GENETIC AND ENVIRONMENTAL INFLUENCES ON HUMAN BEHAVIORAL DIFFERENCES

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ABSTRACT

Human behavioral genetic research aimed at characterizing the existence and nature of genetic and environmental influences on individual differences in cognitive ability, personality and interests, and psychopathology is reviewed. Twin and adoption studies indicate that most behavioral characteristics are heritable. Nonetheless, efforts to identify the genes influencing behavior have produced a limited number of confirmed linkages or associations. Behavioral genetic research also documents the importance of environmental factors, but contrary to the expectations of many behavioral scientists, the relevant environmental factors appear to be those that are not shared by reared together relatives. The observation of genotype-environment correlational processes and the hypothesized existence of genotype-environment interaction effects serve to distinguish behavioral traits from the medical and physiological phenotypes studied by human geneticists. Behavioral genetic research supports the heritability, not the genetic determination, of behavior.

INTRODUCTION

One of the longest, and at times most contentious, debates in Western intellectual history concerns the relative influence of genetic and environmental factors on human behavioral differences, the so-called nature-nurture debate (Degler 1991). Remarkably, the past generation of behavioral genetic research has led

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many to conclude that it may now be time to retire this debate in favor of a perspective that more strongly emphasizes the joint influence of genes and the environment. Nonetheless, the controversy surrounding the recent publication of *The Bell Curve* (Herrnstein & Murray 1994) shows that the proposition that genetic factors influence fundamental aspects of our human nature continues to inflame passions.

Human behavioral genetics seeks to identify and characterize both the genetic and the environmental sources of individual differences (phenotypic variance) in human behavior. As this topic has not been previously reviewed in this series, we have taken a broader perspective than might be customary, electing to consider the past 25 years of behavioral genetic research, albeit with a decided emphasis on research published in the past 5 years. The reader may also want to consult the recent general review of this area by Rose (1995), as well as specific reviews of behavioral genetic research on crime and violence (Bock & Goode 1996), behavioral medicine (Turner et al 1995), psychiatric genetics (Blum & Noble 1997, McGuffin et al 1994), intelligence (Sternberg & Grigorenko 1997), and personality (Loehlin 1992). Our review is organized around three broad aspects of behavioral genetic research—(*a*) the nature of genetic influence, (*b*) the nature of environmental influence, and (*c*) models for the joint influence of genes and the environment—and is focused on three broad domains of psychological functioning—(*a*) cognitive ability, (*b*) personality and interests, and (*c*) psychopathology. We do not review research on mental retardation and neurogenetic disorders such as Alzheimer's disease and epilepsy.

METHODOLOGICAL CONSIDERATIONS

In standard biometrical formulations, the phenotypic variance is decomposed into genetic and environmental components. The genetic component is further decomposed into additive and nonadditive components, the latter reflecting interactive effects within (dominance) and among (epistasis) loci. The environmental component is decomposed into a shared environmental component, representing the effects of characteristics such as family income, parental strategies on child-rearing, and level of intellectual stimulation within the home that are shared by reared together relatives and are thus a potential source of their behavioral similarity; and a nonshared environmental component, representing the effects of characteristics such as accidents, peer affiliations, and differential parental treatment that are not shared by reared together relatives and are thus a source of their behavioral dissimilarity. Three general strategies have been used to resolve the separate influence of genetic and shared environmental factors on the familial resemblance that characterizes the vast majority of behavioral traits: twin studies, adoption studies, and gene identification methods.

The classical twin study involves the comparison of monozygotic and dizygotic twins reared together (MZTs and DZTs). If genetic factors influence the trait in question, MZTs, who share 100% of their genetic material, should be more similar than DZTs, who, like ordinary siblings, share on average only 50% of their genetic material. In a classical twin study, the proportion of phenotypic variance associated with additive genetic factors (i.e. the narrow heritability) is estimated by doubling the difference in correlation between the MZTs and DZTs, the contribution of shared environmental factors is estimated by subtracting the heritability estimate from the MZT correlation, and the contribution of nonshared environmental factors and measurement error is estimated by subtracting the MZT correlation from 1.0. These estimates, like any statistics, can change over time and vary across culture; nonetheless, they have proven to be useful indices for characterizing the sources of individual differences in psychological traits (e.g. Neisser et al 1996). Powerful methods for analyzing twin data and estimating environmental and genetic components of variance are now available (Neale & Cardon 1992). Owing to the availability of several large population-based twin registries in Western Europe, the United States, and Australia, the classical twin study is a popular behavioral genetic design. The assumptions that underlie the classical twin study have drawn substantial empirical attention that has generally supported the basic validity of this method (Plomin et al 1990b).

An adoption study involves determining the degree to which adopted individuals resemble both their biological relatives, an indication of genetic influences, as well as their adoptive relatives, an indication of shared environmental influences. Although there are some notable US adoption studies, most adoption research has been undertaken in Scandinavian countries, where the availability of national registries has allowed researchers to ascertain relatively large and representative cohorts of adopted individuals as well as both their adoptive and biological relatives. As is the case with twin studies, the assumptions that underlie the adoption study have drawn much empirical investigation, most of which is generally supportive of the utility of this method (Cadoret 1986, Plomin et al 1990b). Nonetheless, one limitation bears noting. As adoptive homes are likely to underrepresent those who are living at the extremes of poverty and deprivation, the importance of environmental influences may be underestimated in adoption studies. Environmental inferences may apply only to the broadly constituted middle class.

