Heritability of Intelligence Quotient

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The study of the heritability of IQ investigates the relative importance of genetics and environment for variation in intelligence quotient (IQ) in a population. "Heritability", in this sense, "refers to the genetic contribution to variance within a population and in a specific environment" There has been significant controversy in the academic community about the heritability of IQ ever since research began in the 19th century.

IQ is a polygenic trait under normal circumstances according to recent research. However, certain single gene genetic disorders can severely affect intelligence, with phenylketonuria as an example.

Estimates in the academic research of the heritability of IQ have varied from below 0.5 to a high of 0.9. A 1996 statement by the American Psychological Association gave about .45 for children and about .75 during and after adolescence. A 2004 meta-analysis of reports in Current Directions in Psychological Science gave an overall estimate of around .85 for 18-year-olds and older. The New York Times Magazine has listed about three quarters as a figure held by the majority of studies.

Correlations

The relative influence of genetics and environment for a trait can be calculated by measuring how strongly traits covary in people of a given genetic (unrelated, siblings, fraternal twins, or identical twins) and environmental (reared in the same family or not) relationship. The most common method is to consider identical twins reared apart, with any similarities which exists between such twin pairs attributed to genotype. In terms of correlation statistics, this means that theoretically the correlation of tests scores between monozygotic twins would be 1.00 if genetics alone accounted for variation in IQ scores; likewise, siblings and dizygotic twins share on average half of their alleles and the correlation of their scores would be 0.50 if IQ were affected by genes alone. Practically, however, the upper bound of these correlations are given by the reliability of the test, which tends to be 0.90 to 0.95 for typical IQ tests

If there is biological inheritance of IQ, then the relatives of a person with a high IQ should exhibit a comparably high IQ with a much higher probability than the general population. In 1982, Bouchard and McGue reviewed such correlations reported in 111 original studies in the United States. The mean correlation of IQ scores between monozygotic twins was 0.86, between siblings, 0.47, between half-siblings, 0.31, and between cousins, 0.15.

The 2006 edition of Assessing adolescent and adult intelligence by Alan S. Kaufman and Elizabeth O. Lichtenberger reports correlations of 0.86 for identical twins raised together compared to 0.76 for those raised apart and 0.47 for siblings. These number are not necessarily static. When comparing pre-1963 to late 1970s data, researches DeFries and Plomin found that the IQ correlation between parent and child living together fell significantly, from 0.50 to 0.35. The opposite occurred for fraternal twins.

Another summary:

Same person (tested twice) .95
Identical twins--Reared together .86
Identical twins--Reared apart .76
Fraternal twins--Reared together .55
Fraternal twins--Reared apart .35
Biological siblings--Reared together .47
Biological siblings--Reared apart .24
Unrelated children--Reared together .30
Parent-child--Living together .42
Parent-child--Living apart .22
Adoptive parent-child--Living together .19

Heritability and caveats

"Heritability" is defined as the proportion of variance in a trait which is attributable to genotype within a defined population in a specific environment. Heritability takes a value ranging from 0 to 1; a heritability of 1 indicates that all variation in the trait in question is genetic in origin and a heritability of 0 indicates that none of the variation is genetic. The determination of many traits can be considered primarily genetic under similar environmental backgrounds. For example, a 2006 study found that adult height has a heritability estimated at 0.80 when looking only at the height variation within families where the environment should be very similar. Other traits have lower heritabilities, which indicate a relatively larger environmental influence. For example, a twin study on the heritability of depression in men calculated it as 0.29, while it was 0.42 for women in the same study.

