Genetics and intelligence

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Talent Development III, pp. 19-39
Gifted Psychology Press
1995

This article by Robert Plomin discusses the importance of genetics and intelligence in the development of talent. Plomin states that, "Most of what is currently known about the genetics of intelligence comes from twin and adoption studies, which have documented significant and substantial genetic influence." Genetic analyses have shown that across the life span genetic effects increase and "genetic factors are primarily responsible for stability during development but also affect age-to-age changes." Additional and focused research is needed to reveal the "magnitude of genetic effects." This article presents past and present research findings detailing the influence of genetic factors on talent development.

Although the phrase talent development means different things to different people, talent must certainly include intelligence. The word intelligence in the title of this paper is almost as contentious in the field of gifted education as is the other noun in the title, genetics. Putting the words together to discuss genetics and intelligence may not be politically correct in our zeitgeist, but I believe that the field of gifted education has much to gain by recognizing the importance of intelligence and the importance of genetic influences in the development of this key aspect of talent.

The paper begins with a brief description of the history of genetic research on intelligence. This research makes a stronger case for the importance of genetic factors for intelligence than for any other aspect of behavior. The chapter does not review this research or the methods of behavioral genetics used in the research because the growing acceptance of the genetic contribution to intelligence makes it less important to do so and because reviews of behavioral genetic methods and research on intelligence are available elsewhere in introductory form (e.g., Plomin, 1990) or in textbooks (e.g., Plomin, DeFries, & McClearn, 1990). It is becoming more important to emphasize that this conclusion about the importance of genetic factors in intelligence is nearer to the beginning than it is to the end of the story about genetics and intelligence. Genetic research can go beyond the rudimentary questions of whether and how much genetic factors influence intelligence. For example, genetic research is exploring the developmental course of nature and nurture, the multivariate nature of genetic effects on cognitive abilities, and the genetics of high intelligence. One of the most exciting advances is the emerging possibility that molecular genetic tools can identify some of the specific genes that contribute to intelligence. An ongoing project of this type that focuses on high intelligence will be described.

Genetics

At the outset, I should clarify what I mean by genetics and what I mean by intelligence. The word genetics is used very narrowly (Plomin et al., 1990). It is critically important to recognize that behavioral genetic research is limited to the investigation of the genetic and environmental origins of individual differences within a species, not species-typical development. Genetic research focuses on DNA differences among individuals, not the vast majority of DNA that is the same for all of us, much of which is also the same for primates and other mammals. Genetics is used to refer to inheritance in the sense of DNA differences transmitted from generation to generation. Many DNA events are not inherited-for example, mutations in DNA in cells other than sex cells often cause cancer but are not inherited-and thus are not considered genetic in this narrow sense. In contrast to this very narrow definition of genetics, the word environment is used very broadly to refer to any nongenetic process that makes people different such as perinatal events, illness, and diet, as well as psychosocial processes.

Genetic research describes what is, the genetic and environmental origins of differences among individuals in a particular population at a particular time. It does not predict what could be, nor does it prescribe what should be. Evidence for genetic influence (what is) does not imply that differences among individuals are immutable or irremediable-novel environmental factors could make a difference (what could be). Conversely, expert training that produces impressive skills (what could be) does not contradict evidence for genetic influence in the population with its normal variation in experiences (what is). No research tells us what should be because this is a matter of goals and values.

Intelligence

What I mean by the word intelligence is general cognitive ability. Nearly all reliable measures of cognitive abilities (such as tests of verbal, spatial, and memory) correlate at least moderately. General cognitive ability is what such tests have in common. More complex cognitive processes such as abstract reasoning are better indices of general cognitive ability than are less complex processes such as simple sensory discriminations. Because IQ tests typically assess a broad range of complex cognitive processes, their total scores provide useful indices of general cognitive ability. Because the wordintelligence has so many different meanings, it may be preferable to use the phrase general cognitive ability or its symbol g(Jensen, 1987; Jensen & Weng, in press).

Misinformation about intelligence abounds in the media. This situation prompted 52 scientists to outline 25 conclusions regarded as

mainstream among researchers on intelligence concerning the meaning and measurement of intelligence, its practical importance, and its origins (Arvey et al., 1994). Their first two conclusions concerning the meaning and measurement of intelligence are as follows:

1. Intelligence is a very general mental capability that, among other things, involves the ability to reason, plan, solve problems, think abstractly, comprehend complex ideas, learn quickly and learn from experience. It is not merely book learning, a narrow academic skill, or test-taking smarts. Rather, it reflects a broader and deeper comprehension of our surroundings--"catching on," "making sense" of things, or "figuring out" what to do.