Increasingly, behavioral geneticists are using molecular genetic techniques in an attempt to identify the genes implied to exist by twin and adoption studies, an effort that has been greatly aided by the development of a comprehensive human linkage map. In contrast to classical human genetic phenotypes such as Huntington's disease, phenylketonuria, or cystic fibrosis—which are

fully penetrant, homogeneous, single-gene disorders—behavioral phenotypes are influenced by both environmental and genetic factors and are most likely heterogeneous. Moreover, for psychiatric disorders, risk to MZT cotwins typically exceeds by more than a factor of two the corresponding risk to first-degree relatives, implying that the underlying genetic diathesis is the result of several (oligogenic) or many (polygenic) genes (Risch 1990), adding further complexity to attempts at gene identification. Success in identifying the multiple genes influencing risk for disorders like Type I diabetes (Todd 1995) may provide a useful model for those investigating complex psychiatric phenotypes.

Most systematic efforts at gene identification for behavioral traits have taken one of two approaches. In a linkage study, within-family associations between disease status and genetic marker status serve to identify chromosomal regions likely to contain a disease susceptibility locus. A genome-wide search with approximately 400 to 600 markers distributed throughout the human genome provides an average marker density of less than 10 cM, and a reasonable likelihood of finding linkage if the risk-increasing allele is common (frequency $> .01$) and has a large effect on disease risk (risk ratio ≥ 4.0) (Risch & Merikangas 1996). In an association study, a population association between disease status and genetic marker status indicates that the marker either directly influences disease risk (i.e. is a candidate gene) or is physically proximal and in linkage disequilibrium with a disease susceptibility locus. Currently, there is debate as to which approach is preferable with complex behavioral phenotypes. On the one hand, there is concern that linkage studies may not be sufficiently powerful to identify the genes of modest effect that may constitute the genetic basis for many behavioral phenotypes (Risch & Merikangas 1996). On the other hand, association studies are especially susceptible to false positive findings, owing to imperfect matching of cases with controls, and there are at present a limited number of candidate genes for behavioral characteristics, given the relatively small proportion of genes expressed in human brain that have thus far been identified (Gelernter 1997).

THE NATURE OF GENETIC INFLUENCE

Twin and Adoption Studies Document the Heritable Nature of Most Psychological Traits

COGNITIVE ABILITIES   General cognitive ability, or IQ, has been more extensively studied from a behavioral genetic perspective than any other psychological trait. Model fitting analyses of the combined IQ kinship correlations (Bouchard & McGue 1981) result in heritability estimates of approximately .50, shared environmental influences of .20 and .30, and the balance of variance

being accounted for by nonshared environmental effects and measurement error (Chipuer et al 1990, Loehlin 1989). These analyses, however, do not take age into account, and recent evidence suggests that the heritability of general cognitive ability varies with age. In a landmark longitudinal twin study, Wilson (1983) observed little difference in MZT and DZT correlation for mental ability in the first 3–6 months of life (correlations of about .68) but did observe divergence in correlation thereafter until age 15 years, when the MZT correlation for IQ equaled .86 and the DZT correlation equaled .54. IQ studies of adult twins, although limited in number and size, extend this pattern by finding an average MZT correlation of .83 and an average DZT correlation of .39 (McGue et al 1993). Finkel et al (1995) analyzed general cognitive ability data from adult MZTs and DZTs participating in the Minnesota Twin Study of Adult Development and Aging (MTSADA) and the Swedish Adoption/Twin Study of Aging (SATSA). The heritability of IQ did not vary significantly across the younger (age 27 to 50 years), middle-aged (50 to 65), and older (65 to 88) MTSADA samples (common estimate was .81) but did decline significantly in the older SATSA sample (estimate of .58 in this group). The heritability of IQ thus appears to be substantial throughout much of adulthood, but declines perhaps very late in life.

The five studies of monozygotic twins reared apart (MZAs), almost all of whom were assessed as adults (Bouchard et al 1990a, Juel-Nielsen 1965, Newman et al 1937, Pedersen et al 1992, Shields 1962), have reported IQ correlations ranging from .64 to .78, with a weighted average of .75 (a direct estimate of the total contribution of genetic factors or the broad heritability). The substantial MZA IQ correlation cannot be accounted for by contact between the twins, either prior to or after their separation, or by the placement of the twins in homes similar in their trait-relevant environments (Bouchard 1997a, Pedersen et al 1992). It is moreover inconceivable that MZA twins share rearing environmental factors to a greater degree than two nonbiologically related but reared together siblings (adoptive siblings). The IQ correlations for the latter (a direct estimate of the shared environmental contribution to variance) average only .04 in the four studies of adult samples (Loehlin et al 1997, Scarr & Weinberg 1978, Scarr et al 1993, Teasdale & Owen 1984).

The substantial estimate of IQ heritability from twin studies is consistent with adoption research. Teasdale & Owen (1984) systematically identified four types of siblings from young males who had completed an IQ assessment as conscripts in the Danish military. All males in Denmark (fit for service or not) complete this test, so this is the most representative sample ever used for assessing genetic influences on IQ. They reported correlations of .47 for full siblings reared apart as compared with .52 for full siblings reared together, .22 for half-siblings reared apart, and .02 for adoptive siblings reared together.