Caveats

There are a number of points to consider when interpreting heritability:

Heritability measures the proportion of variation in a trait that can be attributed to genes, and not the proportion of a trait caused by genes. Thus, if the environment relevant to a given trait changes in a way that affects all members of the population equally, the mean value of the trait will change without any change in its heritability (because the variation or differences among individuals in the population will stay the same). This has evidently happened for height: the heritability of stature is high, but average heights continue to increase. Thus, even in developed nations, a high heritability of a trait does not necessarily mean that average group differences are due to genes. Some have gone further, and used height as an example in order to argue that "even highly heritable traits can be strongly manipulated by the environment, so heritability has little if anything to do with

controllability." However, others argue that IQ is highly stable during life and has been largely resistant to interventions aimed to change it long-term and substantially.
A common error is to assume that a heritability figure is necessarily unchangeable. The value of heritability can change if the impact of environment (or of genes) in the population is substantially altered. If the environmental variation encountered by different individuals increases, then the heritability figure would decrease. On the other hand, if everyone had the same environment, then heritability would be 100%. The population in developing nations often have more diverse environments than in developed nations. This would mean that heritability figures would be lower in developing nations. Another example is phenylketonuria which previously caused mental retardation for everyone who had this genetic disorder and thus had a heritability of 100%. Today, this can be prevented by following a modified diet which has lowered heritability.
A high heritability of a trait does not mean that environmental effects such as learning are not involved. Vocabulary size, for example, is very substantially heritable (and highly correlated with general intelligence) although every word in an individual's vocabulary is learned. In a society in which plenty of words are available in everyone's environment, especially for individuals who are motivated to seek them out, the number of words that individuals actually learn depends to a considerable extent on their genetic predispositions and thus heritability is high.
Since heritability increases during childhood and adolescence, and even increases greatly between 16-20 years of age and adulthood, one should be cautious drawing conclusions regarding the role of genetics and environment from studies where the participants are not followed until they are adults. Furthermore, there may be differences regarding the effects on g and on non-g factors, with g possibly being harder to affect and environmental interventions disproportionately affecting non-g factors.

Estimates of the heritability of IQ

Various studies have found the heritability of IQ to be between 0.7 and 0.8 in adults and 0.45 in childhood in the United States. It may seem reasonable to expect that genetic influences on traits like IQ should become less important as one gains experiences with age. However, that the opposite occurs is well documented. Heritability measures in infancy are as low as 0.2, around 0.4 in middle childhood, and as high as 0.8 in adulthood. One proposed explanation is that people with different genes tend to seek out different environments that reinforce the effects of those genes.

A 1994 review in Behavior Genetics based on identical/fraternal twin studies found that heritability is as high as 0.80 in general cognitive ability but it also varies based on the trait, with .60 for verbal tests, .50 for spatial and speed-of-processing tests, and only .40 for memory tests.

In 2006, The New York Times Magazine listed about three quarters as a figure held by the majority of studies, while a 2004 meta-analysis of reports in Current Directions in Psychological Science gave an overall estimate of around .85 for 18-year-olds and older.

Shared family environment

There are some family effects on the IQ of children, accounting for up to a quarter of the variance. However, adoption studies show that by adulthood adoptive siblings aren't more similar in IQ than strangers, while adult full siblings show an IQ correlation of 0.6. Conventional twin studies reinforce this pattern: monozygotic (identical) twins raised separately are highly similar in IQ (0.86), more so than dizygotic (fraternal) twins raised together (0.6) and much more than adoptive siblings (~0.0). However, studies of twins reared apart (e.g. Bouchard, 1990) point to a significant shared environmental influence, of at least 10% going into late adulthood. JR Harris suggests that this might be due to biasing assumptions in the methodology of the classical twin and adoption studies.

There are aspects of environments that family members have in common (for example, characteristics of the home). This shared family environment accounts for 0.25-0.35 of the variation in IQ in childhood. By late adolescence it is quite low (zero in some studies). There is a similar effect for several other psychological traits. These studies have not looked the effects of extreme environments such as in abusive families.