2. Intelligence, so defined, can be measured, and intelligence tests measure it well. They are among the most accurate (in technical terms, reliable and valid) of all psychological tests and assessments. They do not measure creativity, character, personality, or other important differences among individuals, nor are they intended to. (Arvey et al., 1994, p. A-18)

Nature and Nurture

Shakespeare first brought the words nature and nurture together in The Tempest, when Prospero describes Caliban as a "devil on whose nature nurture can never stick." The idea of nature in conflict with nurture was the impetus for the alliterative phrase nature-nurture, used by Darwin's cousin, Francis Galton (1865), more than a century ago. Galton argued that "there is no escape from the conclusion that nature prevails enormously over nurture" (1883, p. 241). Joining these two words created a fission that exploded into the longest-lived controversy in the behavioral sciences. The hyphen in nature-nurture connoted the implicit conjunction "versus." The appropriate conjunction between nature and nurture is "and."

During the past century, the pendulum has swung back and forth several times between nature and nurture. A pessimist's view of these swings is that they are becoming faster, wider, and more divisive, a view that might seem supported by the turmoil raised recently by The Bell Curve (Herrnstein & Murray, 1994). However, I have a more optimistic view that the pendulum is losing its inertia and is coming to rest in between nature and nurture.

Although the pendulum has been swinging for centuries, in 1890 William James' Principles of Psychology, was published. James' book provided the agenda for much psychological research but scarcely mentioned individual differences or heredity. Galton's enthusiasm for the importance of nature over nurture, riding the crest of the wave created by his cousin Darwin and the rediscovery of Mendel's laws of inheritance, kindled interest in heredity during the first few decades of this century. The first twin and adoption studies of cognitive ability were reported in 1924, for example. This swing toward nature also included eugenics, a word coined by Galton. The pendulum was shoved back in the other direction by the horrors of the Nazis. In psychology, this swing back to nurture also coincided with the emergence of behaviorism. Behaviorism refers to a strict focus on observable behavioral responses and this led to an interest in observable environmental stimuli that change behavior. For this reason, behaviorism came to imply environmentalism. J. B. Watson's frequently quoted challenge epitomized this swing of the pendulum to nurture:

> Give me a dozen healthy infants and my own specified world to bring them up in, and I'll guarantee to take anyone at random and train him to become any type of specialist I might select-doctor, lawyer, artist, merchant-chief, and, yes, even beggar and thief, regardless of his talents, penchants, tendencies, abilities, vocations, and race of his ancestors. (Watson, 1924, p. 104)

During the 1940s and 1950s, behaviorism and learning theory dominated American psychology. For mental illness, this was fertile ground for Freud's brand of environmentalism that blamed psychopathology on parental treatment from the first few years of life (Torrey, 1992). For cognitive abilities, the previous decades of twin and adoption studies pointing to strong genetic influence disappeared from textbooks.

By 1960, there were signs that the pendulum was swinging back towards nature. Research on animal behavior such as studies comparing inbred strains of mice and artificial selection experiments provided powerful demonstrations of the importance of genetics on behavior. The trickle of human behavioral genetic research that had begun in the 1920s with twin and adoption studies continued but remained outside the mainstream of the behavioral sciences. This work, especially animal research, led to the first behavioral genetics textbook in 1960 (Fuller & Thompson, 1960).

In 1963, an influential article in Science reviewed family, twin, and adoption data for IQ scores and concluded that genetic influence is important (Erlenmeyer-Kimling & Jarvik, 1963). The twin studies of Steven Vandenberg on cognitive abilities in the 1960s confirmed the results of the earlier twin and adoption studies in pointing to substantial genetic influence (Vandenberg, 1968). This swing of the pendulum towards nature ended, especially in psychology, in 1969 when Arthur Jensen published a paper that reviewed the evidence for genetic influence on IQ scores and suggested that average IQ differences between ethnic groups may in part be due to genetic factors. Jensen's article, combined with a book by Richard Herrnstein (1973) suggesting that IQ differences between classes might also be genetic in origin, provoked a furious response unparalleled in the behavioral sciences. The reaction to Jensen and Herrnstein in the early 1970s threatened the existence of the fledgling field of human behavioral genetics, even though very few behavioral geneticists studied ethnic or class differences. The furor subsided only gradually during the 1970s as new twin and adoption research on cognitive abilities was launched that addressed some of the problems uncovered in the intense scrutiny of earlier research.

In the 1980s, the pendulum again began to swing back towards nature, in part due to this new research (Plomin & DeFries, 1980) and in part due to the excitement caused by advances in molecular genetics. For example, a 1987 survey of more than a thousand social and behavioral scientists and educators indicated that most had accepted a significant role for heredity in IQ scores (Snyderman & Rothman, 1987). The response to The Bell Curve was largely in relation to the same two issues raised by Jensen and Herrnstein 25 years ago--the possibility of genetic influence on ethnic and class IQ differences-even though these are minor issues in the book. However, in contrast to the long-lasting effects of the original controversy in the early 1970s, the reaction to The Bell Curve, as intense as it was in the media, seems to have dissipated quickly, although it may be too soon to tell.

