Genetic Influence on Cognitive Ability

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Introduction

A handbook on giftedness and talent would not be complete without consideration of genetic influence. This chapter focuses on genetic research on one type of talent, general cognitive ability, because more is known about the genetics of this dimension than any other in the social and behavioral sciences. By general cognitive ability, we refer technically to "*g*" which is an unrotated first principal component of diverse tests of cognitive abilities. Because IQ scores from general intelligence tests are reasonable indices of "*g*," we could have used the terms intelligence or IQ scores but we prefer general cognitive ability which is less loaded with unwanted connotations. Although general cognitive ability is important, so are specific cognitive abilities such as verbal, spatial, and memory abilities. However, less is known about the genetics of specific cognitive abilities (Plomin, 1988).

In this chapter, we review research on the genetics of general cognitive ability that converges on the conclusion that this dimension of behavior shows considerable genetic influence. We describe a recent study which is the first to show that genetic factors are important, not only for the normal range of variation in cognitive ability, but also for high cognitive ability. Genetic research on this basic question of the impact of genetics on cognitive ability also raises interesting issues about the environment. In addition, genetic research on cognitive ability has begun to go beyond the basic nature–nurture question concerning the relative effects of genetic and environmental influences to ask more sophisticated questions such as developmental and multivariate questions. The most exciting recent advance is that the power of molecular genetic techniques is beginning to be harnessed in order to identify specific genes responsible for genetic influence on cognitive ability.

Genetics and Ability

Before launching this overview of genetic research on cognitive ability, it may be useful to ask why genetics has been neglected in contemporary discussions of giftedness and talent. For example, most recent books on high ability (e.g., Feldman, 1986; Howe, 1990; Simonton, 1990) pay scant attention to genetics (cf. Storfer, 1990). Indeed, genetics is not mentioned in most of the chapters in this volume. This is ironic because one of the earliest scientific books about ability is also the first book about human genetics. A year before Mendel's seminal paper on the laws of heredity, Francis Galton (1865) published a two-article series on hereditary genius that he expanded into the book *Hereditary Genius: An Inquiry into its Laws and Consequences* (Galton, 1869).

Why is genetic influence neglected? Recent discussions of this issue in relation to the behavioral sciences suggest several possible reasons (Goldsmith, in press; Rowe & Waldman, in press; Rutter, Silberg, & Simonoff, in press). A rudimentary problem is the failure to distinguish between the development of differences among individuals and the development of an individual. Genetic research does not address the development of a single individual. Nor does it address the origins of species-wide developmental themes such as the use of language in the human species. Rather, its focus is on variations (individual differences) on these themes, for example, why some children are delayed in their use of language and why some individuals are more verbally fluent or have larger vocabularies than others.

An historical happenstance also contributes to the neglect of genetics: Behaviorism in the behavioral sciences set the study of behavior apart from the other life sciences. The legacy of behaviorism was to make behavioral scientists uncomfortable with biology. Behaviorism led to environmentalism, especially in the United States. Environmentalism dominated the behavioral sciences for so long that some behavioral scientists still find it difficult to accept a more balanced view that recognizes genetic as well as environmental influences on individual differences in behavioral development.

In our experience, the major source of concern about finding genetic influence in behavioral development

is the mistaken notion that if a trait is influenced genetically it cannot be affected environmentally. This notion is wrong for at least three reasons. First, to recognize genetic influence does not imply that a trait is entirely due to genetic influence. Rarely does heritability exceed 50% for behavioral traits, which means that as much of the phenotypic variance is accounted for by nongenetic as by genetic factors. In this sense, genetic research provides solid evidence for the importance of nongenetic factors.

Second, even if all the variance of a trait can be explained by genetic differences among individuals (and we hasten to add that no behavioral trait begins to approach this hypothetical case), this only describes "what is," not "what could be" or "what should be." Heritability describes the extent to which genetic differences among individuals in a particular population at a particular time contribute to observed (phenotypic) differences for a trait. In this sense, heritability is a descriptive statistic, not a constant like the speed of light. The relative magnitude of genetic and environmental influences can change as the population described changes. For example, one counterintuitive example of change is that genetic differences among individuals would increasingly account for phenotypic differences to the extent that salient environmental factors are equalized. In the case of cognitive ability, this would be the expected outcome of attempts to equalize educational opportunities.

In other words, heritability describes "what is" in a population—the genetic and environmental provenances of measured differences among individuals as they exist in a particular population with its particular mix of genetic and environmental influences. Heritability does not predict "what could be" nor does it prescribe "what should be." If this is understood, the following statement, which is at the crux of concerns about heredity and talent, will not sound paradoxical: Cognitive ability can be highly heritable in a population and yet show dramatic change for an individual who undergoes intense training. Heritability denotes probabilistic genetic influence for a population, not predetermined programming for an individual.