These correlations suggest a substantial heritability and little shared environmental influence on general cognitive ability. In a longitudinal study of IQ that incorporated 14 separate adoptive and biological kinship pairings, Loehlin and colleagues (1997) reported that the heritability of IQ increased from adolescence to early adulthood, equaling .78 (when corrected for unreliability) at last follow-up.

Specific mental abilities (SMAs) appear to be somewhat less heritable than general cognitive ability. In the SATSA study of reared apart and reared together adult twins (average age of 64 years) (Pedersen et al 1992), average heritability estimates for three verbal, three spatial, two perceptual speed, and five memory tests were .58, .46, .58, and .38, respectively. In contrast, the estimate of heritability for the first principal component (a measure of general cognitive ability) was .81. Estimates of common environmental influence were .09, .07, .00, and .00, for the four SMA domains, respectively, and .00 for the general cognitive ability measure. Bouchard and colleagues (1990b) reported an average heritability of .49 (ranging from .14 to .69) for 26 SMA tests from MZA and DZA participants in Minnesota Study of Twins Reared Apart (MISTRA). When the MISTRA data is combined with a meta-analysis of SMA correlations from reared together twins, the estimates of heritability are .48, .60, .64, and .48 for the verbal, spatial, perceptual speed, and memory domains, respectively, while the corresponding estimates of common environmental influence are .21, .00, .00, and .00 (Bouchard 1997b). Finkel & McGue (1993) have also reported heritability estimates ranging from .56 to .64 for tests of memory in an elderly twin sample. The results of recent SMA studies are thus quite comparable to results from previous studies (DeFries et al 1976, Nichols 1978) and suggest that SMAs have a heritability of approximately .50 and a modest shared environmental component.

PERSONALITY AND INTERESTS   The most widely utilized scheme for characterizing personality traits is the Big Five— extraversion, agreeableness, conscientiousness, neuroticism, and openness. Loehlin (1992) organized all personality kinship data using this scheme and fit alternative models to the combined data. Because the DZ correlation was less than half the corresponding MZ correlation for each of the five personality dimensions, Loehlin reported parameter estimates both when the excess MZ twin similarity was attributed to a special MZ twin environmental effect and when it was attributed to epistasis. In either case there were appreciable genetic effects. In the first instance, narrow heritability was estimated to be .36, .28, .28, .31, and .46 (Mean = .34), and the common environmental component was estimated to be .00, .09, .04, .05, and .05 (Mean = .05) for the five personality dimensions, respectively. In the second instance, the broad heritability was estimated to be .49, .35, .38, .41,

and .45 (Mean = .42), and the common environmental effects were estimated to be .02, .11, .07, .07, and .06 (Mean = .04). Bouchard (1994) reported similar estimates of heritability (average of .41 for these five basic dimensions of personality) and shared environmental effects (average of .07) in a combined analysis of MISTRA reared apart twin correlations and correlations on reared together twins.

In contrast to the many behavioral genetic studies of normal personality, there are only a few studies of the personality correlates of psychopathology (reviewed by Nigg & Goldsmith 1994). Research in this domain is founded on the belief that, rather than representing distinct etiological entities, some behavioral disorders are best conceptualized as the extreme of normal variation. Livesley and associates (1993) administered a self-report measure of 18 dimensions underlying DSM-III-R personality disorder diagnoses to a nonpsychiatric twin sample and reported an average heritability estimate of .44. DiLalla and associates (1996) analyzed Minnesota Multiphasic Personality Inventory (MMPI) findings from MISTRA MZA and DZA twins and reported heritability estimates for the standard clinical scales that ranged from .26 to .61 and averaged .43. In a related domain, True and colleagues (1993) reported heritabilities from .13 to .34 for symptoms of posttraumatic stress in Vietnam era twins.

Occupational interests are usually organized according to Holland's (1985) six general occupational themes (GOTs): realistic, investigative, artistic, social, enterprising, and conventional. The results of early twin studies of interests were summarized by Nichols (1978), who reported an average (across studies and interest domains) difference in MZT and DZT correlations of .18 (implying an average narrow heritability of .36) that, except for investigative (where the implied heritability was .50), varied little across interest domain. In a study of reared apart twins, heritability estimates were .41, .66, .50, .52, .50, and .38 for the six GOTs, respectively (Moloney et al 1991). These estimates were higher than those reported by Nichols because of the greater precision with which the GOTs were assessed. Betsworth et al (1993) combined adoption and twin data from brief scales that could be scored from the different versions of the Strong Vocational Interest Inventory/Strong-Campbell Interest Inventory that had been used in several kinship studies. Heritability estimates for the six GOTs were .36, .36, .39, .38, .31, and .38 (Mean = .36), while estimates of shared environmental influences equaled .12, .10, .12, .08, .11, and .11 (Mean = .11). Multiple lines of evidence thus demonstrate that for occupational interests, genetic influences are slightly weaker and shared environmental influences are slightly stronger than for personality. With respect to a rather different aspect of psychological interest, twin and adoption studies in both males (Bailey & Pillard 1991) and females (Bailey et al 1993) suggest substantial genetic influence on human sexual orientation.

