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Genetic influence on human intelligence (Spearman's *g*): How much?

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Abstract
The history and conceptual background of the heritability statistic is briefly discussed. The construct of heritability is embedded in the method of structural equation modeling widely used in modern population genetics and in human behavior genetics. The application of structural equation modeling to behavioral phenotypes is shown to be a useful and informative analytic tool, as it implements the research strategy of 'strong inference'. I describe the application of 'strong inference', via the use of structural equation models in the domain of human intelligence, and demonstrate its utility as a means of refuting well formulated scientific hypotheses. The construct of Spearman's *g* is shown to be a strongly confirmed scientific hypothesis. Genetic and environmental influences are shown to influence *g* differentially over time, with shared environmental influences predominating early in life, but dissipating to near zero by adulthood. The hypothesis of substantively significant genetic influence on adult *g* is documented by multiple lines of evidence and numerous replications.

Keywords: *Intelligence, heritability, twins, adoptees*

Introduction

The heritability coefficient indexes genetic influence on a quantitative trait. It was originally derived, but not named, in the context of population genetics by Sewall Wright (1921) and Ronald Fisher (1918), both of whom had strong interests in agricultural genetics, evolution, and genetic influence on biological characteristics including human behavioral traits (Provine 1986; Mayo 2004). The term 'heritability' comes to us from the agricultural geneticists J. L. Lush (Bell 1977). Its initial use was to allow estimation of 'breeding values' in order to predict 'response to selection' (Thompson 2008; Visscher et al. 2008). The statistic we call 'broad heritability' or 'broad sense heritability' is a theoretical dimensionless population parameter that tells us the proportion of total phenotypic (measurable) variance

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(σ^2_P) in a trait due to all genetic influences (σ^2_G). Thus the formula:

$$h^2_{\text{broad sense}} = (\sigma^2_G)/(\sigma^2_P).$$

Behavior geneticists conceptualize it as a descriptive statistic applicable to a population. As such it has all the advantages and drawback of any descriptive statistic derived to describe a population. It describes the relative influence of genes and environments on a trait for the population studied. The degree to which it might vary from sample to sample and environment to environment is an empirical question, not a drawback of the statistic and part of the focus of this paper.

Since some genetic factors contribute to the phenotypic variance of a trait but do not influence the breeding value, agricultural geneticists are interested in a narrower definition called 'narrow heritability', which includes only the proportion of variance due to genes that act in an additive manner and called additive genetic variance (σ^2_A). Thus the formula:

$$h^2_{\text{narrow sense}} = (\sigma^2_A)/(\sigma^2_P).$$

The basic idea underlying additive genetic variance is that many genes of small effect sum up to influence a trait. With modern genomic techniques such genes have become detectable. For example, flowering time in corn varies from one line of corn to another by as much as 45 days. Researchers have found 50 genes that influence this trait but no one gene influences the trait by more than 3 days (Pennisi 2008).

Genetic variance not due to additive factors include Mendelian dominance (interaction between alleles at the same locus, σ^2_D), and epistasis (interaction between alleles at different loci, σ^2_I). In humans dominance and epistasis are difficult to distinguish in practice. They are often simply called non-additive genetic variance (σ^2_{NA}).

There are, of course, environmental sources of variance. One such source, of considerable interest to psychologists, is variance that is common to specifiable groups, for example biological siblings and adopted siblings. The environmental influence that makes siblings similar (including monozygotic and dizygotic twins) is called shared environmental variance or common environmental variance (σ^2_C). The residual environmental variance that acts to make, for example, siblings different, is called idiosyncratic environmental variance (σ^2_E) and generally includes error variance.

Sources of variance that depend on both genetic and environmental factors taken together include genotype $\times$ environment interactions ($\sigma^2_{G,E}$) and genotype by environment correlations ($\sigma^2_{G \times E}$). Interactions can be quite simple (ordinal) and accounted for (removed) by changing the scale of measurement or quite complex and not removable by scaling. Variance due to genotype by environmental correlations can be of various sorts (i.e. *Passive* – Children with more genes for high IQ are raised by parents who provide better environments; *Reactive* – Children with more genes for impulsiveness elicit more punishment from their parents; *Active* – Children with more genes for high IQ seek out intellectual environments).

How do we estimate these effects?

The heritability of traits is generally estimated using quantitative genetic models. These models are examples of the structural equation models that are commonly used in the social sciences (Evans et al. 2002). The models, while built on simple Mendelian principles, can and do incorporate contemporary ideas discovered by molecular geneticists (Posthuma

et al. 2005). The models have their limitations, as do any methods in science. They do, however, have one highly desirable feature. They implement the principles of strong inference (Platt 1964) requiring the investigator to state hypotheses in an explicit fashion – the hypotheses are testable/refutable (Bouchard, 2009; Epstein 2008). Quantitative genetic models can be formulated to allow testing of almost any genetic or environmental hypothesis.