A third reason why it is wrong to think that genetic influences are immutable is that a necessary connection does not exist between the origins of a trait and intervention. Descriptions of "what is" do not have a necessary relationship to "what could be." Specifically, intervention programs for the gifted can be effective regardless of the origins of giftedness.

These concerns coalesce in the seldom-discussed but often-felt issue of political implications. In a recent discussion of this topic, Goldsmith (in press) makes three important points. First, it is wrong to suggest that scientific understanding of certain issues should not be pursued, although care must be taken to avoid interpretations of genetic determinism and other oversimplifications. Second, the rationale for intervention

should be demonstrated effectiveness, not efforts to denigrate evidence of genetic influence. Goldsmith's third point questions the assumption that genetic research will make people more hereditarian:

> It would seem crucial to know what the general public, as well as political leaders, *currently* believe about the relative influence of inheritance and experience in molding behavior. It is not so clear that the public embraces experience over inheritance. Some of my experience suggests that an accurate description of current behavior-genetic findings to public groups outside academia often moves them toward a *less* hereditarian position (Goldsmith, in press).

Our view concerning social and political implications is that finding genetic influence is compatible with a wide range of actions, including no action at all. Values come into play when decisions are made concerning what is to be done with such knowledge. For example, finding genetic influence on cognitive ability by no means implies that those rich in ability must be made richer. Depending on one's values, it could be argued that scarce educational resources should go to those who most need them to function adequately in our increasingly technological society.

If heritability describes "what is" rather than predicts "what could be," then what does it matter whether or not genetic factors are important? The answer to this question depends on the answer to another question: Important for what purpose? For applied issues such as identification and intervention, genetics is unlikely to be of much specific help to the educator confronted with a gifted child. Knowledge of the importance of genetics might be helpful in more general ways. For example, finding genetic influence might aid identification by taking into account familial loading. Knowledge about genetic patterns of strengths and weaknesses might be useful in designing programs tailored to children's needs. Understanding the role of genetics can also affect our interpretation of other research. For example, most research on the development of talent focuses on familial factors such as parental tuition and encouragement. However, in families in which parents are genetically related to their children, it cannot be assumed that such links between parents and children are due solely to environment (Plomin & Bergeman, 1991). Another point is that just as cures are not necessarily related to causes of diseases, understanding causes can lead to more rational cures. In this sense, knowing "what is" is likely to guide us in the search for "what could be." More broadly, although finding genetic influence bears no necessary implications for social action, better decisions ought to be made with knowledge than without it. Finally, identification of specific genes involved in ability, discussed later in this chapter, would add considerably to the practical value of genetic knowledge. Still, for the present, it must be said that applications such as programs for the gifted would proceed largely

unchanged whether or not heredity contributes to the development of ability.

It should be noted, however, that behavioral genetic studies can contribute a great deal to our knowledge of environmental factors that affect behavior. Traditional environmental studies confound genetic and environmental influences; behavioral genetic research can disentangle genetic and environmental main effects, environment–genotype interaction and environment–genotype correlation. Identification of specific environmental factors that affect behavior may provide the best clues for designing effective intervention programs.

The major reason for wanting to know about the origins of ability is the basic science goal of explanation with no promise of practical application. An important first step, although just a first step, in understanding the origins of individual differences in ability is to ask the extent to which genetic factors are involved. This basic science interest in origins is also largely shared by educators and parents in a sense of curiosity about why children develop the way they do. We know that ability runs in families, but does it do so for reasons of nature or nurture?

Evidence for Genetic Influence

Human quantitative genetic research relies on family, adoption, and twin designs. Family studies of human behavior assess the extent to which genetically related individuals living together resemble each other. Such studies cannot disentangle possible environmental sources of resemblance. This was the problem with Galton's 1869 family study of talent in which he interpreted familial resemblance as due to heredity. Separating genetic and environmental sources of familial resemblance is the point of adoption studies. Genetically related individuals adopted apart give evidence of the extent to which familial resemblance is the result of hereditary resemblance. Genetically unrelated individuals adopted together indicate the extent to which familial resemblance is due to shared family environment.

Twin studies also provide a kind of natural experiment in which the resemblance of identical twins, whose genetic relatedness is 1.0, is compared to the resemblance of fraternal twins, first-degree relatives whose genetic relatedness is .50. If heredity affects a trait, identical twins should be more similar for the trait than fraternal twins. As in any quasi-experimental design, these methods have possible problems, most notably, the equal environments assumption for the twin method and selective placement for the adoption method. However, these are empirical issues and research suggests that these are not major problems. Moreover, the assumptions of the twin method are very different from the assumptions of the adoption method and yet the two methods generally converge on the conclusion that genetic effects are important. Details concerning quantitative genetic methods and their application to

behavior are available elsewhere (e.g., Plomin, 1990a; Plomin, DeFries, & McClearn, 1990).