Eaves & Eysenck (1974) completed the first large scale twin study of social attitudes, and reported heritabilities of .65 and .54 for measures of radicalism and tough-mindedness. Scarr & Weinberg (1981) included a measure of authoritarianism in an adoption study on the expectation that it would show little heritability and a large shared environmental influence. Contrary to expectation, the measure of authoritarianism was substantially heritable, an effect the investigators attributed to the association of authoritarianism with verbal ability and personality. Others have also reported significant heritabilities for personality measures based on the authoritarianism construct (Horn et al 1976, Tellegen et al 1988). In a large twin study, Martin and associates (1986) reported a substantial average MZT correlation (.63) and a somewhat smaller average DZT correlation (.44) for the Wilson-Patterson Conservatism scale, which when modeled along with a rather large assortative mating coefficient (.68) yielded a heritability estimate of .62.

Genetic influences on measures of religious interests, attitudes, and values have also been explored. Using data on reared together and reared apart adult twins, Waller and associates (1990) reported a heritability estimate of .59 for religious leisure time interests and .41 for religious occupational interests; in both cases shared environmental influences were not statistically significant from zero. In a related domain, a number of studies have reported modest heritabilities for job satisfaction (approximately .35; Arvey et al 1994) and work values (approximately .25; Arvey et al 1994, Keller et al 1992).

PSYCHOPATHOLOGY   There have been numerous twin and adoption studies of behavioral disorders, so our summary of this literature is necessarily brief and relies heavily on recent reviews. Table 1 presents MZT and DZT concordances for major behavioral disorders. In compiling this table, we have attempted to identify comprehensive reviews or, when these are lacking, a single large representative study. When possible, we report the probandwise rather than the pairwise concordance rate. The MZT concordance is consistently and substantially larger than the DZT concordance for most behavioral disorders. Indeed, the difference in concordance for behavioral disorders is at least as great, if not greater, than the difference in concordance observed with many medical disorders (Plomin et al 1994b). Significantly, adoption studies of, for example, schizophrenia (Gottesman 1991), affective disorder (Wender et al 1986), criminality (Mednick et al 1984), alcoholism (McGue 1995), and hyperactivity (Morrison & Stewart 1973) support the inference of genetic influence made in twin studies of these disorders. Thus, genetic factors appear to play a substantial role in the etiology of most behavioral disorders. Nonetheless, the less than perfect MZT concordance that characterizes all behavioral disorders implicates the importance of environmental, specifically nonshared environmental, influences, a point we return to below.

Table 1   Monozygotic (MZ) and dizygotic (DZ) twin concordance (C) for behavioral disorders[a]

	MZ		DZ		
	C	N	C	N	Reference
Adult disorders					
Schizophrenia	.48	115	.17	184	Gottesman 1991[b]
Affective illness	.65	146	.14	278	Berrettini 1997[b]
Alcoholism - Male	.41	413	.22	617	McGue 1995[b]
Alcoholism - Female	.34	155	.31	154	McGue 1995[b]
Criminal conviction	.52	229	.23	316	Gottesman & Goldsmith 1994[b]
Panic disorder	.24	67	.11	55	Kendler et al 1993a[c]
Bulimia nervosa	.23	35	.09	23	Kendler et al 1991[c]
Childhood disorders					
Attention deficit/Hyperactivity	.58	69	.31	32	Sherman et al 1997[c]
Tourette syndrome	.53	30	.08	13	Price et al 1985[c]
Autism	.64	45	.09	36	Smalley et al 1988[b]
Juvenile delinquency	.91	55	.73	30	Gottesman & Goldsmith 1994[b]
Reading disorder	.68	186	.38	138	DeFries & Alarcón 1996[c]

[a]When possible probandwise rather than pairwise concordance is reported.
[b]Concordance rate reflects the weighted average of studies reviewed in citation.
[c]Concordance rate from a single study, no compilation available.

The Search for Behaviorally Relevant Genes

SCHIZOPHRENIA   Linkage studies have identified several chromosomal regions as candidates for containing a schizophrenia susceptibility locus. The strongest support is for 6p24–22, where at least four groups have reported positive linkage results (Antonarakis et al 1995, Moises et al 1995a, Schwab et al 1995, Straub et al 1995). Although others have failed to find linkage to schizophrenia in this region (Gurling et al 1995, Mowry et al 1995), these studies do not necessarily constitute a refutation, as only 15% to 30% of the schizophrenia families in the positive linkage studies were estimated to carry the vulnerability locus (Straub et al 1995). A second region of strong interest is 22q, where several groups have reported support for linkage (Coon et al 1994, Lasseter et al 1995, Moises et al 1995b, Vallada et al 1995), and a combined analysis yielded significant results implicating the 22q12 region (Gill et al 1996). The chromosome 22 findings are especially intriguing given the observation of significantly elevated rates of schizophrenia among individuals with velo-cardio-facial syndrome, a disorder associated with micro deletions within 22q11.2 (Lindsay et al 1995). Other regions for which there is some positive evidence for linkage include 8p (Moises et al 1995a, Pulver et al 1995) and 3p (Pulver et al 1995). At this time there are no strong candidate genes for schizophrenia within the regions identified by linkage studies. Studies associating schizophrenia with dopamine system polymorphisms have not proven productive, although at least two groups

have reported positive associations with a serotonin receptor polymorphism (Inayama et al 1996, Williams et al 1996).