The reason for hoping that the pendulum is coming to rest at a point in between nature and nurture is not just that we want everyone to be happy. It is what genetic research tells us. In the 1960s and 1970s the message was that genetics is important. The convergence of evidence across different methods such as twin and adoption designs, across decades, and across countries is impressive. As the message that genetics is important is received, it becomes increasingly important to emphasize that these same data also provide the best evidence for the importance of the environment. Model-fitting estimates derived from all genetic data on IQ suggest that genetic factors account for about half of the variance in IQ scores (Chipuer, Rovine, & Plomin, 1990; Loehlin, 1989), which means that about half of the variance is not accounted for by genetic factors. Correcting for unreliability of measurement, the relative effect size for genetics is greater. Also, as mentioned later, genetics accounts for more of the variance later in life. Although genetic factors might account for somewhat more or less than fifty percent of the variance, the point is that both nature and nurture play a role in the development of individual differences in intelligence.
I hope that the next generation of behavioral scientists will wonder what all the nature-nurture fuss was about-that it will seem obvious to them that individual differences in intelligence are due to genetic differences as well as environmental differences.

Beyond Nature Versus Nurture

More research is needed to pin down the magnitude of genetic influence to intelligence and specific cognitive abilities across the life span. Much remains to be learned about whether and how much genetic factors play a role in new methods for assessing cognitive abilities, such as information-processing and neurological approaches. However, we have reached the point at which it is no longer necessary to conduct twin and adoption studies merely to demonstrate genetic influence on cognitive abilities. What is needed now is research that goes beyond the rudimentary questions of whether and how much genetic factors contribute to cognitive abilities. Three examples are developmental genetic analysis, multivariate genetic analysis, and analysis of the genetics of high ability.
Developmental genetic analysis. Developmental changes in the strength and nature of genetic effects need to be charted (Plomin, 1986). The strength of genetic effects can change as the relative magnitude of genetic or environmental factors changes during development. This question motivated the first twin study by Francis Galton (1876) more than a century ago. It is reasonable to assume that environmental factors become increasingly important as experiences accumulate during life. To the contrary, genetic research on intelligence shows that genetic factors become increasingly important from infancy to childhood to adolescence to adulthood (McGue, Bouchard, Iacono, & Lykken, 1993; Plomin & Thompson, 1987). This could occur, for example, if individuals increasingly seek out environments that foster their genetic propensities, that is, active genotype-environment correlation.
A second type of developmental genetic analysis investigates genetic contributions to change across time using longitudinal data. That is, to what extent do genetic effects at one age differ from genetic effects at another age? For example, to what extent do genetic effects on IQ differ from infancy to early childhood to middle childhood? New techniques are available that can assess genetic contributions to change from age to age as well as to continuity. Longitudinal research using these techniques shows that although genetic factors primarily account for developmental continuity, some evidence can be found for genetic contributions to change, especially during the transition from early to middle childhood (Fulker, Cherny, & Cardon, 1993) and perhaps from middle childhood to late adolescence (Loehlin, Horn, & Willerman, 1989). What this means is that genetic factors that contribute to individual differences in intelligence in middle childhood are to some extent different from genetic factors that affect intelligence in early childhood. Although this could mean that new genes are turned on in middle childhood, it is more likely that the same genes have different effects in the brains of 8-year-olds as compared to 4-year-olds (Plomin, 1986).

Multivariate genetic analysis. Multivariate genetic analysis assesses genetic contributions to covariance among traits rather than to the variance of each trait considered separately. That is, to what extent do genetic effects on one cognitive ability also affect another cognitive ability? Three examples of multivariate genetic analysis include the overlap among specific cognitive abilities, correlations between intelligence tests and tests of school achievement, and associations between intelligence and measures of family environment. Concerning the overlap among specific cognitive abilities, multivariate genetic analyses suggest that genetic effects on all cognitive abilities overlap to a surprising degree, although some genetic effects are unique to each ability. That is, genes associated with one cognitive ability such as spatial ability are also strongly associated with other cognitive abilities such as verbal ability or memory. In other words, if a gene could be found that is associated with spatial ability, the same gene would be expected to be associated with verbal ability. Another facet of this finding of the generality of genetic effects is that the more strongly a particular test loads on a factor of general cognitive ability (that is, an unrotated principal component), the higher its heritability even when differential reliability of these tests is controlled.

A second example of multivariate genetic analysis concerns the relationship between intelligence and scholastic achievement. Achievement at school and intelligence are widely assumed to be different by definition-achievement is what a student achieves by dint of effort, whereas intelligence is thought to involve inherent talent, forgetting that performance on achievement and intelligence tests is substantially correlated. This correlation raises the possibility of genetic links between the two domains. The possibility of genetic links looms larger with the finding that tests of achievement in high school show nearly as much genetic influence as intelligence tests (Plomin et al., 1990). Multivariate genetic analyses provided evidence of strong genetic links between the two domains. Indeed, this research suggests that genetic effects on scholastic achievement overlap completely with genetic effects on general cognitive ability (Thompson, Detterman, & Plomin, 1991), a finding replicated in three other studies (Wadsworth, 1994). This finding implies that the correlation between intelligence and scholastic achievement is due almost entirely to genetic factors common to the two domains. In other words, if a gene were found that is associated with intelligence, the same gene would also be expected to be associated with scholastic achievement. The converse of this finding is equally interesting: Discrepancies between intelligence and achievement, often used to describe underachievers, appear to be largely environmental in origin.

