Genetics and behaviour

Ady Cousins

Robert Plomin *argues that psychologists should prepare to maximise the benefits and minimise the risks that will emerge from DNA research.*

ASK any psychologist to complete the following phrase: 'nature–nurture _____'. The answer will no doubt be 'debate' or 'controversy'. But the controversy that swirled around behavioural genetics research in psychology during the 1970s has largely faded. During the 1980s and, especially, the 1990s, psychology became much more accepting of genetic influence, as can be seen in the increasing number of behavioural genetic articles in mainstream psychology journals and in research grants. One symbol of this change was the 1992 centennial conference of the American Psychological Association. In preparation for the conference, a committee selected two themes that best represented the past, present, and future of psychology. One of the two themes chosen was behavioural genetics (Plomin & McClearn, 1993).

In my view, this choice represents one of the most dramatic shifts in the modern history of psychology. Indeed, the wave of acceptance of genetic influence in psychology is growing into a tidal wave that threatens to engulf key messages coming from behavioural genetic research.

The first message is that genes play a surprisingly important role throughout psychology. But the second message is just as important: individual differences in complex psychological traits are due at least as much to environmental influences as they are to genetic influences. In fact, behavioural genetic research provides the strongest available evidence for the importance of environmental factors. But in some areas of psychology, especially psychopathology, the pendulum representing the accepted view may be swinging too far from environmental determinism to genetic determinism.

Perspectives

Behavioural genetics focuses on questions of why individuals within a species differ

in behaviour (e.g. why children differ in rates of language acquisition), whereas much research in psychology investigates species-typical behaviour (e.g. the average age at which children use two-word sentences). Descriptions and explanations of species-typical behaviour bear no necessary relationship to descriptions and explanations of individual differences within a species. For example, the fact that our species begins to use two-word sentences at the average age of 18 months is an evolutionary adaptation ultimately due to selection of genes, but this does not mean that genetics is responsible for the delayed use of two-word sentences by some children.

The fundamental accomplishment of genetic research in psychology to date has been to demonstrate the ubiquitous importance of genetics throughout psychology. As described later, this evidence consists of twin studies that compare the similarity of identical and non-identical twins; and adoption studies that consider the resemblance of adopted-away children to their biological parents. These methods and the theory that underlies them are called quantitative genetics, in contrast to molecular genetic research, which attempts to identify specific genes. Behavioural genetics includes both quantitative genetic research and, increasingly, molecular genetic research. Although this brief target article is limited to human behavioural genetics, more powerful quantitative genetic and molecular genetic methods are available for analysing animal behaviour (Plomin *et al*., 2001).

We can see this focus on species-typical behaviour in several key areas of psychology. For example, experimental psychology implicitly studies species-typical behaviour, comparing an experimental group with a control group representing typical behaviour, with individual differences considered as an error term in an analysis of variance. Similarly, much molecular genetic research consists of experimentally mutating a gene so that it is no longer expressed, and comparing these mutated animals with normal animals; whereas behavioural genetics has focused on naturally occurring genetic variation. Finally, evolutionary psychology also focuses on species-typical behaviour using comparisons between species as evidence for evolutionary adaptations. Although this is a type of genetic analysis of behaviour, the perspectives and empirical foundations for behavioural genetics and evolutionary psychology are so different that I think it causes confusion to conflate the two fields. I should emphasise that perspectives are not right or wrong, just more or less useful to address particular questions.

Autism as an example

As recently as the 1970s autism was thought to be caused by cold, rejecting parents. Certainly, parents whose children are autistic behave differently towards their children compared with parents of non-autistic children, but the direction-of-effects question looms large: Are the differences in parenting cause or effect? The accident of nature that results in identical (monozygotic) twins or non-identical (fraternal or dizygotic) twins provides one way to address this question. Identical twins are like clones, genetically identical to each other because they came from the same fertilised egg. Non-identical twins, on the other hand, developed from two eggs that happened to be fertilised at the same time. Like other siblings, they are only half as similar genetically as identical twins.

