Genetics of Childhood Disorders: III. Genetics and Intelligence

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Genetic research has made important discoveries about intelligence during the past few decades. To outline some of these findings, I won't spend space on the measurement of intelligence except to say that what I mean by intelligence is general cognitive ability defined as *g*. All reliable and valid tests of cognitive ability intercorrelate at a modest level—*g* is what they have in common. *g* is often assessed as a total score across diverse cognitive tests as in intelligence (IQ) tests, although it is more accurately indexed by an unrotated principal component that best reflects what is in common among the tests. Nearly all genetic data have been obtained using measures developed from this psychometric perspective, primarily IQ tests. One new direction for genetic research on intelligence is to investigate other measures such as information-processing and more direct measures of brain function such as evoked potentials, positron emission tomographic scans, and functional magnetic resonance imaging and to explain how these measures relate to *g*.

g clearly runs in families. The correlations for first-degree relatives living together average 0.43 for more than 8,000 parent–offspring pairs and 0.47 for more than 25,000 pairs of siblings. However, *g* might run in families for reasons of nurture or of nature. In studies involving more than 10,000 pairs of twins, the average *g* correlations are 0.85 for identical twins and 0.60 for same-sex fraternal twins. These twin data suggest a genetic effect size (heritability) that explains about half of the total variance in *g* scores.

Adoption studies also yield estimates of substantial heritability. For example, identical twins reared apart are almost as similar for *g* as identical twins reared together. Adoption studies of other first-degree relatives also indicate substantial heritability, as illustrated below by recent results from the

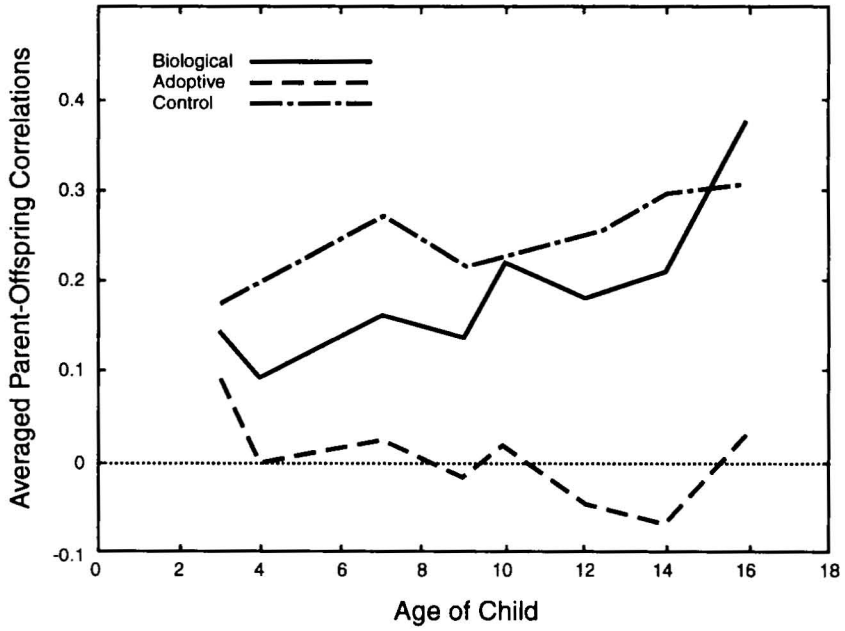

Fig. 1 Parent–offspring correlations between parents' first principal component scores and children's first principal component scores derived from tests of specific cognitive abilities for adoptive, biological, and control parents and their children at 3, 4, 7, 9, 10, 12, 14, and 16 years. The *N*'s range from 27 to 42 for biological fathers, 123 to 195 for biological mothers, 121 to 194 for adoptive parents, and 115 to 216 for control parents. Adapted from Plomin R, Fulker DW, Corley R, DeFries JC (1997), Nature, nurture and cognitive development from 1 to 16 years: a parent–offspring adoption study. *Psychol Sci* 8:442–447 (by permission of publisher).

Colorado Adoption Project (CAP). Model-fitting analyses based on dozens of adoption and twin studies estimate that about half of the total variance can be attributed to genetic factors. Genetic influence on *g* is not only statistically significant, it is also substantial, especially when compared to other research in the behavioral sciences that rarely explains 5% of the variance. Genetic research has moved beyond the question of heritability of intelligence to investigate developmental changes, multivariate relations among cognitive abilities, and specific genes responsible for the heritability of *g*. These 3 issues will now be addressed.