Figure 1 shows some of the simplest structural equation models, under some of the simplest assumptions, in the form of path diagrams (Li 1975). Figure 1a illustrates a widely used statistic in psychology, namely the Hoyt reliability coefficient (Hoyt 1941). It illustrates the fact that the intraclass correlation (r_{ab}) between two parallel tests (A and B), shown in squares to indicate measured variables, is explained by the underlying latent

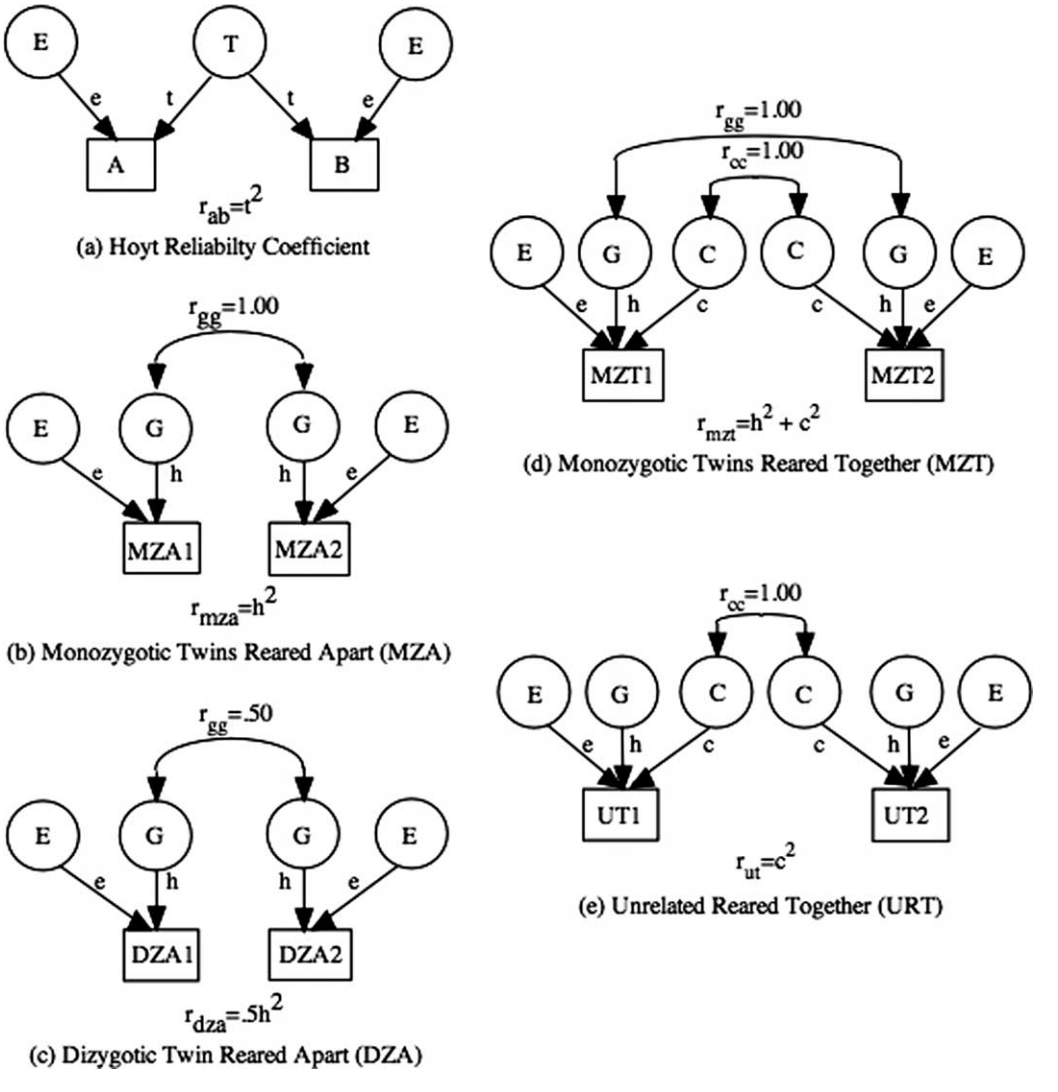

Figure 1. Path diagrams for (a) Hoyt reliability, (b) monozygotic twins reared apart, (c) dizygotic twins reared apart, (d) monozygotic twins reared together and (e) unrelated individuals reared together.

variable called the 'true score (t)'. By the rules of path analysis we simply multiply the paths that connect the measured variables. Note that the correlation directly estimates the variance due to t, it is a variance estimator. In this instance the E in the circle (also a latent trait) represents error variance unique to each test. A simple genetic example is monozygotic twins reared apart (MZAs) and the path diagram is shown in Figure 1b. This path diagram simply quantifies our intuitive understanding of why relatives should be similar or different. Monozygotic twins are genetic clones, consequently if a number of them are randomly assigned to environments we have a combination of two experiments, an experiment of nature (twins) and an experiment of society (adoption). If genes influence the trait under consideration, say intelligence, the correlation computed on their test scores will indicate the amount of variance due to genes, indicated in the Figure 1b as h^2. The symbol h^2 stands for heritability or variance accounted for by genetic factors. In this instance it is the broad heritability as monozygotic twins share all sources of genetic variance. In this and the remaining models the E stands for unshared environmental variance plus error variance.