At the Institute of Psychiatry Michael Rutter and his colleague Susan Folstein were the first to use the twin method to investigate the causes of autism (Folstein & Rutter, 1977). They reasoned that if autism is caused by parental treatment, then non-identical twins ought to be as similar (concordant) for autism as are identical twins, because both types of twins are reared by the same parents in the same place and the same time and presumably get similar treatment. But if autism is influenced by genes, then non-identical twins ought to be less concordant. Folstein and Rutter located 10 autistic children in Britain who were non-identical twins. In none of these cases was the other twin also autistic. This result was not surprising, because autism is rare (an incidence of about one in a thousand) and it was already known that only about 3 per cent of the non-twin siblings of autistic children are autistic. The surprise came in the result for identical twins. Folstein and Rutter found 15 children diagnosed as autistic who were identical twins. Eight of these children were in four concordant pairs of twins in which both identical twins were diagnosed as autistic. The incidence of autism in children who have an autistic sibling, though low in absolute terms, represents a risk that is 30 times higher than that for children whose siblings are unaffected; the incidence in children who have an autistic identical twin represents a 500-fold increase in risk over the general population. The results of this small study have been replicated in other twin studies (as reviewed by Bailey *et al*., 1996).

The twin method Why are identical twins so much more concordant for autism than non-identical twins? The most parsimonious explanation is that identical twins are much more alike genetically. Another hypothesis puts the blame on prenatal factors. Identical twins often share the same chorion (the outermost membrane surrounding the foetus during prenatal development), which might make them more similar than non-identical twins (who never share the same chorion). So far the scanty evidence relevant to this issue is mixed (Sokol *et al*., 1995). Another possibility is that twins may not be representative of the non-twin population because of adverse intra-uterine environments caused by sharing a womb (Phillips, 1993). However, the statistical distributions for most psychological dimensions and disorders for twins and non-twins are generally similar (e.g. Christensen *et al*., 1995).

A subtle but important factor is that identical twins might have more similar experiences than do non-identical twins *after* they are born. The use of the twin method is based on the assumption that the environments of non-identical twins reared in the same family are approximately as similar as the environments of identical twins reared in the same family. This assumption has been tested in several ways and appears reasonable for most traits, although it has not been tested specifically in regard to autism (Bouchard & Propping, 1993). Although the possibility remains that identical twins may be treated more alike by their parents because they are more similar in appearance and behaviour, the twin method provides a rough but useful screen to unpack the 'bottom-line' effects of genes and environment (Martin *et al*., 1997).

The adoption method Although there are no adoption studies of autism, the adoption method is another quasi-experimental design that has a different set of assumptions and potential problems. Family members normally share both heredity (first-degree relatives correlate .50 genetically) and environment (they share the same family). Thus, familial correlations cannot tell us about the relative extent to which genetic and environmental factors contribute to observed resemblance

between family members. The adoption method separates the effects of nature and nurture by studying adopted-apart genetic relatives (to assess the role of genetics) and by studying genetically unrelated individuals brought together by adoption (to assess the role of family environment).

For example, for most psychological traits parents and offspring resemble each other. Because these are genetic-plus-environmental parents, parent–offspring resemblance could be due to nature or nurture. 'Genetic' parents and their adopted-away offspring do not share postnatal environment and thus their resemblance can be attributed to genetics and (in the case of birth mothers but not fathers) prenatal environment. 'Environmental' parents and their adopted children do not share heredity and thus their resemblance can be attributed to family environment. The adoption method also includes siblings or twins reared apart. Because the twin and adoption methods are so different, greater confidence is warranted when results from these two methods converge on the same conclusion – as they usually do.

One issue for adoption studies is that adoptees and their adoptive families might not be representative of the population as a whole, either because they have distinctive characteristics or because they span a narrower range. There is also the possibility that adopted children might be selectively placed with adoptive parents matched to the birth parents. These issues can be examined empirically. For example, in a longitudinal prospective adoption study of normal behavioural development that began in 1975, my colleagues and I found that adoptive families are reasonably representative of the population and that selective placement was negligible (Plomin *et al*., 1997).

Twin and adoption studies can be used not only to demonstrate the statistical significance of genetic influence but also to estimate its effect size, called heritability. Heritability is that proportion of the variance of a particular trait in a population that can be accounted for by genetic factors.

Beyond heritability For autism, the twin research without the additional weight of confirming adoption data was responsible for the shift from thinking about autism as a disorder caused entirely by environmental factors to its current status as one of the most heritable mental disorders. Genetic research has now moved on to other issues. One direction is to examine, using multivariate statistics, the genetic links between autism and other problems. The genetic vulnerability to autism has been shown to extend well beyond classic diagnostic symptoms: it includes milder social and communication difficulties that are found to be more common in the biological relatives of autistic people (Rutter *et al*., 1999).