Family, adoption, and twin studies can be used to estimate the magnitude of genetic effects as well as their statistical significance. This is the descriptive statistic heritability. As mentioned earlier, heritability is an estimate of effect size given a particular mix of existing genetic and environmental factors in a particular population at a particular time. Heritability estimates the proportion of phenotypic variance (i.e., individual differences in a population, not behavior of a single individual) that can be accounted for by genetic variance.

Consider height. Correlations for first-degree relatives are about .45 on average, whether relatives are reared together or adopted apart. Identical and fraternal twin correlations are .90 and .45, respectively, regardless of whether they are reared together or adopted apart. These results indicate significant genetic effects. For these height data, heritability is estimated as 90%. This estimate of effect size indicates that, of the differences among individuals in height in the populations sampled, most of the differences are due to genetic rather than environmental differences among individuals.

When these same methods are used to investigate genetic effects on general cognitive ability, they yield evidence for less but still appreciable genetic influence. Correlations for first-degree relatives living together are similar to their correlation for height. In a review of the world's literature of genetic research on IQ scores (Bouchard & McGue, 1981), the weighted average correlation was .42 for 8433 pairs of parents and their offspring in 32 studies. The weighted average correlation was .47 for siblings reared together (26,473 pairs in 69 studies). Unlike height, adopted-apart first-degree relatives are only about half as similar for IQ as are first-degree relatives living together. The average weighted correlation for 814 pairs of parents and their adopted-away offspring is .22; for 203 pairs of adopted-apart siblings the correlation is .24 (Bouchard & McGue, 1981).

The fact that the correlation for adopted-apart relatives is less than the correlation for relatives living together suggests that shared rearing environment contributes to the IQ resemblance of first-degree relatives living together. This fits with another finding from the adoption literature: Genetically-unrelated parents and offspring and siblings are similar. The average correlation for adoptive parents and adopted children is .19 (1397 pairs) and the average correlation for genetically unrelated children adopted into the same adoptive families is .32 (714 pairs; Bouchard & McGue, 1981).

Thus, in very rough summary, "genetic" relatives adopted apart correlate about .20, "environmental" relatives correlate about .20, and "genetic-plus-environmental" relatives correlate about .40. These adoption results are consistent with a heritability estimate of about .40, about half that for height.

The twin method converges on this conclusion. The average twin correlations are .86 for identical twins

(4672 pairs) and .60 for fraternal twins (5546 pairs; Bouchard & McGue, 1981). Because identical twins are twice as similar genetically as fraternal twins, a rough estimate of heritability doubles the difference between the identical and fraternal twin correlations. This estimate of heritability is about .50. It should be noted that the correlation of .60 for fraternal twins exceeds the correlation of .47 for nontwin siblings, which suggests that shared environmental influences contribute more to the resemblance of twins than nontwin siblings.

One of the most dramatic adoption designs, reared-apart identical twins, suggests a higher estimate of heritability than these other designs, although the number of such twin pairs is small for obvious reasons. For several small studies involving a total of 65 pairs of identical twins reared apart, the average correlation is .72 (Bouchard & McGue, 1981). The correlation for identical twins reared apart provides a direct estimate of heritability. This high heritability estimate has been confirmed in two recent studies of twins reared apart. In one report of 45 pairs of identical twins reared apart, the correlation was .75 (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). In a report of 45 pairs of Swedish identical twins reared apart, the correlation was .78 (Pedersen, Plomin, Nesselroade, & McClearn, 1992). The latter study also included 88 pairs of fraternal twins reared apart whose IQ correlation was .32 and matched identical and fraternal twins reared together. A model-fitting estimate of heritability that incorporates data from the four groups of twins in this study was .81, and a follow-up study three years later yielded similar results (Plomin, Pedersen, Lichtenstein, & McClearn, 1993). A possible explanation for this higher heritability estimate for twins reared apart is that, unlike most of the other twin and adoption studies, these studies involve adults rather than children and adolescents. As explained later, heritability appears to be greater later in life.

Model-fitting analyses that simultaneously analyze all of the family, adoption, and twin data summarized in the review by Bouchard and McGue (1981) yield heritability estimates of about .50 (Chipuer, Rovine, & Plomin, 1990; Loehlin, 1989). The error surrounding this estimate may be as high as .20, so we can only say with confidence that the heritability of IQ scores is .50 ± .20. Nonetheless, even if heritability is at the bottom of this range, it is a remarkable achievement to account for so much of the variance of a trait as complex as general cognitive ability.