Figure 1c shows the model for dizygotic twins reared apart (DZAs). Since dizygotic twins share half their segregating genes the correlation estimates one-half the additive genetic ($\frac{1}{2}\sigma^2_A$) effect plus one-quarter of the dominance effect ($\frac{1}{4}\sigma^2_D$) plus a small amount of epistatic effect. These latter non-additive effects are generally small and ignored in the ordinary twin design as they cannot be estimated separately with any accuracy.

Figure 1d shows the model for monozygotic twins reared together (MZT). They could be alike for two reasons. The first being genes, as in the case of MZA twins. The second being due to the fact that they are reared together. This later effect is called a 'common environmental effect' thus the symbol c^2. The correlation between MZT twins is made up of two parts and therefore confounds genetic and common environmental influences. We can, however, subtract the MZA correlation from the MZT correlation and thus estimate c^2.

If we assume that MZT and DZT twins have similar environments (the equal environments assumption where the c^2 for both types of twins is assumed to be the same) the difference between the two types of twins estimates one-half the genetic effect. It is easy to work out additional informative comparisons. Gene by environment interactions and correlations are ignored in these simple models. More elaborate structural equation models make it possible to estimate these joint effects as well as the relationship between them. Currently most such effects have been shown to be small in magnitude. In addition there are some formidable measurement problems (Eaves 2006). Johnson (2007) provides an elegant exposition of these issues.

Figure 1e illustrates an important non-genetic or environmental path model. These are unrelated individuals reared together (URTs). Conceptually they are the opposite of MZA twins, who share all their genes but not a common environment, and the correlation between them directly estimates the variance due to 'common environmental effect' (c^2). Here we have to assume the participants have been exposed to the full range of trait relevant environments (no restriction of range), an assumption we have to make with MZA twins as well. While it is seldom stated, all the models assume sampling of the full range of genotypes as well.

Many different complications can be added to these models, including test of assumptions necessary in the simpler models, and the parameters specified by the models can be estimated given the appropriate kinships and sample sizes.

Why study genetic influence on psychological traits?

There are three main reasons for studying genetic influence on psychological traits. The first is simple scientific curiosity; to discover how things work. Human are biological organisms and under many of the same influences as the rest of the organic world. The second reason is also simple; more accurate knowledge of the underlying biological mechanisms that influence behavior will improve the quality and comprehensiveness of psychological theories. The third reason is that like every other organism on the face of the earth, human have evolved. The mechanisms underlying evolution are largely genetic mechanisms and they need to be incorporated into our theories of 'evolved psychological mechanisms'.

Almost every human quantitative psychological trait that has been well measured and subjected to a behavior genetic analysis has a substantial genetic component. Turkheimer (2000) has called this the 'First Law of Behavior Genetics'. Actually, it is a special case of what I have called the 'First Law of Quantitative Genetics' (Bouchard 2007) as, 'Almost every character in almost every species that has been studied intensively exhibits nonzero heritability.' (Lynch and Walsh 1998). This paper demonstrates how multiple lines of evidence, characterized as a strong inference strategy, can be used to make a compelling case for a high degree of genetic influence on human intelligence, a construct many believe to be one of the most important tools in psychology's armamentarium (Gottfredson 2003; Deary et al. 2004; Gray and Thompson 2004).

A brief commentary on biases in twin studies

While twin studies continue to be a very powerful design, like any other method the twin design is not perfect. It is, however, very powerful and highly informative (Martin et al. 1997). A key assumption of the standard twin method is that monozygotic twins are genetically identical and that phenotypic differences are due to environmental factors. Somatic mutations have always been understood to be a possible source of differences between monozygotic twins, but they were considered rare and not often studied. A recent study of copy number variants in somatic cells suggests they may be more common than previously believed (Bruder et al. 2008).

DNA methylation of cytosines and modification of histones is known to influence molecular signals that influence gene transcription. These epigenetic processes are also known to differ between monozygotic twins. There are now a number of studies that suggest these processes may help explain behavioral differences between monozygotic twins (Petronis et al. 2003; Kan et al. 2004; Kaminsy et al. 2008). In addition gene expression can be considered a complex quantitative trait under some degree of genetic influence (Zahang et al. 2008).

The phenomena discussed above add to the long list of processes that bias twin studies in the direction of underestimating genetic influences, well documented by Price many years ago (Price 1950, 1978) and seldom mentioned by critics of the twin method.

A common criticism of the twin method is that a meaningful proportion of monozygotic twins are monochorionic, rather than dichorionic (Phillips 1993; Prescott et al. 1999). This means that they share the same placenta during gestation thereby allowing the twins to exchange blood and all the biological influencing factors (hormones, etc.) it may contain. The assumption of critics is that monochorionic twins may be more similar due to prenatal environmental influences than dichorionic twins and this may lead to overestimation of

genetic influence. All dizygotic twins are dichorionic. The assumption to be tested is that shared blood and other factors involved in the exchange influence psychological traits. This hypothesis (the influence of chorion type) can be modeled, and this has been done for IQ with an epidemiological sample of 8- to 14-year-olds in Belgium. No effect was found for total IQ although a very modest effect was found for two subscales (Jacobs et al. 2001). The authors point out that the effects found have confidence intervals so close to zero they should be replicated before too much confidence is placed in them.