The American Psychological Association's report "Intelligence: Knowns and Unknowns" (1995) states that there is no doubt that normal child development requires a certain minimum level of responsible care. Severely deprived, neglectful, or abusive environments must have negative effects on a great many aspects of development, including intellectual aspects. Beyond that minimum, however, the role of family experience is in serious dispute. There is no doubt that such variables as resources of the home and parents' use of language are correlated with children's IQ scores, but such correlations may be mediated by genetic as well as (or instead of) environmental factors. But how much of that variance in IQ results from differences between families, as contrasted with the varying experiences of different children in the same family? Recent twin and adoption studies suggest that while the effect of the shared family environment is substantial in early childhood, it becomes quite small by late adolescence. These findings suggest that differences in the life styles of families whatever their importance may be for many aspects of children's lives make little long-term difference for the skills measured by intelligence tests.

Non-shared family environment and environment outside the family

Although parents treat their children differently, such differential treatment explains only a small amount of non-shared environmental influence. One suggestions it that children react differently to the same environment due to different genes. More likely influences may be the impact of peers and other experiences outside the family.

Lower heritability with lower SES?

The APA report "Intelligence: Knowns and Unknowns" (1995) also stated that:

We should note, however, that low-income and non-white families are poorly represented in existing adoption studies as well as in most twin samples. Thus it is not yet clear whether these studies apply to the population as a whole. It remains possible that, across the full range of income and ethnicity, between-family differences have more lasting consequences for psychometric intelligence."

A study (1999) by Capron and Duyme of French children adopted between the ages of four and six examined the influence of socioeconomic status (SES). The children's IQs initially averaged 77, putting them near retardation. Most were abused or neglected as infants, then shunted from one foster home or institution to the next. Nine years later after adoption, when they were on average 14 years old, they retook the IQ tests, and all of them did better. The amount they improved was directly related to the adopting family's socioeconomic status. Children adopted by farmers and laborers had average IQ scores of 85.5; those placed with middle-class families had average scores of 92. The average IQ scores of youngsters placed in well-to-do homes climbed more than 20 points, to 98."

Stoolmiller (1999) argued that the range of environments in previous adoption studies were restricted. Adopting families tend to be more similar on, for example, socio-economic status than the general population, which suggests a possible underestimation of the role of the shared family environment in previous studies. Corrections for range restriction to adoption studies indicated that socio-economic status could account for as much as 50% of the variance in IQ.

On the other hand, the effect of this was examined by Matt McGue and colleagues (2007), who wrote that "restriction in range in parent disinhibitory psychopathology and family socio-economic status had no effect on adoptive-sibling correlations IQ"

Turkheimer and colleagues (2003) argued that the proportions of IQ variance attributable to genes and environment vary with socioeconomic status. They found that in a study on seven-year-old twins, in impoverished families, 60% of the variance in early childhood IQ was accounted for by the shared family environment, and the contribution of genes is close to zero; in affluent families, the result is almost exactly the reverse.

A study by Nagoshi and Johnson (2005) failed to replicate Turkheimer and colleagues' findings. They concluded that the heritability of IQ did not vary as a function of parental socioeconomic status in the 949 families of Caucasian and 400 families of Japanese ancestry who took part in the Hawaii Family Study of Cognition.

Asbury and colleagues (2005) studied the effect of environmental risk factors on verbal and non-verbal ability in a nationally representative sample of 4-year-old British twins. There was not any statistically significant interaction for non-verbal ability, but the heritability of verbal ability was found to be higher in low-SES and high-risk environments.

Harden and colleagues (2007) investigated adolescents, most 17 years old, and found that, among higher income families, genetic influences accounted for approximately 55% of the variance in

cognitive aptitude and shared environmental influences about 35%. Among lower income families, the proportions were in the reverse direction, 39% genetic and 45% shared environment."
Rushton and Jensen (2010) criticized many of these studies for being done on children or adolescents. They argued that heritability increases during childhood and adolescence, and even increases greatly between 16-20 years of age and adulthood, so one should be cautious drawing conclusions regarding the role of genetics from studies where the participants are not adults. Furthermore, the studies typically did not examine if IQ gains due to adoption were on the general intelligence factor (g). When the studies by Capron and Duyme were re-examined, IQ gains from being adopted into high SES homes were on non-g factors. By contrast, the adopted children's g mainly depended on their biological parents SES, which implied that g is more difficult to environmentally change.
A 2011 study by Tucker-Drob and colleagues reported that at age 2 years, genes accounted for approximately 50% of the variation in mental ability for children being raised in high socioeconomic status families, but genes accounted for negligible variation in mental ability for children being raised in low socioeconomic status families. This gene-environment interaction was not apparent at age 10 months, suggesting that the effect emerges over the course of early development.

