# Genetic and Environmental Influences on Human Psychological Differences

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**ABSTRACT:** Psychological researchers typically distinguish five major domains of individual differences in human behavior: cognitive abilities, personality, social attitudes, psychological interests, and psychopathology (Lubinski, 2000). In this article we: discuss a number of methodological errors commonly found in research on human individual differences; introduce a broad framework for interpreting findings from contemporary behavioral genetic studies; briefly outline the basic quantitative methods used in human behavioral genetic research; review the major criticisms of behavior genetic designs, with particular emphasis on the twin and adoption methods; describe the major or dominant theoretical scheme in each domain; and review behavioral genetic findings in all five domains. We conclude that there is now strong evidence that virtually all individual psychological differences, when reliably measured, are moderately to substantially heritable. © 2003 Wiley Periodicals, Inc. J Neurobiol 54: 4–45, 2003

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## INTRODUCTION—WHY STUDY THE RELATIVE INFLUENCE OF HEREDITY AND ENVIRONMENT ON PSYCHOLOGICAL TRAITS?

The debate over the relative influence of heredity and environment on individual differences in human psychological traits may appear interminable. Appearances can, however, be deceiving. Considerably more is known today about the role of genetic and environmental influences on human behavior than was known only a few years ago. Research in human behavioral genetics has diversified so greatly (c.f., Rose, 1995; Plomin and Crabbe, 2000) that it is no longer possible to cover the entire field in a single review. In this article we focus primarily on quantitative genetic and environmental studies of what might be called "global phenotypes", traits like cognitive ability and personality, which have broad implications for understanding human behavior in diverse settings, as opposed to more narrowly specified characteristics such as the conditioned eye blink response. The distinction is admittedly artificial, and the level at which an investigator works primarily reflects his or her theoretical orientation. Regardless of level, however, researchers strive to reduce the phenomena of study to more basic processes. Thus, for example, the heritable basis and neural circuitry of the eye blink response (Merrill et al., 1999; Bao et al., 2002) have largely been worked out, and *Drosophila* courtship behavior can be effectively decomposed into a sequence of individual behaviors that lead to copulation (Sokolowski, 2001).

Even frankly social phenotypes can benefit from the reductionist's paradigm. Divorce, for example, is a trait that appears to be moderately heritable (McGue and Lykken, 1992), an effect that appears to be partially mediated by inherited personality factors (Jockin et al., 1996), which in turn have a biochemical basis (Zuckerman, 1995). Thus, although we can reject the notion that divorce is predestined in our DNA code, we recognize that genes may influence an individual's likelihood of divorce indirectly by affecting intermediate biochemical and personality systems. Sometimes what appears to be precisely the opposite argument is made. That is, some would argue that global behavioral phenotypes (such as IQ) need no longer be subjected to quantitative genetic analysis because we already know genes are involved and we should focus at the single gene level (Wahlsten, 1999). Some critics argue that heritability studies are uninformative because heritability is a population sta-

tistic that can vary dramatically from one place to another. Of course, we can determine the degree to which this claim is true only by carrying out a wide range of studies on various populations. There is no rational stopping rule; the world—physical, biological, and social—is a seamless whole and the task of science is to work out the links at every level, not declare domains "off limits".

The studies we review were designed to explore both environmental and genetic influences on individual differences in behavior. Behavior genetic methods are unbiased with regard to whether genetic or environmental sources of variance are more important. If there is no genetic source of variance the methods will reveal this fact. The failure to apply behavior genetic methodology more widely has seriously handicapped the psychological sciences by limiting the types of inquiries and classes of acceptable explanation for various phenomena. Genetic and evolutionary hypotheses have been assumed away rather than subjected to empirical examination. A final reason for studying genetic influence on human behavior is the fact that genetic variability has major implications for our understanding of ourselves and the human world. As Bouchard et al. (1990a) put it: "A human species whose members did not vary genetically with respect to significant cognitive and motivational attributes, and who were uniformly average by current standards, would have created a very different society than the one we know" (p. 228).

## Methodological Cautions

When discussing research on human individual differences there are a number of conceptual/methodological errors that occur with regularity, that consistently muddle thinking about important issues and therefore require discussion.

