criticism. The best rebuttal of most of the germane criticisms I have seen is (Sesardic, 2005).

Estimates of Genetic Influence on Human Psychological Traits

Table 1. provides a number of examples of the magnitude of genetic influence on a wide range of psychological traits as well as references to the original studies from which the data was extracted.

Table 1. Estimates of Genetic Influence on a Broad Array of Psychological Traits Reported in the Recent Scientific Literature and References to the Primary Source of Data.

Trait	Genetic Influence	Data Source
Personality (Adults)		
Big Five		Bouchard, et al (2001)
Extraversion	.54	
Agreeableness (aggression)	.42	
Conscientiousness	.49	
Neuroticism	.48	
Openness	.57	

Big Three		Finkel, et al (1997)
Positive Emotionality	.50	
Negative Emotionality	.44	
Constraint	.52	
Intelligence		Boomsma, et al (2002)
By age in Dutch cross-sectional twin data		Posthuma, et al (2002)
Age 5	.22	
Age 7	.40	
Age 10	.54	
Age 112	.85	
Age 16	.62	
Age 18	.82	
Age 26	.88	
Age 50	.85	
In old age (>75 yrs. Old)	.54-.62	Finkel, et al (1998)
Psychological Interests		Betsworth, et al (1994)
Realistic (Outdoor work)	.36	
Investigative (Scientific)	.36	
Artistic	.39	
Social	.37	
Enterprising (Business)	.31	
Conventional	.38	
Work Values and Job Satisfaction		
Values		Keller (1992)
Achievement	.56	
Comfort	.31	
Status	.43	
Altruism	.18	
Safety	.42	
Autonomy	.34	
Job Satisfaction		Arvey, et al (1994)
Intrinsic	.23	
Extrinsic	near zero	
General	.16	
Owl vs. Lark		
Morningness	.54	Hur, et al (1998)
Social Attitudes		Bouchard, et al (2004)
Conservatism		Eaves, et al (1997)
Under age 20 years	near zero	
Over age 20 years	.45-.65	

Right Wing Authoritarianism (adults)	.50-.64	McCourt, et al (1999)
Religiousness		
16 year-olds	.11-.22	Boomsma, et al (1999)
Adults	.30-.45	Bouchard,et al (1999)
Specific Religion	near zero	D'Onofrio, et al (1999)

Conclusion

Genetic influence on psychological traits is pervasive, even for traits widely believed to be shaped predominantly by family processes (e.g., social attitudes) and of such a large magnitude that this fact must be taken into account by researchers and theorists alike else their studies and theories will be at best uninformative or at worst seriously misleading.

References:

Arvey, R. D., McCall, B., Bouchard, T. J., Jr., & Taubman, P. (1994). Genetic influence on job satisfaction and work values. Personality and Individual Differences, 17, 21–33.

Betsworth, D. G., Bouchard, T. J., Jr., Cooper, C. R., Grotevant, H. D., Hansen, J. C., Scarr, S., et al. (1994). Genetic and environmental influences on vocational interests assessed using adoptive and biological families and twins reared apart and together. Journal of Vocational Behavior, 44, 263–278.

Boomsma, D. I., de Geus, E. J. C., Van Baal, G. C. M., & Koopmans, J. R. (1999). A religious upbringing reduces the influence of genetic factors on disinhibition: Evidence for interaction between genotype and environment on personality. Twin Research, 2, 115–125.

Bouchard, T. J., Jr. (1998). Genetic and environmental influences on adult intelligence and special mental abilities. Human Biology, 70, 257–279.

Bouchard, T. J., Jr. (2004). Genetic influence on human psychological traits: A survey. Current Directions in Psychological Science, 13, 149–151.

Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota study of twins reared apart. Science, 250, 223–228.

Bouchard, T. J., Jr., McGue, M., Lykken, D. T., & Tellegen, A. (1999). Intrinsic and extrinsic religiousness: Genetic and environmental influences and personality correlates. Twin Research, 2, 88–98.

Cohen, J. (1988). Statistical power analysis for the behavior sciences. Hillsdale, NJ: Erlbaum.

Collins, W. A., Maccoby, E. E., Steinberg, L., Hetherington, E. M., & Bornstein, M. H. (2000). Contemporary research on parenting: The case for nature and nurture. American Psychologist, 55, 218–232.