MANIC-DEPRESSION   Manic-depressive illness (bipolar disorder) has been linked to 14 different chromosomal regions, none of which can be considered confirmed at this time (Risch & Botstein 1996). The evidence must be considered tentative even for chromosome 18, where three positive linkage studies have been published (Berrettini et al 1994, Freimer et al 1996, Stine et al 1995), as the linked markers span a region longer than 100 cM, including most of both arms of chromosome 18. Recent studies have failed to observe significant linkage to the X chromosome (Baron et al 1993), leaving open the status of one of the oldest hypotheses about linkage for a behavioral disorder. Linkages to 4p (Blackwood et al 1996) and 6p, 13q, and 15q (Ginns et al 1996), all of which await replication, provide additional regions of interest in future linkage studies of bipolar disorder.

Anticipation—decreasing age of onset or increasing severity with successive generations—has been observed with many of the disorders caused by an expanding trinucleotide repeat sequence (e.g. fragile X syndrome, Huntington's disease, myotonic dystrophy). Consequently, the observation in two sets of bipolar families of greater severity and an earlier age of onset (approximately 10 years on average) in the younger as compared to the older generation is of particular interest (McInnis et al 1993, Nylander et al 1994). Also of interest is the observation of excess maternal transmission of bipolar disorder in two separate studies (Gershon et al 1996, McMahon et al 1995). Excess maternal transmission may indicate mitochondrial transmission or imprinting (i.e. gene effect depending on sex of transmitting parent).

ALCOHOLISM   Most attempts to identify single genes contributing to risk of alcoholism have used the association method. Two genetic systems have been implicated. The first involves polymorphisms for the liver enzymes principally involved in the metabolism of alcohol, aldehyde dehydrogenase (ALDH) and alcohol dehydrogenase (ADH). Approximately 50% of individuals of East Asian ancestry inherit inactivity of the mitochondrial form of ALDH (Harada et al 1982). This inactivity produces a heightened sensitivity to the toxic effects of alcohol and is thus a protective factor against alcoholism (Agarwal & Goedde 1989). Two of the six genes for human ADH are polymorphic, and in both cases the high activity form of ADH has been associated with lower rates of alcoholism, again in East Asiatic populations (Thomasson et al 1991).

The report by Blum and colleagues (1990) of an association between the *A1* allele at a Taq1 restriction site near the D2 dopamine receptor (*DRD2*) locus on chromosome 11 set off a flurry of attempts at replication and a puzzling

array of seemingly inconsistent findings. A meta-analysis of published *DRD2* association studies reported an aggregate frequency for the *A1* allele of .21 in alcoholics, .20 in controls not screened for alcoholism, and .12 in controls screened for alcoholism; these results were interpreted by Gelernter and associates (1993) as indicating a lack of significant association between *DRD2* and alcoholism. Alternatively, Neiswanger and associates (1995) have argued that the low frequency of *A1* in controls screened for alcoholism (and for other psychiatric disorders) suggests that absence of *A1* may be a marker for good psychiatric health rather than presence of *A1* being a specific marker for alcoholism. In any case, the Taq1 site is downstream from the *DRD2* coding region, and investigators have failed to find a functional mutation in the *DRD2* gene associated with alcoholism (Gejman et al 1994). At this time, the relevance of *DRD2* to alcoholism risk remains at best uncertain.

OTHER CHARACTERISTICS   Systematic attempts at gene identification have been made with only a few other behavioral phenotypes. A genome-wide linkage study of Tourette syndrome (TS) using more than 600 markers has produced no strong evidence for linkage (Heutnik et al 1993). Comings and associates (1996), however, reported significant associations between features of TS and markers for three dopaminergic genes: DRD2, dopamine beta hydroxylase, and the dopamine transporter gene (*DAT1*). Attention-deficit disorder has also been associated with *DAT1* (Cook et al 1995), as well as with a genetic mutation leading to generalized resistance to thyroid hormone (Hauser et al 1993). Brunner et al (1993) reported that a nonsense mutation of the X-linked monoamine oxidase A gene cosegregated with borderline mental retardation and impulsive aggression in a Dutch pedigree. Targeting the HLA region of chromosome 6 because of reported associations between dyslexia and autoimmune disorders, Cardon and associates (1994) reported significant linkage of reading disability (dyslexia) to 6p21.3 in two independent samples. Grigorenko and colleagues (1997) recently replicated the linkage of dyslexia to 6p and also reported suggestive linkage to a second locus on 15p. Sexual orientation in males, but not females, has been linked to Xq28 in two independent samples (Hamer et al 1993, Hu et al 1995). The X chromosome has also been implicated in a single linkage study of autism (Hallmayer et al 1996).

PERSONALITY AND COGNITIVE ABILITY   Genes influencing normal variation in personality and cognitive ability have been investigated using the candidate gene approach. Lesch and colleagues (1996) report a significant association between the personality trait of neuroticism and variation in a regulatory region of the serotonin transporter gene, and variation in an exonic region of the D4 dopamine receptor locus has been associated with the personality trait of novelty

seeking in two studies (Benjamin et al 1996, Ebstein et al 1996), although a third study failed to find a similar association (Malhotra et al 1996). Plomin and colleagues (1994a, 1995) sought to identify genes contributing to variability in IQ by contrasting marker allele frequency in high and low IQ individuals. One hundred DNA markers were selected for study because they are in or near genes of presumed neurological significance. Significant associations were observed for 5 of the 100 markers, but in only one case, a mitochondrial DNA marker (Skuder et al 1995), did the high versus low IQ frequency difference replicate in a planned independent sample.