The third example of multivariate genetic analysis raises a topic that has been called the nature of nurture (Plomin & Bergeman, 1991).

Many widely used measures of the environment, especially the home environment of children, show genetic influence in dozens of twin and adoption studies during the past five years (Plomin, 1994). Although this might seem paradoxical at first, what it means at its simplest level is that ostensible measures of the environment inadvertently assess genetically influenced characteristics of individuals. For example, one of the most widely used observation/interview measures of the home environment as it relates to cognitive development is a measure called the Home Observation for Measurement of the Environment (HOME; Caldwell & Bradley, 1978). In the Colorado Adoption Project, the HOME was assessed for nonadoptive and adoptive siblings when each child was 12 months old and again at 24 months. Nonadoptive and adoptive sibling correlations for the HOME total score were .50 and .36, respectively, at 12 months, and .50 and .32 at 24 months, suggesting that parental behavior assessed by the HOME substantially reflects genetic differences among children (Braungart, Plomin, Fulker, & DeFries, 1992).

If genetic factors contribute to environmental measures such as the HOME, genetic factors can also be involved in the correlation between the HOME and children's cognitive development. It is at this point that multivariate genetic analysis comes into play, this time in analyzing the genetic contribution to the covariation between the HOME and children's IQ scores. Such analyses yield a surprising result: About half of the HOME's prediction of children's IQ appears to be mediated by genetic factors, even longitudinally when the HOME in infancy is used to predict IQ in early and middle childhood (Plomin, 1994).

Genetic analysis of high intelligence. Finding substantial genetic influence on individual differences within the normal range of intelligence does not necessarily mean that the high end of the distribution of intelligence shows similar levels of genetic influence. For example, at the low end of the distribution, severe retardation shows little familiarity, in contrast to the rest of the distribution (Plomin, 1991). Surprisingly little is known about the origins of high intelligence (Feldman, 1986; Howe, 1990; Simonton, 1989; Storfer, 1990). For example, there has never been a twin or adoption study of high intelligence.

In 1865, a year before Mendel's seminal paper on heredity, Francis Galton published a two-article series on hereditary genius that he expanded into the first book in the field of human behavioral genetics, Hereditary genius: an inquiry into its laws and consequences (Galton, 1869). Using mere reputation as an index, Galton suggested that ability, brains as well as brawn, ran in families. He greatly overinterpreted the results to support his belief that genius is hereditary, although in 1874 he toned down his conclusions (Galton, 1874). Nonetheless, Galton believed that "ability will out" regardless of environment.

The issue is the etiology of high intelligence-why, as a group, high-intelligence individuals on average score so much more highly than the rest of the population on tests of intelligence. In a follow-up study of Terman's gifted individuals, the average IQ of their offspring was 133, a score in the top few percentile of the distribution of IQ scores (Oden, 1968). This suggests that familial factors contribute importantly to high intelligence. Family studies, however, cannot disentangle genetic and environmental influences.

In recent years, genetic researchers have re-analyzed intelligence data from twin and adoption studies of unselected populations for subsamples of high-intelligence individuals (Bailey & Revelle, 1991; Cherny, Cardon, Fulker, & DeFries, 1992; Detterman, Thompson, & Plomin, 1990; Thompson, Detterman, & Plomin, 1993). The results of these analyses conflict concerning the magnitude of genetic influence for high-intelligence individuals. However, these studies have addressed a different issue-why high-intelligence individuals differ from each other-rather than the etiology of high intelligence. That is, these studies asked why one individual has an IQ of 135 and another has an IQ of 145, rather than asking why, as a group, these high-intelligence individuals have such high IQ scores as compared to the rest of the population.

A method has been developed to assess genetic and environmental contributions to the average difference between a selected group and the unselected population (DeFries & Fulker, 1985, 1988), in contrast to the usual genetic analysis that focuses on individual differences. This method has been applied in two studies and both found evidence for substantial genetic influence for high intelligence, one in middle childhood (Plomin & Thompson, 1993) and one in older adults (Saudino, Plomin, Pedersen, & McClearn, 1994). A rough illustration of this finding can be seen in the concordance of identical and fraternal twins for high intelligence for twin pairs selected because at least one member of the pair has an IQ score above 120. In the study in middle childhood, this procedure resulted in concordances of 62% for identical twins and 25% for fraternal twins.

In both studies, the magnitude of genetic influence for high intelligence was comparable to that of the unselected population, suggesting that high intelligence may merely be the high end of the normal distribution of genetic and environmental influences on individual differences in intelligence. However, because both studies involved unselected samples of twins from which high-IQ individuals were selected for analysis, samples were small and subjects had relatively high IQ scores, about one standard deviation above the mean. What is needed is a genetic study that focuses on very high intelligence. Clearly the available studies have nothing to say about the etiology of genius. It has been suggested, for example, that genius might differ genetically from the normal distribution: Genius might be the result of specific constellations of many genes, which is called epistasis, rather than the additive effects of genes that contribute to individual differences in the normal range (Lykken, McGue, Tellegen, & Bouchard, 1992).

