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Current Directions in Psychological Science
1–7
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DOI: 10.1177/09637214251339449
www.psychologicalscience.org/CDPS
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Three Promising Directions in the Study of Intelligence With Genetic Methods

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Abstract
A genome-wide association study (GWAS) tests whether each of several million sites in the human genome is correlated with a trait of interest. For a number of reasons, including replication of GWAS results within families, we can be confident that significant correlations reflect in part the causal effects of DNA-level variation on the studied trait. This level of causal inference, much stronger than in most observational studies, enables some far-reaching conclusions about the antecedents and structure of human intelligence. We discuss some of these conclusions regarding whether brain size affects intelligence and the long-debated issue of how different intelligence tests are related to each other.

Keywords
intelligence, behavioral genetics, genome-wide association studies

A genome-wide association study (GWAS) takes a large sample of individuals—sometimes numbering in the millions—and tests, one by one, whether each of several varying sites across the human genome—often numbering in the millions—is correlated with a trait of interest. Since modern genotyping technology made the GWAS design feasible and enabled its application to the traits of bipolar disorder (Wellcome Trust Case Control Consortium, 2007) and schizophrenia (International Schizophrenia Consortium, 2009) more than 15 years ago, it has provided a valuable complement to the study of resemblance between twins and other relatives that was for so long the mainstay of behavioral genetics.

This article reviews what the GWAS design has uncovered about the trait of intelligence (Table 1). We do not dwell on GWAS findings, such as the extremely large number of associated sites and their tiny effects (Chabris et al., 2015), that apply to most traits. Our focus is on intelligence in particular. Deary (2000) reviewed the research up until that point seeking reductionistic explanations of individual differences in terms of genetics and biology, and the scope of this article is similarly limited.

Genetic Differences Cause Substantial Variation in Intelligence at Least Partially Through Neural Mechanisms

A brief search of Google Scholar will affirm that a GWAS article (e.g., Savage et al., 2018; Trubetskoy et al., 2022) can take only a few years to garner as many citations as a venerable classic of the twin literature (e.g., Bouchard et al., 1990; Gottesman & Shields, 1967). Although some of this disparity is surely the result of a prejudice regarding "molecular" genetics as more "scientific" than kin-based methods, the confidence placed in GWAS results is not all misplaced. This is because there is a specialized study design in GWASs that permits much stronger causal claims than are possible in most nonexperimental research.

Each of us carries two copies of every base pair in the human genome, one inherited from each parent. When we become parents in our own turn, which of

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Table 1. Terminology in the Study of Human Intelligence

Intelligence	Ability to acquire and apply knowledge (Oxford University Press, 2010)
g	Psychometric common factor measured by standardized ability and achievement tests; the same as "intelligence" to the extent that the tests have construct validity (Cronbach & Meehl, 1955; McDonald, 1999), although no item-based standardized test can fully measure up to the concept; embedding test scores within a causal model, of the kind sketched in this article (see Fig. 1), is a kind of construct validation
IQ	Composite of the scores obtained on the various tests within a battery measuring *g*; the same as *g* to the extent that the battery of tests is reliable; whether the different types of test are positively correlated is a component of reliability, but this article addresses the further question of whether the correlations making for measurement of a common attribute are explained by the tests being affected by common causes

the two do we pass on? Mendel discovered not only the paired arrangement of the hereditary units carried by an individual but also that whether it is one or the other member of a given pair transmitted to the individual's offspring is as much of a random 50-50 affair as a good flip of a coin. This means that any genetic differences between parent and offspring (and hence any genetic differences between different offspring of the same parents) are as random as the distinction between treatment and control units in a carefully controlled experiment.

Therefore, as students who have taken a course on research methods might anticipate, whenever we see differences in assayed genotypes within families reliably accompany differences in height, body mass index, cholesterol level, IQ score (Table 1), or whatever it may be, we can be confident that the genetic differences do indeed cause the differences in the trait being studied.

Readers should pause to reflect on this marvelous feature of Mendelian inheritance. Social scientists swarm onto the scene whenever providentially random distributions of lottery wealth, medical insurance, or school vouchers permit inferentially powerful studies of various outcomes, even when there is an even better example of a natural randomized experiment occurring every time that a human life is conceived. Fisher (1952) himself discussed the connections between genetics and experimental design, two fields to which he contributed immensely.