As noted above a critical assumption of the ordinary twin method is the equal trait relevant environment assumption – that MZ and DZ twins experience similar trait relevant environments. The key term in the assumption is trait relevant. MZ and DZ twins can experience quite different environments, but as long as the different environments do not influence the trait under study, they are irrelevant. This argument is often raised by critics who neglect to mention that the assumption has been repeatedly tested and found to be tenable for many traits (Bouchard 1993; Klump et al. 2000; Eaves et al. 2003). Nevertheless, informative as they may be, twin studies should be supplemented by studies of other informative kinships whenever possible.

Human intelligence

The domain of human intelligence is one of the best places to examine genetic influence on a human trait because it is characterized by a well understood construct, and numerous research designs have been applied using some very large representative samples. It provides an example of what can be done with other human traits. There exists a broad general factor of human ability called Spearman's *g* (Neisser et al. 1996; Jensen 1998; Deary 2003; Gray and Thompson 2004). The previously widespread belief that different intelligence tests yield estimates of quite different intelligences is simply false. Virtually any well-developed multi-scale intelligence test measures the same underlying *g* factor (Johnson et al. 2004, 2008).

Genetic influence on *g*

As I have always clearly acknowledged, the heritability estimates I will be presenting should not (Bouchard et al. 1990, p. 227) 'be extrapolated to the extremes of environmental disadvantage still encountered in society' nor do they imply that IQ cannot be enhanced. They do, however, provide a *causal* explanation of a considerable amount of the IQ variance found in populations raised in the ordinary range of environments currently experienced by most people in advanced industrial societies (Sesardic 2005; Tal 2009).

I will make the case for a strong genetic influence on human intelligence. I am not alone in this claim. According to Plomin (2003, p. 108), 'The case for substantial genetic influence on *g* is stronger than for any other (Mackintosh 1998) human characteristic.' One might argue that stature is more heritable, but for adult stature the findings are surprisingly similar. For example in large twin studies conducted in Minnesota and Finland the broad heritability of stature was 0.75 and 0.79 for men, respectively and 0.72 and 0.77 for women, respectively (Silventoinen et al. 2004). As I will show, the results for adult intelligence are in the same range. My conclusions are also similar to those of Deary et al. (2006) who

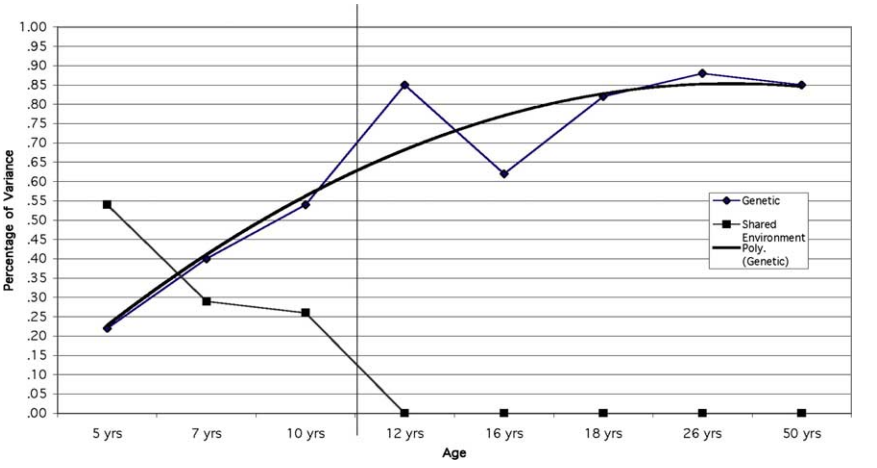

Figure 2. Estimates of genetic and shared environmental influence on IQ by age – Dutch Twins (from Boomsma et al. 2002). NB. The age scale is not linear.

reviewed some of the same literature. The heritability of IQ does, however, vary as a function of age and environmental quality.

The influence of age on the heritability of intelligence

Our research group was one of the first to summarize a large body of data showing systematic age effects on the heritability of IQ (McGue et al. 1993), a fact that we had failed to pay attention to in an earlier review (Bouchard & McGue 1981) as had others (Erlenmeyer-Kimling and Jarvick 1963; Nichols 1978). Wilson (1978), however, had reported on this effects for his large sample of twins in the Louisville twin study and I believe it should be called the Wilson Effect (cf. also Matheny 1990).

One of the clearest empirical demonstrations of this effect has been provided by Dorrett Boomsma's research group at the Free University of Amsterdam. Her group's data (cross-sectional), based on Dutch twins is shown in Figure 2.

A polynomial fits the estimates of genetic influence very well. The broad heritability estimates for the ages 18–50 are above 0.80. This means that over 80% of the variation in IQ in these groups is due to genetic factors.