THE NATURE OF ENVIRONMENTAL INFLUENCE

Shared Versus Nonshared Environmental Influences

Whereas the dominant theoretical and empirical traditions within developmental psychology have emphasized the influence of shared rather than nonshared environmental factors, behavioral genetic research is consistent in showing that environmental influences on most psychological traits are of the nonshared rather than the shared variety (Plomin & Daniels 1987).

For personality characteristics, the MZT correlation has consistently exceeded the corresponding DZT correlation by more than a factor of two. This observation, first noted by Loehlin & Nichols (1976) but replicated in diverse cultures with thousands of twin pairs (Loehlin 1992), implies a shared environmental component of zero. Alternatively, the consistently high ratio of MZT to DZT correlation could reflect genetic nonadditivity, or greater environmental sharing among MZT as compared with DZT twins. It is thus significant that findings from studies of reared together twins have been replicated using alternative research designs. For example, MZTs are not markedly more similar in personality than MZAs. In the four published studies comparing the similarity of MZA and MZT twins on the two most fundamental dimensions of personality, extraversion and neuroticism, the weighted average MZA correlation is .39 for both factors (summarized by Loehlin 1992). The comparable MZT averages are .56 for extraversion and .46 for neuroticism. Secondly, the correlation for nonbiologically related but reared together sibling pairs (i.e. adoptive siblings) provides a direct estimate of the effect of common rearing; in three adoption studies of adults (summarized by Loehlin 1992), the weighted average adoptive sibling correlation was −.07 for extraversion and .09 for neuroticism, while in a single adoption study of adolescents, the adoptive sibling correlation was −.04 for a measure of extraversion and .00 for a measure of neuroticism (McGue et al 1996).

The minimal effect of common rearing appears to hold not only for personality factors but also for most major forms of psychopathology. Adoption studies

of, for example, schizophrenia (Gottesman 1991) and alcoholism (McGue 1995) indicate that risk to the biological offspring of an affected parent is independent of whether the offspring is reared by the affected parent, while twin studies of most behavioral disorders reveal a greater than 2:1 ratio of MZT to DZT concordance (see above). There are, however, two noteworthy exceptions to the general finding of little shared environmental influence on behavioral characteristics: cognitive ability and juvenile delinquency. From a compilation of familial IQ correlations (Bouchard & McGue 1981), the following observations all support the existence of substantial shared environmental influences on general cognitive ability: (*a*) the average MZT IQ correlation (.86) is less than double the corresponding average DZT correlation (.60); (*b*) the average MZA correlation (.72) is moderately lower than the average MZT correlation; and (*c*) the average adoptive sibling correlation (.32) is substantial. Taken together, these observations suggest that from 20% to 30% of the variance in IQ is associated with shared environmental effects (Chipuer et al 1990).

The overwhelming majority of the twin and adoptive sibling correlations for IQ are based on preadult samples, for which the effect of shared environmental factors may be maximal. As noted above, when twin IQ correlations are categorized according to the age of the twin sample (McGue et al 1993), the ratio of MZT to DZT correlation is found to increase with age such that in adult samples the average MZT correlation (.83) exceeds the average DZT correlation (.39) by more than a factor of two, suggesting no shared environmental influence at this life stage. Moreover, the average adoptive sibling IQ correlation equals .32 in studies of children or adolescents (summarized in Bouchard 1997a), but, as already noted, only .04 in studies of adults. The adoptive sibling correlation decreased with age in each of the three of these studies that involved longitudinal assessment of IQ. Shared environmental influences on IQ, although substantial in childhood, appear to decrease markedly in adulthood.

The pooled concordance rates for male juvenile delinquency are high and similar for MZT (91%) and DZT (73%), suggesting a substantial influence of shared environmental factors (Gottesman & Goldsmith 1994). Similarly, twin correlations for delinquency assessed quantitatively (e.g. as number of delinquent acts) rather than categorically find evidence for strong shared environmental effects (Rowe 1994, Silberg et al 1996). Like IQ, the influence of shared environmental factors on adolescent antisocial behavior may diminish in adulthood. In a sample of more than 3000 US veteran twin pairs, Lyons and colleagues (1995) reported that the heritability of antisocial behavior increased from .07 in adolescence to .43 in adulthood, while the proportion of variance associated with shared environmental effects decreased from .31 in adolescence to .05 in adulthood. The matter is not fully resolved, however, as a subsequent investigation of more than 2500 Australian twins (Slutske et al

1997) reported significant heritability (.71) and no shared environmental effect for retrospectively assessed adolescent conduct disorder.

The Heritability of Experience

The failure of behavioral geneticists to find much evidence of shared environmental influences appears inconsistent with an extensive empirical literature in developmental psychology demonstrating a strong association between rearing circumstances and psychological outcomes. For example, individuals with high IQs tend to have been reared in homes that were intellectually stimulating, aggressive individuals tend to have been reared by parents who were both punitive and arbitrary, and alcohol abusers tend to have been reared by parents with marital problems who used ineffective child-rearing methods. This inconsistency can be resolved by recognizing that environmental measures may reflect the influence of genetic factors (Plomin 1994).