Family units are more practically difficult to sample, but much has already been done, and more should certainly be done in the future. Right now we can say the following. For each of the three individuals in a trio consisting of two parents and their offspring, a predictor of IQ ("polygenic score") can be constructed from a combination of GWAS results and the individual's genotyping data. If the offspring score is used to predict IQ, the parental scores can be used as control variables. This control confines the analysis of the offspring score to its random deviation from the scores of the parents. Without this control, the apparent effect of the polygenic score on IQ might be biased by confounders—just as the apparent positive effect of alcohol on health is biased by differences between light drinkers and teetotalers whose effect is mistakenly attributed to alcohol. For accessible accounts of the confounding differences between bearers of distinct genotypes that might be affecting traits examined in GWASs, readers can turn to Davies et al. (2024).

One study exploiting within-family variation in the polygenic score found that the random deviation of the offspring score from the scores of the parents has an effect on actual IQ about 80% as large as the potentially biased naive effect that does not use the parental scores as controls (Okbay et al., 2022). The greater part of the polygenic score's predictive power, then, reflects the causal effect of genetic variation.

A GWAS-based predictor of this kind aggregating several sites in the genome may tend to weight the most statistically significant sites more heavily. It is also possible to examine within-family variation one site in the genome at a time (Tan et al., 2024). This approach has found that sites with relatively large effects on IQ tend to have biases in the opposite direction, leading to the true effects being on average larger than the naive effects estimated in samples of unrelated individuals (Tan et al., 2024; Young et al., 2022). Tan et al. (2024) even found that the net impact of all biases comes out to a surprising closeness between family-based and population estimates of how much IQ variability is attributable to the genetic variants studied in GWASs (a subset of the genetic variants contributing to the resemblance between twins). Although these results point to the sore need for more family-based studies and population studies of representative samples, they already suggest that the existing statistics from GWASs of IQ can be cautiously relied on for downstream inferences.

Earlier authors have addressed the criticism, originating at least as far back as Jencks et al. (1972), that a

genetic effect on IQ is consistent with the causal path going through a mediator such as physical attractiveness (e.g., Sesardić, 2005). The idea behind this criticism is that there may be genetic effects on some superficial trait that go on to affect how one is treated by others that go on to affect learning, self-esteem, and ultimately performance on an IQ test. What GWAS data add to this discussion are clues to the biological mechanisms through which the most strongly associated sites affect IQ. Much is known about the functions of the genomic regions encompassing any given site, and the overrepresentation of top sites in regions of a given type can render certain hypotheses about mediation less plausible. GWASs of IQ have shown that the top sites tend to act through biological processes such as "regulation of nervous system development, central nervous system neuron differentiation, and regulation of synapse structure or activity" (Savage et al., 2018, p. 913). These results are not what one would expect if attributes such as physical appearance were powerful mediators of genetic effects.

In the next section we turn to a more specific biological attribute that GWAS data *have* implicated as a mediating cause of intelligence.

The Association Between Brain Volume and Intelligence Is Probably Causal

Psychologists studying individual differences have long sought to establish correlations between opposite sides of the Cartesian gap between the physical and the mental (Paterson, 1930). Their efforts have led to modest success. For example, it is now known that the correlation between brain volume and IQ is about .25 (Pietschnig et al., 2022). Because correlations can range in absolute value from 0 to 1, this particular correlation may seem small. But we know of no anthropometric attribute as highly correlated with IQ as brain size, and conversely we know of no psychological trait as highly correlated with brain size as IQ. But, again, let us take ourselves back to research methods: Does correlation here imply causation? Is brain size a cause of IQ, or are they correlated because confounders affect both?

Here is an issue for which traditional methods based on twins and other kinships can complement newer GWAS techniques to produce a fairly definitive resolution. Many of the latter techniques can be thrown off by a form of confounding called "cross-assortative mating," the tendency of individuals with a certain attribute to prefer mates with a certain other attribute (Border et al., 2022; Jensen & Sinha, 1993). An example is the tendency of individuals with higher IQs to prefer mates who are taller. After some generations of such cross-assortative mating, a genome carrying IQ-increasing genes will also tend to carry height-increasing genes at distinct sites (Keller et al., 2013; Tan et al., 2024). How can we be sure that cross-assortative mating is not likewise the explanation of the correlation between brain size and IQ? This possibility can be ruled out by the replication of the correlation within twin or sibling pairs: Does the sibling with the larger brain also tend to have the higher IQ? Whereas under cross-assortative mating a given parent may tend to carry genes increasing both traits, the randomness of Mendelian assortment means that there will be no tendency for an offspring inheriting the gene increasing brain size at a site in the genome at which the parent carries both the increasing and decreasing genes to also inherit the gene increasing IQ at a distant site that is similarly heterozygous. Lee et al. (2019) did in fact find highly significant within-family correlations between IQ and two measures of brain size (MRI-measured volume, external head circumference), putting to rest the idea that cross-assortative mating between the intelligent and the large-brained accounts for this correlation in the wider population.