If half of the variance of IQ scores can be accounted for by heredity, the other half cannot and is attributed to environment. In this sense, these same genetic data provide strong evidence for the importance of the environment. These data also indicate how the environment works. Some of this environmental influence appears to be shared by family members making them similar to one another. For example, as indicated earlier, pairs of genetically unrelated children adopted into the same adoptive homes yield an average correlation of .32. This

suggests that about a third of the total variance of IQ scores may be due to a shared rearing environment. The average correlation of .19 between adoptive parents and their adopted children suggests less shared environmental influence, although it seems reasonable that parents and their children share less similar environments than do siblings. However, when we consider these data from a developmental perspective, a very different picture emerges, as described in the following section.

Beyond Nature–Nurture

A survey of more than 1000 social and behavioral scientists and educators indicated that most had accepted the evidence for a significant effect of heredity on IQ scores, traditionally one of the most controversial areas in behavioral genetics (Snyderman & Rothman, 1988). General acceptance of the important genetic contribution to individual differences in *g* makes it possible to go beyond this most basic nature–nurture issue to ask more interesting questions. Three examples will be mentioned briefly. The first concerns development.

Developmental Genetic Analysis: Genetic Change as Well as Continuity

When Galton first studied twins in 1876, he investigated the extent to which the twins' initial similarity or dissimilarity changed during development. Other early studies were also developmental, but this developmental perspective faded from genetic research until recent years.

Two types of developmental questions can be addressed in genetic research (Plomin, 1986). First, does heritability change with age? It is reasonable to suppose that environmental factors increasingly account for variance in cognitive ability as experiences accumulate during the course of life. To the contrary, genetic research suggests that cognitive ability shows a nearly linear increase in genetic influence from infancy through early childhood (Cardon, Fulker, DeFries, & Plomin, 1992; Fulker, DeFries, & Plomin, 1988; Wilson, 1983) and perhaps continuing throughout the lifespan (McCartney, Harris, & Bernieri, 1990; McGue, Bouchard, Iacono, & Lykken, in press; Plomin & Thompson, 1987). For example, as mentioned earlier, a recent report of the first genetic study of older adults reports a heritability of 80% for cognitive ability using the powerful design of twins reared apart and twins reared together (Pedersen et al., 1992).

Not only does heritability appear to increase during the lifespan, but the effects of shared environment appear to decrease. Although genetic research suggests substantial influence of shared environment as discussed in the previous section, recent evidence suggests that shared environmental influence that affects IQ scores may be much less after adolescence (Plomin, 1988).

The strongest evidence for the importance of shared environment comes from the correlation for adoptive siblings, that is, pairs of genetically unrelated children adopted into the same adoptive families. As indicated earlier, their average IQ correlation is .32. However, these studies happened to study adoptive siblings in childhood. In 1978, the first study of older adoptive siblings yielded a strikingly different result: The IQ correlation was −.03 for 84 pairs of adoptive siblings from 16 to 22 years of age (Scarr & Weinberg, 1978). Other studies of older adoptive siblings have also found similarly low IQ correlations (Kent, 1985; Teasdale & Owen, 1984). The most impressive evidence comes from a 10-year longitudinal follow-up study of over 200 pairs of adoptive siblings. At the average age of 8 years, the IQ correlation was .26. Ten years later, their IQ correlation was near zero (Loehlin, Horn, & Willerman, 1989). These data suggest that shared environment is important for IQ during childhood when children are living at home and then fades in importance after extrafamilial influences become more important.

A second type of developmental question concerns genetic contributions to changes with age and continuity in longitudinal analyses. It is important to recognize that genetic factors can contribute to change as well as to continuity in development (Plomin, 1986). Although genetic effects on cognitive ability contribute substantially to stability of cognitive ability during childhood, what is more surprising is the extent to which genetic effects appear to contribute to change with age (Fulker, Cherny, & Cardon, in press). Particularly interesting is the suggestion of substantial new genetic variation during the transition from early to middle childhood.

Multivariate Genetic Analysis: Genetic g

A second example of research that goes beyond the basic nature–nurture question is multivariate genetic analysis, which extends the univariate genetic analysis of the variance of a single trait to multivariate analysis of the covariance between traits. Multivariate genetic analysis makes it possible to estimate the extent to which genetic effects on one trait overlap with genetic effects on another trait. Analyses of this type in the realm of cognitive abilities indicate that specific tests and group factors show some genetic effects unique to each test and factor (Pedersen, Plomin, Nesselroade, & McClearn, 1993). Nonetheless, much of the genetic effects are shared in common across diverse tests and factors (Cardon & Fulker, in press).

Another recent finding makes a related point: The heritabilities of cognitive tests are strongly correlated with their *g*-loadings, their factor loadings on an unrotated first principal component (Jensen, 1987). That is, the more a test measures *g*, the more heritable it is. For example, in the study mentioned earlier of twins reared apart and twins reared together, the correlation between heritabilities and *g*-loadings

was .77 after differential reliabilities of the tests were controlled (Pedersen et al., 1992).