Rowe (1983) reported that adolescent MZT rated their rearing homes more similarly than adolescent DZT in warmth ($r = .63$ versus .21) but not permissiveness ($r = .44$ versus .54). This finding of greater genetic influence on ratings of parental warmth than on ratings of control has been replicated in studies of reared together and reared apart adult twins who retrospectively rated their rearing homes (Hur & Bouchard 1995, Plomin et al 1988) and in a study of 707 sibling pairs that included MZT and DZT twins as well as full, half, and nonbiologically related siblings (Plomin et al 1994c). Significantly, similar heritable effects are observed when aspects of the parent-offspring relationship are assessed directly through observational studies as well as indirectly through self-ratings (O'Connor et al 1995, Rende et al 1992). Moreover, the level of intellectual stimulation in the home (Braungart et al 1992), parental marital discord (McGue & Lykken 1992), exposure to psychological stress and trauma (Kendler et al 1993b, Lyons et al 1993, Plomin et al 1990c), and access to support in one's social network (Bergeman et al 1990, Kessler et al 1992) all appear from twin studies to be partially heritable.

The heritable nature of environmental exposure implicates genotype-environment correlational processes and the mechanisms by which genes and environments jointly influence the development of phenotypes (considered below); it also has significant implications for the methods psychologists use to identify environmental risk. The dominant paradigm within psychology for identifying environmental risk has involved the study of intact nuclear families, in which case an association between parental behaviors and offspring outcomes is characteristically interpreted as reflecting environmental mechanisms. Any association, however, may also reflect genetic mechanisms. Indeed, when the genetic basis of parent-offspring resemblance is controlled by studying adoptive families, the association between child-rearing strategies and offspring behavior (McGue et al 1996) and the relationship between home characteristics and

intellectual achievement (Scarr 1997) are nearly eliminated. Behavioral genetic research on the minimal effect of shared environmental factors and the heritability of experience challenges the validity of a vast amount of psychological research aimed at identifying environmental risk.

The Nature of Nonshared Environmental Influences

The finding that nonshared factors constitute the major source of environmental variation for many psychological characteristics has led to several systematic attempts, with limited success, to identify specific nonshared effects. Differential parental treatment is one potential source of nonshared environmental influence. Although there is a strong tendency for parents to treat their multiple children similarly, parents do sometimes treat multiple offspring differently, especially in the domains of parental negativity and parent-offspring conflict (Dunn et al 1990). This differential parental treatment does appear to contribute to nonshared environmental variance, although the overall magnitude of this contribution appears to be small. In a study of twins and nontwin siblings, Reiss and associates (1995) found that 60% of the variance in adolescent antisocial behavior and 37% of the variance in adolescent depressive symptoms could be predicted by negative and conflictual parental behavior directed specifically at the adolescent. In a follow-up analysis, however, Pike and associates (1996) reported that most of the association between parental behavior and adolescent outcomes was genetically mediated and that differential parental treatment accounted for a small proportion of the nonshared environmental effect (2% to 10%).

A major class of nonshared environmental factors that does appear to exert a substantial influence on some psychological characteristics is pre- and perinatal factors. Obstetrical complications and prenatal exposures have been consistently associated with risk of major psychopathology (Gottesman 1991), and criminal behavior and violence (Raine et al 1994). In an investigation notable for its novel use of a behavioral genetic design, Torey and colleagues (1994) studied 27 pairs of MZT twins discordant for schizophrenia in an attempt to identify the nonshared environmental factors contributing to this disorder. They found that obstetrical complications contributed to the disorder in 30% of cases and that approximately 30% of the schizophrenic twins had early central nervous system dysfunction. Wolf and colleagues (1996) used the same approach to determine that differences in D2 dopamine receptor binding in the caudate nucleus strongly predicted MZT discordance for TS. Prenatal stress and exposure has also been associated with diminished cognitive functioning (Neisser et al 1996). The importance of these factors within the normal range of personality and cognitive ability, however, remains largely unexplored.

Structural variables such as birth order and spacing, and relationship variables such as sibling and peer influences are other potential sources of nonshared environmental influence that have yet to be explored fully from this perspective

[see, however, Sulloway (1995) for a provocative treatment of birth order effects on personality]. Alternatively, the nonshared environmental component may defy easy identification, as it includes errors of measurement due to temporal instability, and may reflect either the aggregate effect of many microenvironmental events (Jensen 1997) or random and largely idiosyncratic early biological factors that can influence individual developmental course (Molenar et al 1993).

MODELS FOR THE JOINT INFLUENCE OF GENES AND THE ENVIRONMENT

Genotype-Environment Correlation

Genotype-environment correlation, the nonrandom assortment of genotypes across environments, can arise through one of three mechanisms (Scarr & McCartney 1983). Passive genotype-environment correlation occurs when parents, who transmit to their offspring genes that might promote the development of a psychological characteristic, also provide a rearing environment that encourages the development of that characteristic. Passive genotype-environment correlations have been observed for cognitive ability, at least during childhood where high IQ parents both transmit genes that promote intellectual achievement and also tend to provide an intellectually stimulating rearing environment (Loehlin 1989), and, to a far lesser degree for personality, where parents who are high in extraversion and low in neuroticism tend to have homes that are rated as warm and nurturant (Chipuer et al 1993).