Molecular Genetics

The most exciting way in which research is moving beyond the nature versus nurture question is to begin to harness the power of molecular genetics to identify specific genes responsible for the substantial influence of genetics on intelligence. Although molecular genetic techniques are now routinely used to find the chromosomal location of single-gene disorders, the task of finding specific genes in complex systems such as intelligence, which is influenced by multiple genes as well as multiple environmental factors is much more difficult (Plomin, Owen, & McGuffin, 1994). The challenge is to use the thousands of new DNA markers to identify not the gene for intelligence but the many genes, each of which makes a small contribution to variance in the population. In this section, an ongoing study is described that attempts to identify specific genes that affect high intelligence.

At the outset, it is important to consider the implications of molecular genetic research that seeks to identify specific genes. The major

implication of identifying genes that affect intelligence will be for basic research. Even a small handhold on the genetic contribution to individual differences will help in the climb towards understanding how genes act and how they interact with the environment. It should be noted that such genes are not likely to be useful in predicting IQ in the general population because of the sheer numbers of genes involved. Indeed, it is doubtful whether genes in sufficient number and strength will ever be identified that can reach levels of prediction that rival those that can be made at present on the basis of parental IQ. The regression of offspring on the average IQ of mother and father is about .60. In other words, parents' IQ can predict more than one third of the total variance in offspring IQ scores, which is more than two-thirds of the genetic variance if heritability is .50. If genes responsible for genetic variance in IQ scores typically account for less than one percent of IQ variance in the population, dozens of such genes might be required to make a reasonable prediction of children's IQ. Moreover, if many genes individually account for far less than 1% of the variance, as I suspect, most of these genes will never be identified and thus genes that can be identified will fall far short of predicting all of the genetic variance of IQ.

As is the case with most important advances in science, identifying quantitative trait loci (QTL) for intelligence could raise new ethical problems (Wright, 1990). For single-gene disorders, identification of the responsible genes has already led to concerns such as employment and insurance discrimination (e.g., Bishop & Waldholz, 1990; Nelkin & Tancredi, 1989). The heat that will be generated when genes for intelligence are found can be anticipated from the vehement responses to a 1988 editorial in Science that argued that "we must step boldly and confidently across the threshold" (Koshland, 1989, p. 189). A 1993 commentary in Nature hyperbolized that "the isolation of the first gene involved in determining 'intelligence' (whatever that is) will be a turning point in human history." (Miller-Hill, 1993, p. 492)

However, this same commentary wisely argued:

> Anticipating such conflicts, many may conclude that we do not need or want this genetic knowledge. I disagree. The knowledge will simply unveil reality, emphasizing the injustice of the world....Laws are necessary to protect the genetically disadvantaged. Social justice has to recompense genetic injustice. (Miller-Hill, 1993, p. 492)

The potential uses for understanding the etiology of intelligence seems likely to outweigh its potential abuses. Moreover, forewarned of problems and solutions that have arisen in the case of single-gene disorders, we should be forearmed as well to prevent abuses.

Single-gene effects. More than 100 rare single-gene disorders include mental retardation among their symptoms (Walhsten, 1990). The classic example of a single-gene cause of severe mental retardation is phenylketonuria (PKU). The recessive gene responsible for PKU is necessary and sufficient to cause the disorder given a normal diet that contains phenylalanine. (A diet low in phenylalanine circumvents damage to the developing brain caused by the buildup of a metabolite of phenylalanine that cannot be broken down by PKU individuals.) PKU is quite rare, affecting about 1 in 10,000 individuals. In 1991, fragile X mental retardation was shown to involve a single gene on the X chromosome (Verkerk et al., 1991). It is the most common cause of mental retardation after Down's syndrome, a chromosomal abnormality that usually occurs spontaneously rather than being inherited. Still, fragile X mental retardation is quite rare, with an incidence of about 1 in 1250 males and 1 in 2500 females. Another marker on the X chromosome has been linked to an even rarer form of fragile X mental retardation (Knight et al., 1993). Like PKU, these genes for fragile X mental retardation involve single genes that are necessary and sufficient to produce the disorder.

A much more common cognitive disorder is the progressive memory loss and confusion of Alzheimer's disease (AD) which generally appears late in life and affects as many as 15% of individuals over 80 years of age. AD also includes a rare type, accounting for fewer than 1% of AD cases, that appears much earlier in the life span and is strongly familial. This early onset, familial form of AD has been shown to be linked to a gene on chromosome 14 (Schellenberg et al., 1992).

PKU, fragile X mental retardation, and the early-onset familial form of AD are examples of single-gene disorders that are necessary and sufficient to cause the disorder. We all first learn about genetics from Mendel's studies of dichotomous traits in the pea plant, in which he showed such single-gene effects. Several thousand single-gene disorders, most very rare, have been documented (McKusick, 1990). For single-gene disorders, traditional linkage analysis is guaranteed to find the chromosomal location of the gene, even when nothing is known about the gene product. Linkage traces co-transmission of a marker and a disorder within a family pedigree. The exemplar is Huntington's disease, which, in 1983, was the first disorder linked to a chromosome using DNA markers even though nothing was known about the product of the gene responsible for Huntington's disease.