Maternal (fetal) environment

A meta-analysis by Devlin and colleagues (1997) of 212 previous studies evaluated an alternative model for environmental influence and found that it fits the data better than the 'family-environments' model commonly used. The shared maternal (fetal) environment effects, often assumed to be negligible, account for 20% of covariance between twins and 5% between siblings, and the effects of genes are correspondingly reduced, with two measures of heritability being less than 50%. They argue that the shared maternal environment may explain the striking correlation between the IQs of twins, especially those of adult twins that were reared apart.

Bouchard and McGue reviewed the literature in 2003, arguing that Devlin's conclusions about the magnitude of heritability is not substantially different than previous reports and that their conclusions regarding prenatal effects stands in contradiction to many previous reports. They write that:

Chipuer et al. and Loehlin conclude that the postnatal rather than the prenatal environment is most important. The Devlin et al. (1997a) conclusion that the prenatal environment contributes to twin IQ similarity is especially remarkable given the existence of an extensive empirical literature on prenatal effects. Price (1950), in a comprehensive review published over 50 years ago, argued that almost all MZ twin prenatal effects produced differences rather than similarities. As of 1950 the literature on the topic was so large that the entire bibliography was not published. It was finally published in 1978 with an additional 260 references. At that time Price reiterated his earlier conclusion (Price, 1978). Research subsequent to the 1978 review largely reinforces Price's

hypothesis (Bryan, 1993; Macdonald et al., 1993; Hall and Lopez-Rangel, 1996; see also Martin et al., 1997, box 2; Machin, 1996).

Dickens and Flynn model

Dickens and Flynn (2001) argued that the "heritability" figure includes both a direct effect of the genotype on IQ and also indirect effects where the genotype changes the environment, in turn affecting IQ. That is, those with a higher IQ tend to seek out stimulating environments that further increase IQ. The direct effect can initially have been very small but feedback loops can create large differences in IQ. In their model an environmental stimulus can have a very large effect on IQ, even in adults, but this effect also decays over time unless the stimulus continues (the model could be adapted to include possible factors, like nutrition in early childhood, that may cause permanent effects). The Flynn effect can be explained by a generally more stimulating environment for all people. The authors suggest that programs aiming to increase IQ would be most likely to produce long-term IQ gains if they taught children how to replicate outside the program the kinds of cognitively demanding experiences that produce IQ gains while they are in the program and motivate them to persist in that replication long after they have left the program.

Regression toward the mean

Regression towards the mean is a statistical phenomenon that occurs when an outcome is determined by many independent factors. If an outcome is extreme, then this occurred because most of the independent factors agreed by chance. This is unlikely to occur again so to the next outcome is likely to be less extreme. If IQ is determined by many factors, genetic and/or environmental, then they must mostly agree in the same direction in order to produce an extreme IQ. The child of a person with an extreme IQ is unlikely to have all the factors agree so similarly so the child is on average likely to have a less extreme IQ.

People in professional occupations have on average 25 points higher IQ than unskilled workers. For their children the difference is 21 points. This is in itself not evidence for genetics or environment since the environment for the children likely differs greatly with it on average being more stimulating for the children of professionals.

Illustration of linear regression on a data set.

The equation asserts that, on average, the IQ of a child tends to the mean IQ of the population. For instance, if the heritability of IQ is 50% and the mean IQ of a population is 100, then a couple with an average IQ of 120 will, on average, have a child with an IQ of 110. Similarly, a couple with an average IQ of 80 will, on average, have a child with an IQ of 90.