Another direction for research concerns the effects of the environment. Because identical twins are not 100 per cent concordant for autism even though they are identical genetically, twin studies also provide strong support for the importance of environmental factors (though no support for theories that place the blame on the parents' behaviour). Environmental factors must contribute to differences in autism for two children – even identical twins – growing up in the same family. Such environmental influences are called 'non-shared' to distinguish them from shared environmental influences that make children growing up in the same family similar. What are the specific non-shared environmental factors that make identical twins growing up in the same family different and that widen the differences between other sibling pairs? Much remains to be learned about this important issue, for autism and for other psychological characteristics (Harris, 1998).

Another example of the use of genetic research to help us understand the environment has been called 'the nature of nurture' (Plomin, 1994). Twin and adoption studies have shown that genetic factors can have effects on the environment itself and that such effects can be found on aspects of the environment measured in psychological research. Such effects, known as 'genotype–environment correlations', could operate in various ways. Genetic factors could affect the reactions we evoke in others and the experiences that we select, construct and re-construct in memory. For example, autistic children evoke distancing responses in others and select non-social experiences that reinforce their genetic tendency towards social and communication abnormalities. Environmental influences need to be examined in genetically sensitive designs, and genetic influences need to be examined in environmentally sensitive designs that incorporate specific measures of the environment (Rutter *et al*., 1997).

A new direction for research is to attempt to identify some of the specific genes responsible for the genetic

HYPERACTIVITY

Several twin studies of attention deficit hyperactivity disorder (ADHD) have been carried out in recent years. These studies have consistently pointed to high heritability for hyperactive symptoms and for the ADHD diagnosis itself. Again, genetic links between the normal and abnormal have been found. Several molecular genetic studies have indicated a role for dopamine genes in the aetiology of hyperactivity (Thapar *et al*., 1999).

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READING DISABILITY

Reading disability shows moderate heritability – concordances for non-identical and identical twins are about 40 per cent and 70 per cent respectively. As with autism and many other disorders, the genetic liability for reading disability extends beyond the dichotomy imposed by a yes-or-no diagnostic procedure. On average, the non-identical twins of reading-disabled probands (index cases) are much better readers than the probands themselves, but they are significantly worse readers than the rest of the population. In contrast, the identical twins of reading-disabled probands read almost as poorly as the probands. As with autism, these results suggest a genetically influenced continuum between normal and abnormal. That is, genetic factors that affect diagnosed reading disability may be the same genetic factors that contribute to the quantitative dimension of reading ability (DeFries & Alarcón, 1996).

Results that demonstrate a continuum between the normal and abnormal have major implications for molecular genetic research, because they imply that many different genes contribute to the heritability of common disorders (Plomin *et al*., 1994). Unlike the relatively rare disorders in which a single gene is necessary and sufficient for the development of the disorder, common disorders like reading disability are complex traits influenced by environmental factors and by many genes of varying but relatively small effect size (called quantitative trait loci or QTLs). Multiple-gene systems require more powerful genetic designs that can detect genes of small effect. Reading disability is the first common behavioural disorder to which a QTL approach was applied. A linkage between reading disability and DNA markers on the short arm of chromosome 6 (Cardon *et al*., 1994) has been consistently replicated (e.g. Gayán *et al*., 1999). This QTL appears to be broad in its effect, involving both phonological (auditory) and orthographic (visual) aspects of reading disability.

contribution to various disorders, even though it seems likely that the target is many genes of small effect size rather than one or two genes of major effect. An international team initially led by Rutter and Monaco has identified a region on chromosome 7 that may be linked with vulnerability to autism (International Molecular Genetic Study of Autism Consortium, 1998). Finding specific genes that contribute to vulnerability to autism and other behavioural disorders and dimensions will provide the strongest evidence for genetic influence and will make it easier to explore the developmental interplay between nature and nurture (Plomin & Rutter, 1998).

Other examples

For milder psychological disorders too, a more balanced view that accepts the role of nature as well as nurture is beginning to prevail, and researchers are making progress in identifying specific genes. Two further examples are reading disability and hyperactivity (see boxes).

Other behavioural disorders and dimensions Moderate genetic influence has been found for most behavioural disorders that have been studied to date, including schizophrenia, mood disorders and anxiety (Plomin *et al*., 2001). Indeed, behavioural disorders tend to show greater genetic influence than common medical disorders such as breast cancer or heart disease (Plomin *et al*., 1994). The reason for the greater genetic influence for behavioural disorders may be that many different biological pathways can affect behaviour. Behaviour can be seen as the downstream outcome of many different biological systems; thus, genetic influences can operate on behaviour via all of the upstream systems.