Quantitative trait loci (QTL). The problem is that behavioral dimensions are different. Genes that affect behavioral traits are transmitted hereditarily according to Mendel's laws in the same way as genes that affect any other phenotype, but behavior is special in three ways. First, unlike Mendel's smooth and wrinkled seeds, most behavioral dimensions and disorders are not distributed in simple either/or dichotomies, although we often pretend that there is a line that sharply separates the normal from the abnormal. Second, behavioral traits are substantially influenced by non-genetic factors: Heritabilities rarely exceed 50%. Third, behavioral dimensions and disorders are likely to be influenced by many genes, each with small effects. The challenge is to use DNA markers to find genes in complex systems such as those which involve multiple genes as well as multiple nongenetic factors. Such genes of varying effect size that contribute to quantitative traits are called quantitative trait loci (QTL). Because QTL contribute interchangeably and additively as probabilistic propensities, any particular QTL within a multiple-gene system is neither necessary nor sufficient.

The traditional approach of linkage can only identify a major gene largely responsible for a disorder. The breathtaking pace of advances in molecular genetics makes it reasonable to predict that by the turn of the century we might be investigating multiple-gene influences for behavioral dimensions and disorders using techniques completely different from those currently available. In the meantime, one strategy that has been used successfully is called allelic association. Linkage refers to a DNA marker and a disorder linked to the same chromosome that is detected by co-segregation of the marker and the disorder in family pedigrees. In contrast, allelic association occurs

when a DNA marker is itself the QTL, that is, the DNA marker may directly affect the trait. The main advantage of allelic association is that it is much more powerful than linkage in finding QTL of small effect size (Plomin et al., 1994).

QTL and cognitive ability and disability. The best QTL example for cognitive disability is the allelic association between late-onset Alzheimer's disease (AD) and a particular allele of the gene for apolipoprotein E (Apo-E; Corder et al., 1993). Apo-E is interesting because it is involved in cholesterol transport from cells and is synthesized in the brain and other organs. The marker is functional in that it indexes the three major Apo-E isoforms which are the products of three alleles of a single gene on chromosome 19. The frequency of one of these alleles (called allele 4) is 40% in AD individuals as compared to 15% in control populations. Individuals who have this allele are six times more likely to develop AD. However, some 40% of AD individuals do not have this allele and many individuals with this allele do not succumb to AD. In this sense, the AD association with Apo-E is a QTL effect because the allele is neither necessary nor sufficient to develop the disorder. Nonetheless, this is a big QTL effect in that it accounts for approximately 15% of the population variance in liability to develop AD (Owen, Liddle, & McGuffin, 1994). Functional DNA markers such as the Apo-E marker greatly enhance the power of allelic association to detect QTL (Sobell, Heston, & Sommer, 1992). Many new markers are of this type. However, most extant DNA markers are not functional because they involve DNA differences among individuals that are not transcribed from genomic DNA into messenger RNA that leaves the nucleus of the cell and is translated into polypeptide sequences. However, allelic association can also occur when a nonfunctional DNA marker is so close to a functional QTL that the DNA marker is associated with the QTL and is thus indirectly associated with the trait. Until more functional DNA markers become available, one strategy to find allelic association is to use DNA markers in or near candidate genes. In the case of high intelligence, such candidate genes could include most of the thousands of genes involved in the nervous system.

The IQ QTL Project

Intelligence is a reasonable candidate for molecular genetic research because it is one of the most heritable dimensions of behavior. A few studies have investigated allelic associations between classical genetic markers such as blood groups and IQ without success (e.g., Ashton, 1986; Gibson et al., 1973; Mascie-Taylor et al., 1985). Some evidence suggests that carriers for recessive disorders such as PKU show slightly lowered IQ scores (Bessman et al., 1978; Propping, 1987).

The new DNA markers are being used in an allelic association study of intelligence called the IQ QTL project (Plomin et al., 1994, 1995). An allelic association strategy is employed in this ongoing research that uses DNA markers that are in or near genes likely to be relevant to neurological functioning, such as genes for neuroreceptors. Allelic frequencies for these DNA markers are compared for groups differing in IQ. Rather than genotyping individuals throughout the IQ distribution, the project focuses on the extremes of the distribution. The project features built-in replication in order to attenuate the possibility of false positive results.