School Achievement and g: Same Genes, Different Environments

The third example also involves multivariate genetic analysis, but it is especially relevant to the origins and development of ability. School achievement is interesting from a genetic perspective because it is widely assumed that achievement and ability are different, almost by definition. Achievement is what a student achieves by effort, whereas ability is thought to involve inherent talent. For this reason, achievement test scores are assumed to be environmental in origin. However, a neglected finding is that achievement and ability tests are moderately correlated, which raises the possibility of genetic overlap between the two domains.

Although several twin studies of scholastic achievement have been reported in adolescence, until recently no research was available in middle childhood. In the *Western Reserve Twin Project* (WRTP), specific cognitive abilities and school achievement were investigated for a sample of 146 pairs of identical twins and 132 pairs of fraternal twins aged 6–12 (Thompson et al., 1991). Although school achievement tests yielded significant heritability estimates, these estimates were much lower than heritabilities for cognitive abilities—about .20 vs about .70. Most important are the results of multivariate genetic analysis: The well-known correlation between cognitive abilities and school achievement tests is due almost entirely to genetic factors in common to the two domains, a finding that has been replicated in another study (Wadsworth, in press) as well as in two studies focused on reading achievement (Brooks, Fulker, & DeFries, 1990; Cardon, DiLalla, Plomin, DeFries, & Fulker, 1990). Conversely, ability–achievement discrepancies are exclusively environmental in origin.

Although ability–achievement discrepancies have not been studied for high cognitive ability from a quantitative genetic perspective, a study addressing this question could have important practical implications. For instance, would the environment completely mediate ability–achievement discrepancies for high cognitive ability as it does for the entire range? For high cognitive ability these discrepancies must be manifested as underachievement. Identification of specific environmental factors that lead to underachievement in an extremely talented group could lead to changes in educational practice that would minimize underachievement by talented children.

Quantitative Genetics and High Cognitive Ability

The research described above addresses the etiology of individual differences in cognitive ability in the normal range. Much less is known about the origins of high

ability. It cannot be assumed that the etiology of the extremes of a dimension is the same as the etiology in the normal range. For example, at the low end of the ability spectrum, severe retardation shows little familiality in contrast to the rest of the distribution of cognitive ability (Plomin, 1991).

Galton's 1869 study of talent was a family study. Since there was no satisfactory way to quantify ability, Galton relied on reputation as an index. By "reputation," he did not mean notoriety for a single act, nor mere social or official position, but "the reputation of a leader of opinion, of an orginator, of a man to whom the world deliberately acknowledges itself largely indebted" (1869, p. 37). The designation "eminent" was applied to those individuals at the rank of 1 in 4000. The majority of individuals Galton considered to be the cream of this elite group, ranked as one in a million, and were termed "illustrious."

Taking the most eminent person in each family as a reference point, the other individuals who attained eminence were tabulated with respect to closeness of family relationship. Galton's results indicated that eminent status was more likely to appear in close relatives, with the likelihood of eminence decreasing as the degree of relationship became more remote. Eminence was attained by 26% of the fathers of the 100 most distinguished men, 23% of their brothers, and 36% of their sons. Second-degree relatives such as grandfathers, uncles, nephews, and grandsons achieved eminence to a much lower degree (about 7%), but much higher than the overal incidence of 1 in 4000 (i.e., .025%). These results conflate abilities of many different sorts, not just cognitive ability. Moreover, as indicated earlier, such familial resemblance cannot be attributed to heredity as Galton did. Galton started the nature–nurture controversy by overinterpreting his results to conclude that "ability will out" regardless of environment.

It is surprising that during the past century very little research has addressed the issue of the genetic and environment origins of high cognitive ability. Oden (1968) reported that, in a sample of gifted individuals who all had IQs of over 135 in Terman's study of the gifted, the 1571 offspring of these individuals yielded an average IQ of 133. Another study reported parent–offspring resemblance in another reanalysis of the Terman data (McAskie & Clarke, 1976). The analyses included 559 gifted parents with IQs over 135 and their 1027 offspring. Interestingly, the parent–offspring correlation was not significantly different from zero ($r = 0.08$) even though as a group the offspring mean resembled their parents' mean, as seen in the Oden (1968) report. McAskie and Clarke's finding is most likely due to the extremely truncated sample which attenuates the parent–offspring correlation. This is a problem that is inherent in studies of individual differences in extreme groups.

These two studies illustrate an important distinction that must be made when genetic and environmental influences on the origins of high cognitive ability are explored. The isue is whether genetic factors affect high ability and how the magnitude of this genetic influence compares to the magnitude of genetic factors that contribute to individual differences in the normal range. The issue is not the heritability of high ability in the usual sense of genetic contributions to differences among high-ability individuals. The reasons why one child has an IQ of 150 and another an IQ of 145 are less important than understanding why both children have such high IQ scores as compared to the rest of the population.