Genetic influences are not limited to disorders, they also contribute to normal variation in personality (Loehlin, 1992) and in cognitive abilities (Plomin & DeFries, 1998), as well as to psychopathology. For self-report questionnaires, most personality traits show moderate genetic influence, with twin correlations of about 0.50 for identical twins and 0.20 for non-identical twins. In the case of self-report personality questionnaires, the far fewer adoption studies appear to suggest only about half as much genetic influence as twin studies, perhaps because twin studies capture different types of genetic variance than adoption studies (Plomin & Caspi, 1998). Genetic factors even appear to be involved in social and political attitudes and occupational interests (Plomin *et al*., 2001). Genes responsible for the heritability of personality are beginning to be identified (Hamer & Copeland, 1998).

One of the best documented cases in all of science for genetic influence is general cognitive ability (*g*), although it remains controversial in psychology (Plomin *et al*., 2001). Dozens of studies including more than 8000 parent–offspring pairs, 25,000 pairs of siblings, 10,000 twin pairs, and hundreds of adoptive families all converge on the conclusion that genetic factors contribute substantially to *g*. Estimates of the effect size, called heritability, vary from 40 to 80 per cent; but estimates based on the entire body of data are about 50 per cent, indicating that genes account for about half of the variance in *g*. Sorting the results by age indicates that heritability increases from about .20 in infancy to about .40 in childhood to .60 or higher later in life (McGue *et al*., 1993), even for individuals 80+ years old (McClearn *et al*., 1997).

This increase in the heritability of *g* throughout the lifespan is interesting because it is counterintuitive in relation to the accumulation of life's experiences. It may be that heritability increases because individuals seek and create environments correlated with their genetic propensities. That is, genetic propensities might be best considered as appetites rather than aptitudes, making some children hungrier for knowledge and more able to digest it. Molecular genetic studies are also beginning to identify genes associated with *g* (Fisher *et al*., 1999).

New avenues Genetic research to date has only scratched the surface of possible applications in psychology, even within the best-studied domains of psychopathology, personality, and cognitive disabilities and abilities. For psychopathology, genetic research has just begun to consider disorders other than schizophrenia and the major mood disorders. Developmental psychopathology has recently become an active area of genetic research (Rutter *et al*., 1999). Personality is so complex that it can keep researchers busy for decades, especially as they go beyond self-report questionnaires to use other measures such as observations and ratings by others (Riemann *et al*., 1997). A rich territory for future exploration is the links between psychopathology and personality (Nigg & Goldsmith, 1994). New directions for genetic research on cognitive abilities and disabilities includes the systematic analysis of psychological theories of cognition (Mackintosh, 1998), the use of information-processing measures of reaction times (e.g. Neubauer *et al*., 2000), and brain-imaging measures (Kosslyn & Plomin, 2001).

The vast majority of human genetic research in psychology has centred on psychopathology, personality, and cognitive disabilities and abilities because these areas have long been the focus of research on individual differences. Three new areas of psychology that are beginning to be explored genetically are psychology and aging, health psychology, and evolutionary psychology (Plomin *et al*., 2001). Some of the oldest areas of psychology – perception and learning, for example – have not emphasised individual differences and as a result have yet to be explored systematically from a genetic perspective. Entire disciplines within the social and behavioural sciences, such as economics, education, and sociology, are still essentially untouched by genetic research.

Genetic research in psychology is moving beyond heritability, as illustrated by the examples of autism, reading disability and hyperactivity. Asking whether and how much genetic factors affect psychological dimensions and disorders are important first steps in understanding the origins of individual differences, but these questions are only a beginning. The next step involves the question 'how' – that is, the study of the mechanisms by which genes have their effects. Avenues for genetic research in psychology include developmental change and continuity, links between the normal and abnormal, multivariate genetic analysis of heterogeneity and comorbidity, and the interplay between genes and environment. An especially exciting direction for research is identification of some of the specific genes responsible for the heritability of psychological disorders and dimensions.

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DNA

Although attention is now focused on finding specific genes associated with complex traits, the greatest impact on psychology will come after genes have been identified.

Using DNA Few psychologists are likely to join the hunt for genes because it is difficult and expensive, but once genes are found, it is relatively easy and inexpensive to use them (Plomin & Rutter, 1998). DNA can be obtained from cheek swabs, rather than blood, at a cost of less than £10 per individual. One cheek swab yields enough DNA to genotype thousands of genes, and several genes can be genotyped for less than £5 per individual. Microarrays the size of a postage stamp, called DNA chips, are becoming available that can genotype thousands of genes in a few minutes at costs that will eventually be very low per individual.