Phase I. The first phase of the project focused on 100 DNA markers genotyped for Caucasian children with high versus low IQ scores in an original sample and significant markers were genotyped in an independent replication sample. In the original sample, the average IQ scores were 82 and 130 for the low and high IQ groups, respectively. The replication sample consisted of children of even lower and higher IQ, with average IQ scores of 59 and 142 for the low (N = 17) and high (N = 27) groups. Selecting extreme subjects and samples of this size only provide statistical power to detect relatively large allelic frequency differences, differences of about .20 or greater. QTL associations of this magnitude account for 2% or more of the population variance of IQ. From a linkage perspective, this is a very small effect size, but from a QTL perspective, it is a large effect. For this first phase of the IQ QTL project, allelic association results in the original sample were reported for 100 DNA markers that included the marker for ApoE as well as markers for neurologically relevant genes such as catechol methyl transferase, monoamine oxidase A, and a serotonin receptor (Plomin et al., 1994, 1995). Of the 100 markers analyzed in the first phase of the IQ QTL project with its low resolving power, only one significant association was found that cleanly replicated in the independent replication sample (Plomin et al., 1995). Before discussing this significant association, results for other markers are mentioned.

Apolipoprotein E. The results for ApoE are interesting and illustrative. In addition to its association with AD, it has been reported that the Apo-E allele 4 is also significantly associated with cognitive decline in a Dutch population of elderly men (Feskens et al., 1994). However, the allelic frequency difference between cases and controls was small, about 17% versus 12% in the low scoring subjects as compared to control subjects. This difference is in the same direction as the much larger difference of about 45% versus 15% seen in AD versus control subjects. The small difference was significant because the Dutch sample was large (181 cases and 278 controls). As shown in Table 1, the frequencies for this allele in the IQ QTL project were 21% in the low IQ group and 17% in the high IQ group, a difference in the same direction and magnitude as the Dutch study, although not nearly significant because of the much smaller sample size of the IQ QTL project. A difference of this magnitude would account for far less than 1% of the population variance of IQ, which implies that it would be difficult to replicate without very large samples.

TABLE 1. IQ QTL Associations for Apo-E. (Derived from Plomin et al., 1995.)*

The other major Apo-E allele (called allele 3) yielded interesting results, although this allele has not been implicated in AD or cognitive decline in the elderly. In the IQ QTL project, this allele was less frequent in the low IQ group than in the high IQ group in both the original and replication samples. Although these allelic frequency differences were not significant in the original or replication samples, when the original and replication samples were combined, the difference was significant ($p < .05$).

CTG-B33 and RIA. Two other markers yielded significant associations in the combined sample, although they did not reach statistical significance in the replication sample and thus do not meet the replication criterion for establishing QTL. Nonetheless, one marker, CTG-B33 was marginally significant ($p = .05$) in both the original and replication samples. The marker is interesting because it is expressed in the brain and represents a new generation of markers found by screening a human DNA brain library (Li et al., 1993). The other marker, HLA-A, involves a newly discovered gene that appears to be unique to the human species and is in a gene-rich region of chromosome 6 (Venditti et al., 1994). It is also the region where a gene linked to reading disability has recently been reported (Cardon et al., 1994).

TABLE 2. IQ QTL Associations for CTG-B33 and HLA. (Derived from Plomin et al., 1994.)*

EST00083. EST00083 is a cloned fragment of a gene that is expressed in the brain but whose function is unknown. Thousands of such gene fragments have been identified and sequenced during the past five years (Adams et al., 1991). We discovered a two-allele marker for this particular brain-expressed gene fragment and genotyped the low and high IQ samples for this marker. Table 3 describes the allelic frequencies for EST00083 in the original, replication, and combined samples.
TABLE 3. IQ QTL Associations for EST00083. (Derived from Plomin et al., 1995.)*

The results are similar and significant for the original and replication samples: The less common allele (called allele 2) is rarely found in the high IQ sample, although it occurs in about 25% of the low-IQ sample. Despite these significant results, EST00083 appeared not even to warrant further consideration because when we looked at the genotypic results (alleles taken two at a time as they exist in individuals) rather allelic results, a bizarre finding emerged. Of 107 individuals genotyped for this marker, not a single heterozygote was found. That is, individuals either had two A1 alleles or two A2 alleles, but never one A1 allele and one A2 allele, even though laws of heredity would predict that 27 such individuals should be observed. We discovered that this marker, which was thought to be derived from chromosomal DNA in the nucleus of the cell, was in fact mitochondrial DNA from the cell's cytoplasm (Skuder et al., 1995). We pinned down the marker to DNA nucleotide base pair number 15,925 of the 16,500 mitochondrial base pairs, which is in a gene that codes for the transfer RNA for threonine.

Mitochondria evolved as bacteria living symbiotically with early cells with nuclei. Most of the mitochondrial genes code for 13 of the 100 proteins involved in extracting and storing energy and in coding transfer RNAs and ribosomal RNAs needed to synthesize these proteins. The mitochondrial genome is interesting in several ways, but the critical point in the present context is that mitochondria is inherited from the cytoplasm of the egg. This explains why no heterozygotes are found for our EST00083 marker-there is only one allele, which is inherited from the mother. Mutations in mitochondrial genes are known to cause several rare diseases such as a type of optic nerve degeneration called Leber's hereditary optic neuropathy, myoclonic epilepsy, and another disease causing seizures and muscle weakness called ragged red fiber disease (Wallace, 1992). More recently, a maternally transmitted late-onset type of diabetes combined with deafness has been traced to mitochondrial DNA (Ballinger, Shoffner, Gebhart, Koontz, & Wallace, 1994). Of greatest potential relevance is speculation that mitochondrial DNA is involved in other neurological disorders during childhood that might affect IQ (Taylor, 1992). Thus, it is not inconceivable that our ESTOO083 marker might be associated with IQ. However, the unexpected nature of this marker suggests caution in claiming that this marker for mitochondrial DNA is indeed a QTL for IQ until the association receives additional replication.