This question can be addressed using a new approach that leads to an estimate of what is called *group heritability*, in contrast to the traditional heritability statistic which could be called *individual heritability*. Group heritability refers to the genetic contribution to the average difference between a selected group such as children of high ability and the rest of the population. The typical approach to group heritability in genetic research on disorders is to establish a cut-off score as a diagnostic index of the disorder (i.e., normal vs abnormal). Concordances can be calculated and compared for identical and fraternal twins, or liability (tetrachoric) correlations can be used which assume a continuous distribution even though the data, as they are used, are discontinuous.

A far superior approach has been developed by DeFries and Fulker (1985) and has been called DF analysis after its developers (Plomin & Rende, 1991). DF analysis requires that the continuum be assessed rather than assumed. It assesses group heritability as the differential regression to the population mean of the co-twins of identical and fraternal twin probands for a quantitative measure. That is, IQ scores of co-twins of probands ascertained because of high IQ scores are expected to regress toward the mean of the unselected population. However, to the extent that high ability is due to genetic factors, the regression to the mean will be less for identical twin co-twins than for fraternal twin co-twins. DF analysis was first applied to reading disability (DeFries et al., 1987). Group heritability for reading disability was found to be only about half the magnitude of individual heritability for reading ability, suggesting that the disorder of reading disability may be different etiologically than the dimension of reading ability.

Is group heritability of high ability significant? What is the magnitude of group heritability for high ability? The WRTP twin sample was used to estimate group heritability (Thompson et al., 1993). The DF approach was applied to IQ scores from traditional intelligence tests (Weschler Intelligence Scale for Children-Revised and the Peabody Picture Vocabulary Test) expressed as a composite standard score with a mean of 0.0 and a standard deviation of 1.0. High ability was operationally defined as IQ scores 1.25 standard deviations above the sample mean.

As a preliminary analysis, concordances were calculated

separately for identical and fraternal twin pairs in which at least one member of the pair scored 1.25 standard deviations above the sample mean. Probandwise concordances were used in which the total number of affected individuals in concordant pairs is divided by the sum of the total number of affected individuals in concordant and discordant pairs. The probandwise concordances were 62% (18/29) for identical twins and 25% (6/24) for fraternal twins, suggesting genetic influence for high ability.

These twin concordances suggest that group heritability for high ability is additive in that the DZ (dizygotic) concordance is roughly half the magnitude of the MZ (monozygotic) concordance. This finding for high ability does not contradict the hypothesis that rare genius may be epistatic (Lykken et al., 1992). The genetics of genius may differ from the genetics of high cognitive ability. For example, specific constellations of many genes may be required for genius. The hallmark of such epistatic interactions is resemblance for MZ twins but not for first-degree relatives.

DF analysis provides an estimate of the magnitude of group heritability using continuous IQ scores rather than dichotomizing the scores. The gist of the DF approach to group heritability compares the mean score of the co-twins of identical twin probands to that of the co-twins of fraternal twin probands. Group heritability is suggested to the extent that the mean score of the former group regresses less far to the population mean than does the mean score of the latter group. For the high IQ sample, the mean standardized score of the probands is 1.67 and the population mean is 0.00. The co-twins of the identical twin probands regress very little to the population mean—their mean score is 1.53. In contrast, the co-twins of the fraternal twin probands regress more than halfway to the population mean—their mean score is 0.76. This pattern of results supports the suggestion of group heritability for high ability that emerged from the comparison of twin concordances.

DF analysis employs an elegant regression model which tests the differential regression to the mean for identical and fraternal twin probands, taking into account mean differences between the probands. The analysis yields estimates of group heritability and provides standard errors of the estimates. The analysis, described elsewhere (DeFries & Fulker, 1985, 1988), estimates group heritability as .67 ± .24 for high ability. This significant group heritability suggests that about two-thirds of the difference between the IQs of the children in the high-ability group and the average IQ in the sample can be attributed to genetic factors.

This estimate of group heritability is similar to the traditional individual heritability for cognitive ability obtained in this same sample (Thompson et al., 1991). Together these results suggest an hypothesis with far-reaching implications: In terms of genetic influence, high cognitive ability may be merely the extreme of the normal continuum of cognitive ability.

Molecular Genetics and High Cognitive Ability

We are at the dawn of a new era in which molecular genetic techniques will revolutionize genetic research on behavior by identifying specific genes that contribute to genetic variance in behavioral dimensions and disorders (Plomin, 1990b). We have begun to employ these techniques in our research in order to identify specific genes that affect high cognitive ability (Aldhous, 1992).