When Francis Galton first studied twins in 1876, he investigated the extent to which the similarity of twins changes over the course of development. Other early twin studies of *g* were also developmental, but this developmental perspective faded from genetic research until recent years. One of the most interesting findings about *g* is that heritability increases steadily from infancy (20%) to childhood (40%) to adulthood (60%). For example, a recent study of twins aged 80 years and older reported a heritability of about 60.

The 20-year longitudinal CAP confirms this finding using the adoption design. CAP is a 25-year study of 245 children separated from their biological parents at birth and adopted in the first month of life. Correlations are shown between *g* scores of the biological parents and their adopted-away children, the adoptive parents and their adopted children, and nonadoptive or control parents and their children matched to the adoptive families. Correlations between nonadoptive parents and children increase from less than 0.20 in infancy to about 0.20 in middle childhood and to about 0.30 in adolescence. The correlations between biological mothers and their adopted-away children follow a similar pattern, indicating that parent–offspring resemblance for *g* is due to genetic factors. In contrast, parent–offspring correlations for adoptive parents and their adopted children hover around zero, which suggests that family environment shared by parents and offspring does not contribute importantly to parent–offspring resemblance for *g*.

Why does heritability of *g* increase during the life span? Perhaps completely new genes come to affect *g* as more sophisticated cognitive processes develop. A more likely possibility is that relatively small genetic effects early in life snowball during development, creating larger and larger phenotypic effects, perhaps as individuals select or create environments that foster their genetic propensities.

There is more, however, to cognitive abilities than *g*. In the widely accepted hierarchical model of cognitive abilities, specific cognitive abilities include components such as spatial, verbal, speed-of-processing, and memory abilities, each indexed by what is in common among several tests of each ability. Less is known about the genetic and environmental origins of individual differences in specific cognitive abilities, but they also appear to show substantial genetic influence, although less than *g*.

A surprising finding concerning specific cognitive abilities is that multivariate genetic analyses indicate that the same genetic factors largely influence different abilities. What this finding means concretely is that if a specific gene were found that is associated with verbal ability, the gene would also be expected to be associated with spatial ability and other specific cognitive abilities. This finding is surprising because it goes against the tide of the popular modular theory of cognitive neuroscience that assumes that cognitive processes are specific and relatively independent of one another. The multivariate genetic results are consistent with a top-down model in which genetic effects of *g* pervade a broad range of cognitive processes. An even more surprising finding in 4 out of 4 studies is that genetic effects on measures of school achievement overlap almost completely with genetic effects on *g*. The converse of this finding of genetic overlap is equally interesting. Although genetics accounts for the overlap between school achievement and *g*, discrepancies between school achievement and *g*, often used to describe underachievers, are largely environmental in origin.

Heritability of complex dimensions such as *g* seems likely to be due to multiple genes of varying but small effect size rather than a single gene that has a major effect. Genes in such multiple-gene systems are called quantitative trait loci (QTLs). Unlike single-gene effects like PKU that are necessary and sufficient for the development of a disorder, QTLs contribute interchangeably and additively like probabilistic risk factors. Traditional methods for identifying single-gene effects are unlikely to succeed in identifying QTLs.

A QTL study applying new genetic approaches to *g* yielded a replicated association in a study comparing groups of children of high *g* and children of average *g*. The gene is insulin-like growth factor-2 receptor (IGF2R) on chromosome 6, which has recently been shown to be especially active in brain regions most involved in learning and memory. The frequency of one of the alleles was twice as high in 2 groups of children with high *g* compared with 2 groups of children with average *g* (about 30% versus 15%).

Identifying replicable QTLs associated with *g* will make it possible to address questions about development, differential diagnosis, and gene–environment interplay through the use of measured genotypes rather than indirect inferences about heritable influence based on familial resemblance. Such QTLs will also provide discrete windows through which to view neurophysiological pathways between genes and cognitive development. As is the case with most important advances, identifying genes for cognitive abilities and disabilities will also raise new ethical issues. These concerns must be taken seriously, but they are based largely on misconceptions about genetic research on complex traits that are influenced by multiple genes as well as multiple environmental factors.

WEB SITES OF INTEREST

http://205.153.39.175/programs/sfkids/showarchive/sfkc.98.01.02.html
http://www.nhgri.nih.gov/HGP/HGP_goals/5yrplan.html

ADDITIONAL READINGS

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