Phase II. Although the first phase of the IQ QTL project could detect QTL of much smaller effect size (about 2%) than can be detected by linkage, even greater power is needed from a QTL perspective. Many genes are likely to affect intelligence and are likely to vary in effect size in a highly skewed distribution with many small effects and very few QTL of effect sizes as large as 2%. For this reason, a second phase of the IQ QTL project is underway that will increase the power to detect QTL associated with IQ by obtaining even more extreme samples, larger samples, and additional replication groups. Specifically, the second phase of the project will include a sample of 50 individuals with IQs exceeding 175. A step in the direction of specific cognitive abilities is also being taken by including two additional samples of 50 individual each: a group in the top .0001 verbally with mathematical scores at least one standard deviation lower, and a group in the top .0001 mathematically with verbal scores at least one standard deviation lower.
The high-ability samples for the second phase of the project have been selected from the Study of Mathematically Precocious youth (SMPY), begun two decades ago by Julian Stanley and now co-directed by Camilla Benbow and David Lubinski (e.g., Lubinski & Benbow, 1992). SMPY includes a total of more than 5,000 gifted students who are currently being tracked. Subjects are selected through above-level testing, a procedure in which 7th and 8th graders scoring in the top 2- 3% on conventional achievement tests are invited to take the College Board Scholastic Aptitude Test (SAT). These students generate score distributions on the SAT indistinguishable from those of 11th and 12th grade high school students. The especially able children are selected for in-depth assessments plus extensive longitudinal tracking at 5- to 10-year intervals. Since 1972, more than a million 7th and 8th graders have been tested with the SAT, and more than 100,000 such students now take the SAT annually. These tests are remarkably predictive of exceptional academic achievements into adulthood (Benbow, 1992).

The second phase of the IQ QTL project focuses on the high end of the IQ distribution as compared to average IQ rather than low IQ in order to focus on high intelligence. An additional control of 50 average-IQ individuals will be included. The rationale for focusing on high intelligence is to attempt to identify positive QTL, that is, QTL that contribute positively to cognitive, functioning. EST00083 is particularly interesting because, unlike the other markers, It shows an allelic frequency difference, not just between low and high IQ groups, but also between average and high IQ groups, suggesting that this marker may be a positive QTL. Combining Phase I and Phase II subjects, permanent cell lines, which provide a renewable resource for DNA for these subjects, will be available for 200 high-IQ individuals and 100 control individuals of average IQ, which will provide power to detect QTL that account for less than 1% of the variance in the population.

Conclusions

Most of what is currently known about the genetics of intelligence comes from twin and adoption studies, which have documented significant and substantial genetic influence. More research of this type is needed to go beyond merely demonstrating the importance and estimating the magnitude of genetic effects. For example, developmental genetic analyses have found that the magnitude of genetic effects increase during the life span and that genetic factors are primarily responsible for stability during development but also affect age-to-age changes. Multivariate genetic analyses suggest that the same genes largely overlap in their effect across most specific cognitive

abilities, that the strong association between intelligence and scholastic achievement is almost exclusively due to the same genes that affect both domains, and that genetic factors mediate the association between cognitive development and home environment. Genetic analyses also suggest that genetic effects on high intelligence may merely be the high end of the normal distribution of genetic influences on individual differences in intelligence.

Such twin and adoption studies will guide molecular genetics research that aims to identify specific genes. So far, only a few genes have been identified that affect cognitive disabilities. Most of these involve relatively rare disorders such as PKU and fragile X for which a single gene is the necessary and sufficient cause of distinct types of mental retardation. The allelic association between Apo-E4 and late-onset Alzheimer's disease is the best example of a QTL associated with cognitive disability. The goal of the IQ QTL project is to identify specific genes responsible for the substantial genetic contribution to intelligence. Although the project has not yet yielded clear evidence for QTL, several interesting leads for future research have been uncovered. The project provides a glimpse of an exciting new chapter in the story of genetics and intelligence.

Acknowledgements

The QTL research was supported by U.S. National Institute of Child Health and Human Development grants HD-27694 and HD-21947. The IQ QTL project is a collaboration with the following co-investigators: Gerald E. McClearn and Deborah L. Smith at the University Park campus of The Pennsylvania State University, Michael J. Chorney at the Milton S. Hershey Medical Center of The Pennsylvania State University, Lee A. Thompson and Douglas K. Detterman at Case Western Reserve University, Michael J. Owen and Peter McGuffin at the University of Wales College of Medicine in Cardiff, Wales, and David Lubinski and Camilla Benbow at Iowa State University.

* Please refer to original for all tables.

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