It was only ten years ago that the now-standard techniques of the "new genetics" were first employed to identify genes responsible for single-gene disorders. As described elsewhere (e.g., Plomin et al., 1990), the discovery of restriction enzymes, which led to recombinant DNA and the ability to sequence DNA, also produced thousands of new DNA markers, genetic differences among people that involve DNA itself rather than gene products such as the blood groups. These new DNA markers can be used to identify a chromosomal region and, eventually, to isolate a gene and a gene product for single-gene disorders.

Notable early successes include cystic fibrosis and Duchenne muscular dystrophy. These are dichotomous traits, like Mendel's smooth vs wrinkled seeds, in which one gene is necessary and sufficient to explain the observed difference. Although several thousand single-gene disorders, most very rare, have been reported, behavior is much more complex. Behavior reflects the functioning of the whole organism and it is dynamic, changing in response to the environment. Genes that affect behavioral traits are transmitted hereditarily according to Mendel's laws in the same way as genes that affect any other phenotype. However, behavior is special in three ways. First, unlike Mendel's smooth vs wrinkled seeds, most behavioral dimensions and disorders are not distributed in simple either/or dichotomies such as talented vs not talented, although in psychopathology we often pretend that a line exists that sharply separates the normal from the abnormal. Second, behavioral traits are substantially influenced by nongenetic factors: heritabilities rarely exceed 50%. Third, genetic effects on behavioral dimensions such as cognitive ability are likely to involve many genes of variable but generally small effect size. Each of these points applies to the dimension of cognitive ability and to the dichotomy of convenience that we call high cognitive ability. The challenge is to use DNA markers to find genes in these complex systems of behavior that 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).

Linkage

For a single-gene trait, linkage is a method guaranteed to find the chromosomal location of the gene, even when nothing is known about the gene product. Linkage traces

the co-transmission of a marker and a disorder within a family pedigree. The exemplar is Huntington's disease, which was the first disorder mapped to a chromosome using the new DNA markers. Huntington's disease has long been known to be a single dominant gene that is lethal later in life regardless of a person's other genes or environment. Other single-gene disorders are quickly being put on the genome map through the use of linkage.

The problem is that behavioral dimensions are different. Most importantly, they show no suggestion of simple single-gene inheritance. Linkage can only identify a major gene that is largely responsible for a disorder. For the analysis of behavior, reliance on linkage techniques that can only detect major-gene effects is an example of losing one's wallet in a dark alley but looking for it in the street because the light is better there. It is now generally recognized that no major gene for behavioral dimensions or disorders is likely to be found in the population. However, current linkage research assumes that a major gene can be found in certain families. For this reason, linkage studies focus on large pedigrees with many affected individuals in the hope of finding a major gene responsible for the disorder in a particular pedigree. In this view, multiple-gene influence is seen at the population level because different major genes are responsible for the disorder in different families.

The alternative view espoused here is that major genes will not be found for behavior either in the population or in a family. Rather, for each individual, many genes make small contributions to variability and vulnerability. In this view, the genetic quest is to find, not *the* gene for a behavioral trait, but the QTL that affect the trait in a probabilistic rather than predetermined manner. Although some sledgehammer effects of major genes may be found, it seems more likely that many other alleles nudge development up as well as down for many individuals and do not show dramatic effects as in the classical single-gene disorders.

The point is not that behavior is too complex to take advantage of the new DNA markers, but rather that we need to bring the light of molecular genetics into the dark alley. New strategies are needed to identify genes that affect behavioral traits, even when the genes account for only a small amount of variance, when nongenetic factors are important, and when the traits are quantitatively distributed. That is, we need to use molecular genetic techniques in a quantitative genetic framework.

Allelic Association

Given the breathtaking pace of technological advances in molecular genetics, the safest bet is that at the turn of the century we will be investigating multiple-gene influences for complex dimensions and disorders using completely different techniques from those in use today.

However, one strategy that we are using in the meanwhile is called allelic association. Linkage refers to loci rather than alleles—linked traits such as hemophilia and color-blindness do not occur together in the population. In contrast, allelic association occurs when a DNA marker is so close to a relevant gene (or it is part of the gene) that affects the trait that a marker allele is correlated with the trait in unrelated individuals in the population.

The best case is when the marker is the relevant gene itself. That is, the marker can be in the coding region of a gene and thus code for actual polypeptide differences among people. Some background information is necessary here. Most DNA involves noncoding regions between genes that are not transcribed into messenger RNA and thus are not translated into amino acid sequences. Moreover, much DNA in a transcribed gene is spliced out (so-called introns) and is not translated into amino acid sequences that form polypeptides. Of the thousands of known markers, only a handful are known to be in coding sequences (exons). An example of functional markers that happen to be in coding sequences are markers for two of the five types of dopamine receptors, D_3 and D_4 dopamine receptors. In the case of dopamine D_4 receptor, it is known that receptor binding differs for people with different marker genotypes. However, the vast majority of current markers are likely to be in noncoding regions because natural selection permits variation in nonfunctional DNA much more than in functional DNA. Finding such functional markers is a high priority for research because of the power they provide for identifying QTL (Sobell, Heston, & Sommer, 1992).