Although some psychology departments already have DNA laboratories, it is likely that most psychological research with DNA will be accomplished through collaborations with molecular geneticists or through commercial arrangements. It is critical for the future of psychology as a science that we be prepared to use DNA in our research and eventually in our clinics. What has happened in the area of dementia in the elderly will be played out in many areas of psychology. The only known risk factor for late-onset Alzheimer's disease (LOAD) is a gene, apolipoprotein E (ApoE), involved in cholesterol transport. A form of the gene called allele 4 quadruples the risk for LOAD but is neither necessary nor sufficient to produce the disorder; hence it is a QTL. The association between allele 4 and LOAD was first reported only in 1993, but it has already become *de rigueur* in research on dementia to genotype subjects for ApoE to ascertain whether the results differ for individuals with and without this genetic risk factor. Genotyping ApoE will become clinically routine if this genetic risk factor is found to predict differential response to interventions or treatments.

Scientific implications Among geneticists it is generally believed that we will be awash in genes associated with complex traits including behaviour in the next few years. This is especially likely as the Human Genome Project completes sequencing all 3.5 billion DNA bases (the four-letter alphabet of the genome that forms the steps in the spiral staircase of DNA) and identifies all genes and the several million DNA bases that differ among us. The future of genetic research lies in moving from finding genes (genomics) to finding out how genes work (functional genomics). Functional genomics is usually considered in terms of bottom-up molecular biology at the cellular level of analysis. However, a top-down psychological level of analysis may be even more valuable in understanding how genes work at the level of the intact organism, in understanding interactions and correlations between genes and environment, and in leading to new treatments and interventions. For example, top-down approaches for genes associated with learning and memory could trace the effects of genes through cognitive processes as outlined by theories of cognitive psychology (e.g. Mackintosh, 1998), in contrast to a bottom-up approach that begins with the cellular functioning of neuroreceptors. The phrase 'behavioural genomics' has been suggested to emphasise the importance of top-down levels of analysis in understanding how genes work (Plomin & Crabbe, in press). Bottom-up and top-down levels of analysis of gene–behaviour pathways will eventually meet in the brain. The grandest implication for science is that DNA will serve as an integrating force across diverse disciplines.

Clinical implications Geneticists need clinical psychology to define phenotypes, to design treatment, intervention and prevention programmes, and to evaluate these programmes. For clinical psychology, DNA may eventually lead to gene-based diagnoses and treatment programmes. The most exciting potential is secondary prevention. Because DNA analysis can be used to predict genetic risk for an

individual, it offers the hope for intervention before disorders create cascades of complications.

Interventions for complex psychological traits, and even for single-gene disorders, are likely to involve environmental rather than genetic engineering. For example, phenylketonuria (PKU), a metabolic disorder that can cause severe mental retardation, is caused by a single gene on chromosome 12. A particular form of the gene, found in 1 in 10,000 babies, damages the developing brain postnatally. This form of mental retardation has been largely prevented, not by high-tech solutions such as correcting the mutant DNA or by eugenic programmes or by drugs, but rather by a change in diet that prevents the mutant DNA from having its damaging effects. For this reason newborns have been screened for decades for PKU to identify those with the disorder so their diet can be changed. The example of PKU serves as an antidote to the mistaken notion that genetics implies therapeutic nihilism, even for a single-gene disorder. This point is even more important in relation to complex disorders that are influenced by many genes and by many environmental factors as well.

Social implications Psychologists should participate constructively in the discussion of scientific, clinical and social implications of the advances brought about by DNA research. The search for genes involved in behaviour has led to a number of ethical concerns: there is fear that the results will be used to justify social inequality, to select individuals for education or employment, or to enable parents to pick and choose among their foetuses. These concerns are largely based on misunderstandings about how genes affect complex traits such as the mistaken implication of therapeutic nihilism mentioned earlier, the assumption that genetic factors imply determinism, or that genetics justifies the status quo (Rutter & Plomin, 1997).

'DNA' For these reasons, it is crucial that psychologists be prepared to maximise the benefits and minimise the risks that will emerge from DNA research. Students in psychology must be taught about genetics to prepare them for this future. Otherwise this opportunity for psychology will slip away by default to geneticists, and genetics is much too important a topic to be left to geneticists! Clinical psychologists use the acronym 'DNA' to note that their clients 'did not attend.' It is critical to the future of psychology as a science that, for all psychologists, DNA denotes deoxyribonucleic acid rather than 'did not attend'.

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