A trait-level correlation within twin and sibling pairs might still be the result of reverse causation or confounders that vary within families. We can use data from GWASs of both traits to eliminate these alternatives as well, using a method that can be explained intuitively as follows (Lee et al., 2019; O'Connor & Price, 2018; Pickrell et al., 2016).

Suppose that we believe some site in the human genome to be a cause of brain size. Then the site must go on to show a *concordant* effect—that is, an effect of the same sign such that the gene at the site associated with more of one trait is also associated with more of the other—on any trait that is affected by brain size, such as *g* ("general intelligence"; Table 1). This is because of the chain *genetic site* → *brain size* → *g* (Fig. 1). In contrast, any variable ascribed to be a cause of *g* need not show a concordant effect on brain size; there may be genetic effects on *g* acting through biological mechanisms other than brain size (e.g., fine-tuning of synaptic plasticity).

Figure 2 displays the results from a study by Jansen et al. (2020) testing these predictions of a *brain size* → *g* causal link. Each data point represents the effects of a genetic site on brain volume and IQ. Figure 2a displays a perfect concordance of genetic effects: Of the 24 top signals from this GWAS of brain volume, all 24 showed effects on IQ in the same direction. Figure 2b displays a much weaker concordance: Of the 243 top signals from this GWAS of IQ, only 151 (62%) showed effects on brain volume in the same direction. The latter concordance was greater than expected by chance but consistent with the expectation that far from all variants affecting intelligence do so through brain volume.

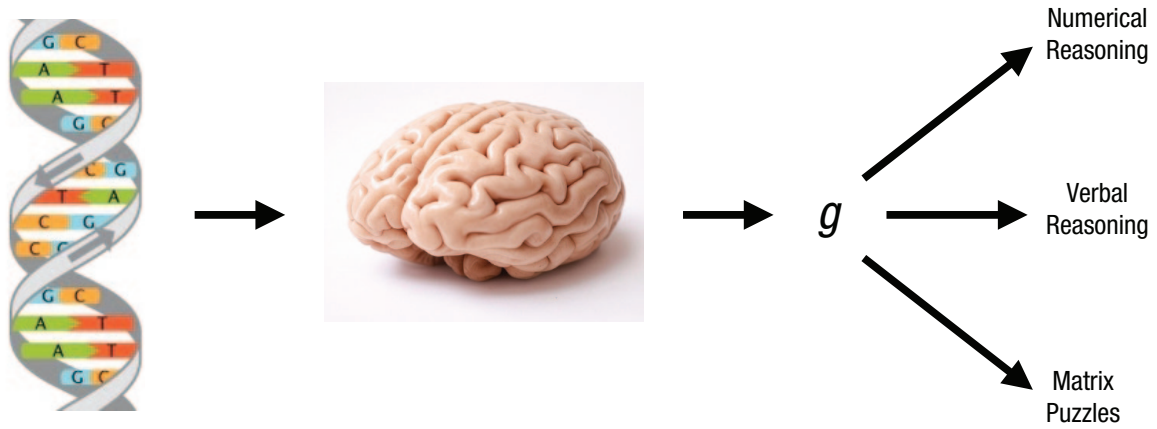

Fig. 1. Causal links between genetic variation, brain size, and intelligence. Genome-wide association studies have mustered preliminary evidence for the causal chains depicted here, in which genetic variation affects brain size, which goes on to affect *g* ("general intelligence"), which acts as a common factor.

As impressive as these results may seem, they must be interpreted judiciously. Other studies have indicated that the pattern in Figure 2a weakens with the addition of genetic sites ascertained from larger GWASs of brain size (Grasby et al., 2020; Nawaz et al., 2022). The best interpretation may be that coarse measures of size such as the volume of the whole brain or the surface area of the cortex are good proxies for some other attribute—possibly the number of neurons in certain critical regions—that is the true causal variable.