Allelic association makes it possible to use markers that are not functional themselves but are very close to functional genetic variation. A particular combination of a marker allele and a functional variation in DNA that happen to be on the same chromosome is rarely separated by recombination (meiotic crossing over of chromosomes) if their loci are very close together on the chromosome.

Allelic associations have been found between disease states and markers in the HLA histocompatibility complex (Tiwari & Terasaki, 1985). In other words, particular alleles in this complex increase risk for certain diseases. For normal variation, the best example of allelic association is serum cholesterol levels for which about a quarter of the variance can be explained by four apolipoprotein gene markers (Sing & Boerwinkle, 1987). In psychiatry, a marker in the D_2 dopamine receptor has been reported in several studies to be associated with alcoholism (Cloninger, 1991). That is, the frequency of this allele appears to be greater in severe alcoholics than in controls, although failures to replicate have been reported.

A major advantage of allelic association analysis is that it can use samples of unrelated individuals, whereas linkage requires pedigrees of related individuals. In addition, allelic association is just as applicable to

quantitative traits as to disorders. Most importantly, by increasing the sample size of relatively easy-to-obtain unrelated subjects, association analysis can be made sufficiently powerful to detect small genetic effects.

A problem is that there are so many DNA markers. The allelic association approach is like a myopic search for a few needles in a haystack. In contrast to linkage which can detect a major gene far away from a marker, allelic associations can only be detected when a marker is very near a gene that affects the trait. For behavioral traits influenced by many genes as well as nongenetic factors, a near-sighted strategy such as association may be needed to see fine details of the landscape near a marker even though it has to sacrifice the ability to see distant mountains. This is not really a sacrifice because there are no mountains to be seen. Nonetheless, there are so many markers that randomly drawing straws from the haystack is unlikely to pay off. The odds can be stacked in our favor by beginning the search using markers in or near genes of neurological relevance. The odds can also be improved by using large samples and well-measured extreme groups to increase the power to detect small effects. The goal is to identify some, certainly not all, genes that contribute to the ubiquitous genetic variance found for behavioral traits.

Allelic Association and High Ability

We are using this allelic association approach in an attempt to identify QTL associated with high cognitive ability. DNA markers are employed that are in or near genes with possible neurological relevance such as the many neuroreceptor genes and genes involved in the regulation of these genes. From the WRTP sample of more than 500 children, we selected three groups of Caucasian children: 24 children with the highest IQ scores, 21 with average IQ scores, and 18 with the lowest IQ scores. The average IQ scores of the three groups are 130, 104, and 80, respectively.

We have obtained blood from these children and established permanent cell lines by transforming the lymphocytes with Epstein–Barr virus. Permanent cell lines provide unlimited amounts of DNA for marker analyses as well as a permanent resource for future DNA analyses of these samples. We have compared allelic frequencies for these groups for more than 20 DNA markers, including dopamine receptor 1 and 3, monoamine oxidase B, myelin associated protein, neurofilament protein, and fragile X repeat length. Although interesting preliminary results are beginning to emerge, we have agreed not to publish these results until we have replicated them in an independent sample. Our replication sample includes children with even higher IQ scores (mean IQ of 142), as well as children with even lower IQ scores (mean IQ of 74).

Nearly all molecular genetic research focuses on disorders, looking for DNA markers linked or associated with disruptions of normal development. We are especially interested in identifying "increasing" alleles that contribute to high ability. For this reason, the pattern of results we are particularly interested in finding is one in which the allelic frequency is similar for the low-IQ and middle-IQ groups but different for the high-IQ group.

Conclusion

More is known about the origins of individual differences in general cognitive ability than any other behavioral dimension, although we are still closer to the beginning than to the end of the behavioral genetics story (Plomin & Neiderhiser, 1991). It is clear that genetics plays a major role in the story, and our DF analysis indicates that high ability is also strongly heritable. The convergence of evidence for the importance of genetic influence suggests that the current neglect of genetics in research on giftedness and talent needs to end.

Molecular genetics provides powerful tools that can be used to identify DNA differences among individuals without relying on familial resemblance. In addition to providing indisputable evidence of genetic influence, it will revolutionize genetic research by providing a measured genotype for investigating multivariate and longitudinal genetic issues, the links between the normal and abnormal, and interactions and correlations between genotype and environment. In a broader perspective, it will help to integrate genetic research in the increasingly fractionated biological and behavioral sciences at the universal level of DNA. The much-used phrase *paradigm shift* seems no exaggeration for advances of this magnitude. As is the case with most important advances, it will raise new ethical issues as well (Wright, 1990).

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