It is noted that the above equation relates only statistical averages and is not deterministic. Furthermore, the equation is a general equation based in the inheritance of genetically-based characteristics (in this case, phenotypes), and so it is implicitly assumed that environmental factors are, for the sake of correctly assessing the genetic contribution to IQ, the same across the population.

Operating under the assumption that child and parent are raised in exactly the same environment (unlikely, but usually closer to the truth than in the completely dissimilar environment that the previous equation assumes), h2 can be replaced by h, which is simply the correlation between parent and offspring IQ. In this case, regression towards the mean is no longer partially caused by environmental differences and therefore only by random genetic variation.

Finally, it is important to note that the expected IQ of the offspring is normally distributed around the mean calculated using the above equation, so in many cases regression towards the mean does not actually occur; as the values are normally distributed, there is a chance that offspring IQ will be more deviant from the mean than that of the parental average.

The search for specific genes

Many studies attempting to find loci in the genome relating to IQ have had little success. For example, a study by Robert Plomin using groups of around 100 people investigated 1,842 DNA markers in a high-IQ group and in an average-IQ control group. The study used a five-step replication process to eliminate false positives, and no gene met this rigid criterion for replicability.

The failure to find a specific gene associated with IQ indicates that cognitive abilities are very complex and are likely to involve several genes (polygenic). Some estimate that as much as 40% of all genes may contribute to IQ. The more genes that contribute to a trait the more the trait will be continuous instead of discrete. A 2008 study of 500,000 single nucleotide polymorphisms (SNPs) from 7,089 children did not substantially improve on earlier studies. The study did not find any SNPs that accounted for more than 0.5% of the variance in general intelligence.
Mutations in GDI1 have been linked to X-linked nonspecific mental retardation.
There is "a highly significant association" between the CHRM2 gene and intelligence according to a 2006 Dutch family study. The study concluded that there was an association between the CHRM2 gene on chromosome 7 and Performance IQ, as measured by the Wechsler Adult Intelligence Scale-Revised. The Dutch family study used a sample of 667 individuals from 304 families. A similar association was found independently in the Minnesota Twin and Family Study (Comings et al. 2003) and by the Department of Psychiatry at the Washington University.
Microcephalin and ASPM are two genes that are associated with brain development. Mutations in these genes are associated with microcephaly, and hence they were initially associated with general intelligence. However recent studies have found no association with general cognitive

abilities.
STX1A correlates significantly with intelligence in Williams syndrome patients.
A 2007 study by Caspi and colleagues found that a gene called FADS2 along with breastfeeding adds about 7 IQ points to those with the C" version of the gene. Those with the "G" version see no advantage.
Copy number variation has also been associated with idiopathic learning disability.
There are number of known cases where the homozygotes have severe cognitive deficits and the heterozygotes show a small decrease of IQ. In such cases further alleles are investigated to estimate their influence on IQ. For example, one minor allele of the gene ALDH5A1 is associated with an IQ difference of around 1.5 points.

Between-group heritability

Although IQ differences between individuals are shown to have a large hereditary component, it does not automatically follow that mean group-level disparities (between-group differences) in IQ can be assumed to have a genetic basis. An analogy, attributed to Richard Lewontin, illustrates this point:

Suppose two handfuls are taken from a sack containing a genetically diverse variety of corn, and each grown under carefully controlled and standardized conditions, except that one batch is lacking in certain nutrients that are supplied to the other. After several weeks, the plants are measured. There is variability of growth within each batch, due to the genetic variability of the corn. Given that the growing conditions are closely controlled, nearly all the variation in the height of the plants within a batch will be due to differences in their genes. Thus, within populations, heritabilities will be very high. Nevertheless, the difference between the two groups is due entirely to an environmental factor - differential nutrition. Lewontin didn't go so far as to have the one set of pots painted white and the other set black, but you get the idea. The point of the example, in any case, is that the causes of between-group differences may in principle be quite different from the causes of within-group variation.

Arthur Jensen agrees that this is technically correct but argues that a high heritability increases the probability for that genetics play a role in average group differences.