Evocative genotype-environment correlation occurs because an individual's experiences are in part a function of the reactions his or her genetically influenced behavior can elicit from others. Lytton (1990) has shown, for example, that the ineffective child-rearing strategies used by parents of conduct-disordered boys is in large part a reaction to, rather than simply a cause of, the child's defiant behavior. Pike and colleagues (1996) reported that much of the association between parental negativity and both adolescent antisocial behavior and depression was genetically mediated, again implicating evocative genotype-environment correlational processes. Of interest is whether these evocative processes produce reciprocal effects. In a small but important adoption study, Ge and colleagues (1996) found a significant association between biological parents' psychiatric status and adoptive parents' child-rearing behavior. This genotype-environment association was mediated largely by adoptee antisociality and, in the case of the mother, appeared to owe to reciprocal effects, such that the adoptee's antisocial behavior led to the mother's harsh and inconsistent parenting, which in turn exacerbated the adoptee's level of antisociality.

Active genotype-environment correlation occurs when individuals' inherited dispositions affect their life choices. Some individuals spend inordinate amounts of time viewing television, while others prefer to spend their time more actively engaged. Some young men volunteered for duty in Southeast Asia during the Vietnam era, while others did all they could to avoid combat. Some individuals pursue higher education, while others end their education earlier. Each of these life choices is likely to have both immediate and long-term effects on the nature of individual experience; each also appears to be partially heritable (Lyons et al 1993, Plomin et al 1990a, Plomin 1994, respectively), presumably because these life choices are influenced by heritable dimensions of personality and ability. The existence of genotype-environment correlations, and in particular reactive and active processes, serve to distinguish the meaning of heritability for some psychological traits from the meaning of heritability for medical or physiological traits. For traits like social attitudes, interests, and even antisocial behavior, the social environment is likely an important mediating step between primary gene product and behavior. As Rose (1995, p. 648) has stated, "We inherit dispositions, not destinies. Life outcomes are consequences of lifetimes of behavior choices. The choices are guided by our dispositional tendencies, and the tendencies find expression within environmental opportunities that we actively create." The heritability of psychological function does not imply the genetic determinism of human behavior.

Genotype-Environment Interaction

The existence of genotype-environment interaction ($G \times E$, or differential sensitivity of genotypes to environments) for psychological characteristics, although intuitively plausible, has been difficult to demonstrate empirically. Attempts to identify $G \times E$ effects for personality (Bergeman et al 1988) and general cognitive ability (Capron & Duyme 1989) did not yield significant findings. Moreover, a significant $G \times E$ for antisocial behavior (Cadoret et al 1995) has not been observed in other similar studies (Mednick et al 1984, Willerman et al 1992), while a $G \times E$ for alcohol abuse (Cloninger et al 1981) has been difficult for others to replicate. Failure to observe replicable $G \times E$s may mean that our intuitions are wrong and that the world is largely additive. Alternatively, and we think more plausibly, our methods may presently lack the precision to detect the existence of $G \times E$ effects. Wahlsten (1990) has argued that large samples are needed before $G \times E$ effects can be detected. More importantly, $G \times E$s may only exist at the extremes of environmental and genetic variation, and may be detectable only when both the genotype and environment can be accurately assessed. The classic example of a $G \times E$ for a behavioral trait, phenylketonuria, was detected only after both the obligatory genotype and the

obligatory environment were both identified. Current methods for the detection of G × E in human behavioral genetics are, however, largely indirect. One of the most significant consequences of gene identification for behavioral traits may be that it will provide human behavioral geneticists with the tools needed to systematically investigate the G × E effects many believe to exist.

CONCLUSION

Twin and adoption studies of diverse psychological and psychopathological characteristics are consistent in implicating the influence of genetic factors on individual differences in behavior. Heritability estimates from twin and adoption studies of variables such as IQ, alcoholism, personality traits, and even social attitudes are at times quite substantial. Nonetheless, despite some effort, there are a limited number of confirmed linkages or gene associations for behavioral traits. There are, however, many promising leads. The failure to identify the genes underlying specific human behavioral phenotypes may indicate that we have been misled by the twin and adoption study findings. Alternatively, and we believe more plausibly, the current failure may simply reflect the difficulty of gene identification with complex and heterogeneous phenotypes. Additional molecular genetic research should provide the necessary observations to resolve these two possibilities.

Behavioral genetic research is also consistent in indicating that nonshared rather than shared environmental factors constitute the major source of environmental influence on behavior. The adoptive sibling correlation provides a direct estimate of shared environmental influences, and for many psychological traits this correlation is near zero, especially when assessed in adulthood. Despite their apparent importance, little progress has been made in identifying the specific nonshared factors that contribute to individual differences in behavior; a failure that may reflect the random, idiosyncratic, and micro nature of nonshared environmental effects.

Genotype-environment correlational and interaction processes serve to distinguish behavioral phenotypes from medical or physiological phenotypes. Genotype-environment correlational processes have been observed with some behavioral traits, and the existence of these processes serves to illustrate how genetic influences on some aspects of behavior can be mediated by the social environment. Genotype-environment interactions for human behavioral traits, although hypothesized to be extensive, have been difficult to detect empirically. The future success of gene identification efforts should address current methodological limitations in efforts to identify gene-environment interactions. The field of human behavioral genetics may be poised on the threshold of an era where the identification of behaviorally relevant genes using molecular genetic

methods leads to greater insight into not only the genetic, but also the environmental basis of human behavioral differences.

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