The *g* Factor Is Not a Statistical Artifact

Factor analysis is taught in courses more advanced than introductory research methods, but its basics can be simply explained. Assuming that the correlation between two indicators (i.e., items or subtests) reflects their measurement of a common attribute (Fig. 1), factor analysis converts the correlations between all pairs of observed indicators into quantitative estimates of each indicator's sensitivity as a measure of the common attribute. It is also possible to estimate each examinee's level of the attribute. Although an essential method in psychometrics and the study of individual differences, factor analysis has given rise to controversy for nearly 100 years, essentially because the attributes ("factors") invoked to account for the correlations—such as the *g* factor when the correlations are between types of IQ tests (Table 1)—are unobserved and thus tend to engender disputes over whether they really exist (e.g., Kovacs & Conway, 2019; Mulaik, 2005).

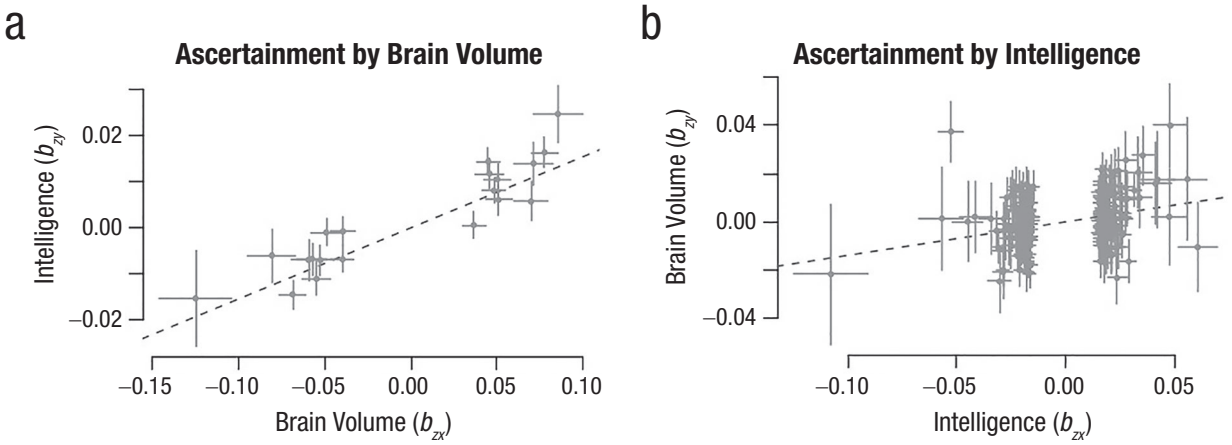

Fig. 2. Nature of the causal relationship between brain volume and IQ inferred from the asymmetry of results from GWASs. The 24 genetic sites reaching genome-wide significance in a GWAS of (a) brain volume all showed concordant effects on IQ (Jansen et al., 2020). Only 62% of the 243 genetic sites reaching genome-wide significance in a GWAS of (b) IQ showed concordant effects on brain volume (Savage et al., 2018). GWAS = genome-wide association study. Adapted from Jansen et al. (2020).

A powerful strategy to test the hypothesis that there might indeed be biological causes affecting all indicators of a common factor—causes that in this respect formally resemble the constructs posited by factor analysis—is rather similar to the one used to support brain size being a cause of IQ. One cannot directly examine whether a site in the genome ascertained for a significant effect on *g* goes on to affect a cognitive test (Fig. 2) because *g* cannot be directly observed. But factor analysis depends on there being multiple indicators of a putative latent trait, and one can examine whether a genetic site with an effect on one subtest has effects on other subtests consistent with their joint dependence on *g*, the latent trait that they are all supposed to measure (Grotzinger et al., 2019). This was more or less the strategy adopted by de la Fuente et al. (2021), and they did indeed find 27 sites in the genome with concordant effects on all of the cognitive subtests in their study—exactly as expected from each of these subtests being an endpoint of the chain depicted in Figure 1. Some of these sites were statistically significant only in the multivariate approach considering all subtests jointly.

This demonstration is perhaps not definitive because of the nonrepresentative character of biobank volunteers (Meredith, 1993). At the very least, however, it shows again the potential power of GWASs in behavioral research. Credibly identifying several independent causes of a given trait can illuminate many additional links in the chain of explanation (Fig. 1), including some of particular and long-standing concern to the practitioners of scientific psychology.

Recommended Reading

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Transparency

Action Editor: Robert L. Goldstone
Editor: Robert L. Goldstone
Declaration of Conflicting Interests
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.
Funding
The authors received no financial support for the research, authorship, and/or publication of this article.

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