It is often argued that twin samples are self-selected volunteer samples and do not sample the entire range of environments in which twins develop. We now have an answer to that problem. Ian Deary's group in Edinburgh have shown, 'Large, consistent estimates of the heritability of cognitive ability in two entire populations of 11-year-old twins from Scottish Mental Survey of 1932 and 1947' (Benyamin et al. 2005). The data are shown in Table I where they are compared to a comparably aged sample from the Minnesota Twin Family Study.

The Scottish twins were not volunteers. They were simply included in a complete population survey. The instrument used was the Moray House Test No. 12, which is an excellent multi-scale instrument (Deary et al. 2000). There is little difference between the two Scottish samples so the combined estimate for heritability of 0.70 is a good overall estimate. The combined estimate of shared environment is 0.21. The heritability of IQ

Table I. Estimates of heritability, shared-environmental influence and non-shared environmental influence on IQ based on twin samples from two entire populations of 11-year-old twins from the Scottish mental surveys of 1932 and 1947 compared to the same estimates from a comparable aged twin sample from the Minnesota Twin Registry.

Sample	Heritability	Shared-environment	Non-shared environment plus error
Scottish population samples			
MHT 1947 sample	0.67	0.26	0.07
MHT 1932 sample	0.73	0.16	0.11
MHT combined sample	0.70	0.21	0.09
Minnesota Twin Registry sample			
WISC MN Twin Family Study 11 years (WISC IQ test)	0.67	0.26	0.07

MHT, Moray House Test; WISC, Wechler Intelligence Scale for Children.
From Benyamin et al. (2005) and Johnson et al. (2005).

(Wechsler Intelligence Scale for Children) for 11-year-olds in the large Minnesota Twin Family Study is 0.67. Common family environmental influence is 0.26 (Johnson et al. 2005), results identical to the 1947 Scottish sample. How do the Scottish and Minnesota data sets compare to the Dutch twin data? I have drawn a line through age 11 in Figure 2. The Dutch data would have predicted a heritability of about 0.62 and shared environmental influence of 0.12. Given that the Scottish data and the Minnesota data are from different populations and different generations and different instruments were used, I would say that the findings are remarkably close. Figure 3 shows data our group published 13 years ago summarizing what was known about age effects on heritability using twin data.

If we draw a line at either year 6 or year 16 we see that, relative to the Scottish population data, the ordinary twin data underestimates heritability (it is about 0.55 vs 0.70) and in this instance overestimates shared-environmental influence (it is about 0.30 vs 0.21). The data in Figure 3 is based on twins and is cross-sectional (confounds age and cohort). There is one large-scale longitudinal study of adoptees and their parents that disentangles these

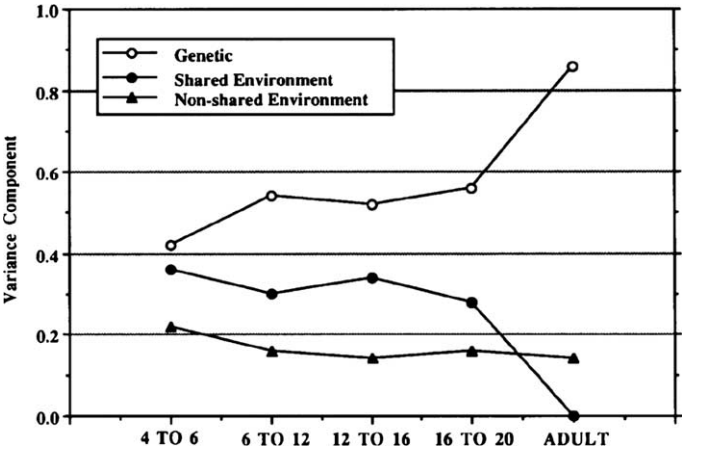

Figure 3. IQ variance component estimates derived from published IQ twin correlations. Estimates are based on the standard assumptions used with the Falconer heritability formula (from McGue et al. 1993).

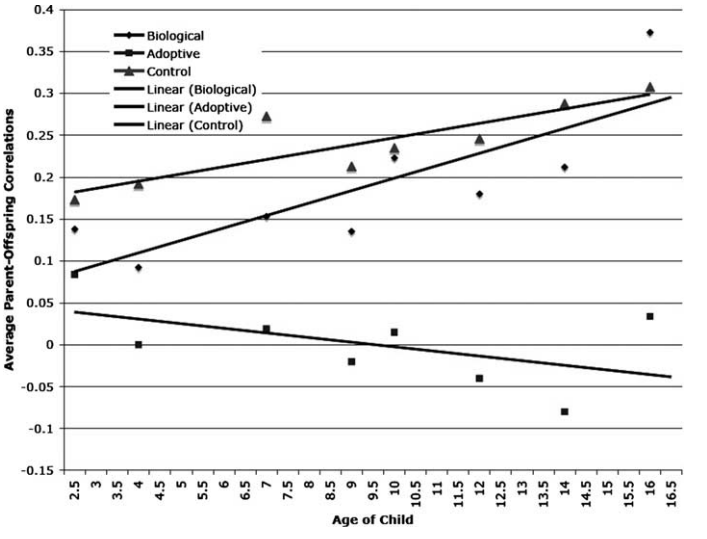

Figure 4. Parent–offspring correlations from the Colorado Adoption Project – first PC (*g*) (from Plomin et al. 1997: Redrawn by the author).

factors, the Colorado Adoption Project (Plomin et al. 1997). This study makes use of longitudinal data contrasting the correlation between biological parents and their children vs adoptive parents and their children. The data shown in Figure 4 reveal the same developmental trend as the cross-sectional data in Figure 2.