D'Onofrio, B. M., Eaves, L. J., Murrelle, L., Maes, H. H., & Spilka, B. (1999). Understanding biological and social influences on religious attitudes and behaviors: A behavior genetic perspective. Journal of Personality, 67, 953–984.

Eaves, L. J., Martin, N. G., Heath, A. C., Schieken, R., Meyer, J., Silberg, J., et al. (1997). Age changes in the causes of individual differences in conservatism. Behavior Genetics, 27, 121–124.

Finkel, D., Pedersen, N. L., Plomin, R., & McClearn, G. E. (1998). Longitudinal and cross-sectional twin data on cognitive abilities in adulthood: The Swedish adoption/twin study of aging. Developmental Psychology, 34, 1400–1413.

Harris, J. R. (1998). The nurture assumption: Why children turn out the way they do. New York: Free Press.

Hayes, W. L. (1973). Statistics for the social sciences (2 ed.). New York: Holt, Rinehart & Winston.

Hemphill, J. F. (2003). Interpreting the magnitude of correlation coefficients. American Psychologist, 58, 78–79.

Hoyt, C. (1941). The reliability estimated by analysis of variance. Psychometrika, 6, 153–160.

Hur, Y.-M., Bouchard, T. J., Jr., & Lykken, D. T. (1998). Genetic and environmental influence on morningness-eveningness. Personality and Individual Differences, 25, 917–925.

Keller, L. M., Arvey, R. D., Bouchard, T. J., Jr., Segal, N. L., & Dawis, R. V. (1992). Work values: Genetic and environmental influences. Journal of Applied Psychology, 77, 79–88.

Loehlin, J. C., & Horn, J. M. (2000). Stoolmiller on restriction of range in adoption studies: A comment. Behavior Genetics, 30, 245–247.

Lubinski, D., & Humphreys, L. G. (1997). Incorporating general intelligence into epidemiology and the social sciences. Intelligence, 24, 159–202.

Lynch, M., & Walsh, B. (1998). Genetics and analysis of quantitative traits. Sunderland, Mass: Sinauer.

McCourt, K., Bouchard, T. J., Jr., Lykken, D. T., Tellegen, A., & Keyes, M. (1999). Authoritarianism revisited: Genetic and environmental influence examined in twins reared apart and together. Personality and Individual Differences, 27, 985–1014.

Neale, M., Boker, S. M., Xie, G., & Maes, H. H. (1999). Mx: Statistical modeling (5 ed.). Box 126 MCV, Richmond, VA 23298: Department of Psychiatry.

Posthuma, D., de Geus, E. J. C., & Boomsma, D. I. (2002). Genetic contributions to anatomical, behavioral, and neurophysiological indices of cognition. In R. Plomin, J. C. DeFries, I. C. Craig & P. McGuffin (Eds.), Behavioral genetics in the postgenomic era. Washington, DC: APA Books.

Scarr, S. (1981). Having the last word. In S. Scarr (Ed.), Race, Social Class, and Individual Differences in IQ (pp. 523–532). Hillsdale, NJ: Erlbaum.

Scarr, S. (1985). Constructing psychology: Making facts and fables for our times. American Psychologist, 40, 499–512.

Scarr, S. (1997). Behavior genetic and socialization theories of intelligence: Truce and reconciliation. In R. J. Sternberg & E. L. Grigorenko (Eds.), Intelligence: Heredity and environment (pp. 3–41). New York: Cambridge University Press.

Sesardic, N. (2005). Making sense of heritability. Cambridge: Cambridge University Press.

Stoolmiller, M. (1998). Correcting estimates of shared environmental variance for range restriction in adoption studies using a truncated multivariate normal model. Behavior Genetics, 28, 429–441.

Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. Psychological Bulletin, 125, 392–409.

Teasdale, T. W., & Owen, D. R. (1984). Heritability and familial environment in intelligence and educational level – a sibling study. Nature, 309, 620–622.

Truett, K. R., Eaves, L. J., Meyer, J. M., Heath, A. C., & Martin, N. G. (1992). Religion and Education as mediators of attitudes: A multivariate analysis. Behavior Genetics, 22, 43–62.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. Current Directions in Psychological Science, 9, 160–164.