We see very little correlation between parents and their children for adoptive families, but a growing correlation with age for a matched control group of biological families and the same thing for an ordinary group of families. The broad heritability of *g* at age 16 is 0.77 and shared environmental influence is 0.01.

The fact that shared environmental influence is near zero in most adult twin studies is quite surprising to most people (Loehlin 2007). The simplest way to demonstrate that this effect is not dependent on the use of twins is to employ a design using unrelated individuals reared in the same home – the URTs shown in Figure 1e. They directly estimate the influence of being reared in the same home (c^2). How similar would you expect such individuals to be? The findings are clear and are shown in Figure 5.

If studied in childhood the correlation is 0.26 but if studied in adulthood the correlation is 0.04, results highly consistent with the data in Figure 2. A striking feature of this figure is the paucity of data in adulthood. It is surprising that there are so few studies of this rather common and exceptionally informative group. Nevertheless, there are a number of drawbacks with this data. Firstly, unlike twins, the pairs of individuals differ in age. Secondly, most of the studies are not longitudinal. Only the data points connected by the arrows are longitudinal. Thirdly, the studies involve adoptees and it has been argued that restriction in range of environments to which adoptees are exposed attenuates the estimate of environmental influence (Stoolmiller 1998, 1999). The first two problems are being addressed using a unique design, the longitudinal study of virtual twins, introduced by Nancy Segal (2000). Virtual twins are same-age pairs of unrelated individuals (one may be a biological child of the parents) reared together since infancy. In 2005 she reported a correlation of 0.26 for 113 such pairs with an average age of 8.1 years (Segal and Hershberger 2005). These are included in Figure 5 as 0.43 ($n = 34$) for Natural–Adoptive pairs and 0.13 ($n = 79$) for Adoptive–Adoptive pairs. The difference is not statistically

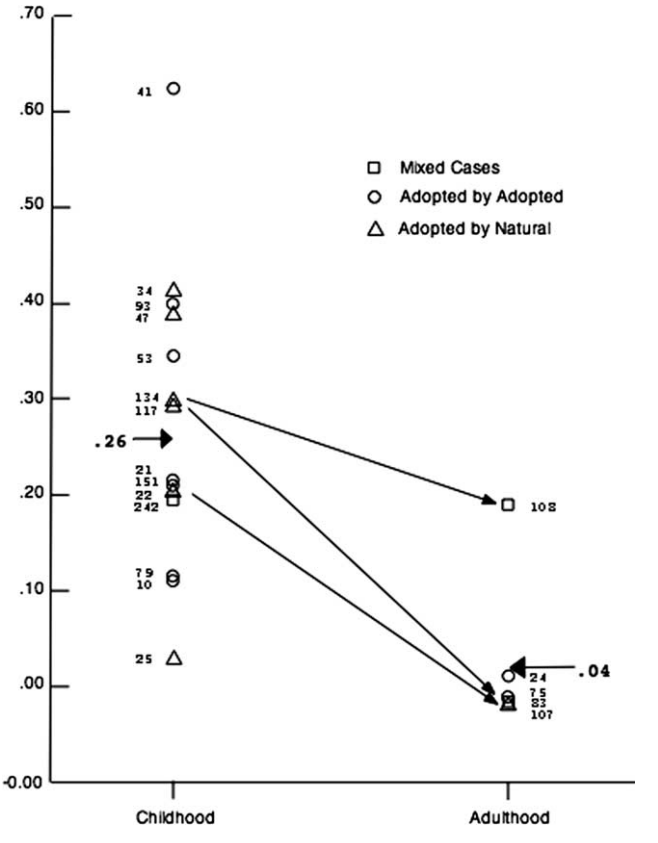

Figure 5. IQ correlations, sample sizes, and weighted mean correlations (arrows) for unrelated individuals reared together organized as pairs measured in childhood and pairs measured in adulthood. Data from longitudinal studies connected by arrows.

significant and the same is true for the entire data set shown in Figure 5. Recently Segal and her team reported on the IQ correlations for 43 such pairs tested at ages 5.11 years of age and retested at 10.77 years of age (Segal et al. 2007). The correlations (direct estimates of shared environmental influence) were 0.30 and 0.11 and are not included in Figure 5. These figures show precisely the expected downward trend so clear in Figure 5. Studies included in Figure 5, but not cited above or below are Burks (1928), Freeman et al. (1928), Leahy (1935), Skodak (1950), Horn et al. (1979), Loehlin et al. (1997), and Scarr and Weinberg (1977, 1978).

There is to my knowledge only one adoption study, with unrelated individuals reared together, that has directly addressed Stoolmiller's assertion that restriction of range is a serious problem for estimates of environmental influence on IQ. That study (McGue et al. 2007) shows that it is not a serious problem for IQ and a number of other variables. According to the authors. 'restriction in range in parent disinhibitory psychopathology and family SES had no effect on adoptive–sibling correlations for delinquency, drug use, and IQ. These data support the use of adoption studies to obtain direct estimates of the importance of shared environment effects on psychological development.' (p. 449). The URT correlation in this study was 0.19 (mean age = about 15 years, $n = 242$) and it is included in Figure 5. Corrected for restriction of range the correlation becomes 0.20. The

authors do, however, make it clear that their results apply to the broad middle class and not to homes characterized by abuse and severe deprivation. Loehlin and Horn (2000) also discuss limitations of Stoolmiller's approach to this question.

What is the basis for age effects on heritability?

We cannot be certain regarding causation but in younger individuals it is associated with biological maturation. We have known for a longtime, from EEG work, that the brain undergoes change with development (Bell and Fox 1992). Modern tools in the domain of neuroscience have shown us in a much more precise manner just where the brain undergoes these changes. Sowell et al. (1999) 'describe *in vivo* documentation for a temporal and spatial progression of post adolescent maturation into the frontal lobes, highlighting the potential importance of frontal/striatal maturation to adult cognition' (p. 861). Changes in brain morphology with age have also been reported by Gogtay et al. (2004), Shaw et al. (2006) and Giedd et al. (1999). Toga and Thompson (2005) have provided a recent review of this literature.

Most studies of IQ and brain morphology have focused on measures of total brain volume. It is possible to study the relation of specific areas of the brain to IQ and ask if there is a genetic correlation between them. This is done by looking at the correlation between measures of specific areas of the brain in one twin and correlating them with the IQ score of the co-twin. The results are very clear.

> Genes influenced individual differences in left and right superior occipitofrontal fascicle (heritability up to 0.79 and 0.77), corpus callosum (0.82, 0.80), optic radiation (0.69, 0.79), corticospinal tract (0.78, 0.79), medial frontal cortex (0.78, 0.83), superior frontal cortex (0.76, 0.80), superior temporal cortex (0.80, 0.77), left occipital cortex (0.85), left postcentral cortex (0.83), left posterior cingulate cortex (0.83), right parahippocampal cortex (0.69), and amygdala (0.80, 0.55). Intelligence shared a common genetic origin with superior occipitofrontal, callosal, and left optical radiation WM and frontal, occipital, and parahippocampal GM (phenotypic correlations up to 0.35). These findings point to a neural network that shares a common genetic origin with human intelligence (Pol et al. 2006, p. 10235).

Age-related changes in the heritability of IQ and inspection time are discussed in some detail in Edmonds et al. (2008). Lenroot et al. (2009) point out that 'The observation that regions associated with complex cognitive processes such as language, tool use, and executive function are more heritable in adolescents than children is consistent with previous studies showing that IQ becomes increasingly heritable with maturity'. Bergen et al. (2007) review age-related changes in heritability of a number of phenotypes in addition to IQ.

The heritability of IQ in adulthood

Table II shows the results of the five extant studies of MZA twins, who are mostly adults (Newman et al. 1937; Shields 1962; Juel-Nielsen 1980; Pedersen et al. 1992; Johnson et al.

Table II. Intraclass correlations from the five extant studies of monozygotic twins reared apart.

Study	Correlation	Sample (pairs)
USA 1937	0.71	19
Denmark 1965	0.69	12
England 1962	0.75	37
USA 2007	0.73	74
Sweden 1992	0.78	45
Weighted average	0.74	187

See text for sources.

2007). There is very little variability from study to study and they all yield estimates fairly close to that yielded by studies of ordinary twins.

Because twin studies are often accused of overestimating heritability I include the results of a study that does not make use of twins but rather makes use of all adult male siblings who are members of four informative adopted kinships drawn from the comprehensive Danish conscript records. All were born between 1938 and 1947. A fifth group of full siblings reared together was drawn from another study but is comparable to the adopted groups. This study contains one data point used in Figure 5. The correlations are shown in Table III.

The most striking contrast is between Full Siblings Reared Apart and Unrelated Individual Reared Together, 0.47 vs 0.02. The first figure estimates one-half the genetic influence ($h^2 = 0.94$) and the second figure estimates common environment ($c^2 = 0.02$). The difference between Full Siblings Reared Apart and Full Siblings Reared Together (if the later are more similar than the former) also estimates c^2 and in this instance it is 0.05.

Model fitting results, not reported in the paper but computable from data in the tables, yield an h^2 of 0.96 and a c^2 of 0.02. As far as I know this is the highest heritability for IQ ever reported in the scientific literature. For contrast the height data for this sample yields an h^2 of 0.83 and a c^2 of 0.11 supporting the earlier suggestion that IQ is just about as heritable as height in modern industrialized societies. The sample sizes in this study are quite small so the results should be interpreted cautiously. On the other hand the sample is actually quite close to the entire population of adopted individuals for the years of birth chosen, as every male in Denmark is required to register for military service and be tested.

Additional estimates of the heritability of adult IQ, based on twin studies, have recently been reported for samples from Australia 0.69 (Posthuma et al. 2005), from Belgium 0.82 (Jacobs et al. 2007), from Russia 0.86 (Malykh et al. 2005) and from England 0.83 (Edmonds et al. 2008).

Table III. IQ correlations for five Danish male kinship groups ages 18–26.

Full siblings reared apart ($n = 28$)	Maternal half-sibs reared apart ($n = 34$)	Paternal half-sibs reared apart ($n = 30$)	Unrelated individuals reared together ($n = 24$)	Full siblings reared together ($n = 73$)
0.47	0.11	0.30	0.02	0.52

Data compiled from Teasdale and Owen (1984).

Heritability of IQ and the quality of the environment

It is well known that that as the quality of the environment improves morphological traits express themselves more fully. This is well documented for human height and age at menarche although this process appears to be leveling off in more advanced industrialized societies (Cole 2003). The Flynn effect (increasing raw scores on mental ability tests over time) seems to be an example in the behavioral domain and it also appears to be leveling off (Colom et al. 2005; Shayer 2007; Teasdale and Owen 2008) although additional factors may well play a role, including fertility patterns (Rönnlund and Nilsson 2008; Sundet et al. 2008) and heterosis (Mingroni 2007). Less well know is the evidence that suggests that heritability of morphological traits increases with environmental quality (Visscher et al. 2008). Recent studies have demonstrate that heritability of IQ increases with social class (Turkheimer et al. 2003) although all the studies are not consistent (van den Oord and Rowe 1997; Guo and Stearns 2002; van der Sluis et al. 2008).

Conclusion

Multiple independent lines of evidence support the conclusion that human intelligence is under strong genetic influence when assessed in the ordinary range of environments in advanced industrialized societies. None of the lines of evidence is either perfect or foolproof. Nevertheless, as Scarr (1981) has argued in response to earlier criticism of behavior genetic studies, 'the most important fact is that the flaws of one study are not the same as those of another, there are non-overlapping cracks in the evidence. . .Each study can be criticized for its lack of perfection, but laid on top of one another, the holes do not go clear through.' (p. 528). Picking apart individual studies and pointing out possible flaws in the face of a coherent body of evidence within which the hypotheses has already been tested using alternate designs is common, but it is a regressive approach to science (Urbach 1974a,b; Sesardic 2005) and deserves a name. I have called it pseudoanalysis (Bouchard 1982). To put the argument in another way, it is possible to subject the various proposed causes of IQ score variance (often posed as criticisms of behavior genetic designs) to multiple empirical tests (strong inferences strategy) and the consistent result has been failure to support the hypothesis that environmental factors are the sole cause of variance in IQ. Scientists in most disciplines typically chose the most powerful model system available to test scientific hypotheses. The two most powerful model systems available to study human individual differences are the URT design (no genes in common, highly similar rearing environments) and its inverse the MZA design (almost all genes in common, dissimilar rearing environments). These designs show that, in the range of environments under discussion, growing up in the same family is at best a very modest source of adult IQ similarity and that genetic factors are a major source of similarity. All the other behavior genetic designs support this finding. The most parsimonious conclusion that can be drawn from this large and continually expanding body of evidence is that human intelligence, when assessed in the ordinary range of environments in advance industrialized societies, is more highly heritable than psychologists have previously thought.

A high heritability for IQ, however, is not the end point of research on intelligence. It is only a starting point as it does not imply that intelligence is fixed at conception, nor does it imply that differences in measured intelligence between groups with different levels of economic, social, industrial, or academic development are fixed and inevitable. It does, however, vitiate the validity of correlational studies that focus solely on ordinary (non-twin) biological families and

confound genetic and environmental sources of influence on individual differences. Such studies continue to be common despite disclaimers to the contrary (Bouchard, 2009). In addition it strongly justifies the application of molecular genetic approaches to the problem (Butcher et al. 2008; Gosso et al. 2008). The coming of the age of molecular genetics, genomics and the study of genetic network architecture (Rouzic and Carlborg 2007; Benfey & Mitchell-Olds 2008) will add dramatically to our understanding of how genes influence the phenotype of IQ. Findings from these domains can and will be incorporated into quantitative genetic models and there is reason to believe that twin and adoption studies will continue to contribute to our understanding of the genetic roots of Spearman's *g* (Boomsma et al. 2002; Johnson et al., in press).

Note: Haworth et al. (2009) have recently reported an analysis of the combined raw IQ data from six large twin studies (3 from the United States, 1 from the United Kingdom, 1 from Australia and 1 from the Netherlands). This study had a great deal of statistical power and a model with additive genetic influence, shared environment, and non-shared environment provided the best fit for the data from Childhood (Mean age of 9; .41, .33, .26) Adolescence (Mean age of 17; .55, .18, .27) and Young adulthood (Mean age of 17; .66, .16, .19).

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