1. **Correlations between Biological Relatives (i.e., IQ Correlations between Siblings or Parents and Offspring) Reared Together Are Etiologically Ambiguous.** Behavior geneticists are quick to point out that "familial does not equate to genetic" (Martin et al., 1997, p 387). Unfortunately, social science researchers are not as quick to recognize that the complement is also true, that "familial does not equate to environmental". Parent-offspring correlations for psychological traits (and most other traits as well) when gathered on ordinary biological families are completely confounded, reflecting both the influence of genetic and environmental factors. This simple fact has been known for many years and was first put into scientific form by Galton (1869/1914), who suggested the adoption design as a means of circumventing the

problem. That this problem, well known to behavior geneticists, is still not fully appreciated by many psychologists is nicely illustrated by the furor over the recent book by Judith Harris (1998)—*The Nurture Assumption*. Harris, a developmental psychology textbook writer, points out that for years she and most authors of developmental psychology text books, as well as most researchers in the domain, made the assumption that nurture was the predominant source of the similarity between parents and offspring and did not subject the assumption to empirical scrutiny.

2. **The Correlations between Specific Measures of Parental Treatment (Child Rearing Practices) and Their Children's Characteristics (Personality) Are Uninterpretable When Based on Biological Relatives Reared Together.** This second caution is a corollary of the first, namely that the correlations between parental behavior and offspring characteristics are also completely confounded and uninterpretable when based on biological relatives reared together. Harris explains the problem using the socialization research of Baumrind (1967, 1971, 1989; Baumrind and Black, 1967) as an example. Many socialization researchers refuse to accept the need for adoption (or comparable) controls (Hoffman, 1991; Baumrind, 1993) on the grounds that the behavior genetic studies showing sizeable genetic influence on numerous psychology traits are not persuasive due to serious flaws in the designs and unacceptable assumptions. We hope to persuade the reader that these investigators are mistaken in their failure to use appropriate controls even if there are problems with behavior genetic methods. There are no infallible methods. The best protection against erroneous inferences is corroboration across methods that make different assumptions and have different strengths and weaknesses.

This caution is not as trivial as it sounds and the error is not rare. It is committed by even quite sophisticated researchers. For example, Kagan (1998), in an explicit critique of Harris' *The Nurture Assumption* titled "A Parent's Influence Is Peerless", says: "Consider, for example, that the best predictor of a child's verbal talent is the frequency with which the parents talk with and read to the child. A verbally talented child is more likely to get better grades in school and, therefore, a little more likely to attend a better college. That, in turn, makes it more likely that in adulthood he or she will land a better job". The U.S. News and World Report cites Kagan as saying, "That fact alone is enough to discredit her thesis" (Leo, 1998, p. 14). Kagan's argument is based on the study by Hart and Risley (1995), which is generally credited with establishing the influence of parental speech on children's

verbal ability. To simplify this study somewhat the authors followed 42 families (one child per family) over two and one-half years, making monthly hour long observations in each home. The primary dependent variable was vocabulary growth in the child and the primary independent variable was verbal production by the caregivers (recorded on audio tape). The investigators tell us that with regard to their measures children behave like their parents: "The size of the children's recorded vocabularies and their IQ scores were strongly associated with the size of their parent's recorded vocabularies ($r = .77$) and the parent's scores on a vocabulary pretest ($r = .70$). By the age of 34–36 months, the children were also very similar to the averages of their parents" (p. 176). What is unrecognized in this interpretation, however, is the possibility that bright parents provide their children not only with a verbally stimulating environment but also a genetic potential for intellectual achievement. Without unconfounding the two sources of parental influence we are unable to unambiguously infer the existence of either effect. The number of additional examples of this problem, such as correlations between physical facilities in a child's home (i.e., number of books, etc.; cf. Longstreth et al., 1981) and his/her IQ, is too numerous to discuss here. Plomin (1994) provides a comprehensive treatment of this problem, pointing out that such environmental measures themselves are often somewhat heritable. Fortunately, progress is being made and recent studies are beginning to carefully constrain their interpretations of correlational findings. A recent study of "Childhood Parenting Experiences and Adult Creativity" (Koestner et al., 1999), for example, explicitly acknowledges the possibility of a genetic interpretation for their finding of a within-family correlation between parenting and offspring creativity.

## Frame of Reference for Interpreting Human Behavior Genetic Findings

It is widely believed that the sole purpose of human behavior genetics is to estimate heritability. We hope we have already made it clear that behavior genetic designs are needed to understand environmental influence as well. Indeed, the complement of heritability, "environmentality", indexes the contribution of environment to phenotypic variation. Bouchard and Loehlin (2001) recently laid out a series of epidemiological questions regarding sources of population variance in psychological traits that can be used to guide behavior genetic research. The list is given in Table 1. The list is not exhaustive, multivariate genetic research is not even discussed, but it is compre-

**Table 1    Major Epidemiological Questions Regarding Sources of Population Variance in Psychological Traits**

A. Environmental sources of variance
  1. To what extent is the trait influenced by environmental factors?
  2. What kind of environmental action is involved?
    a. Is it prenatal, nutritional, or hormonal?
    b. Is it postnatal, physical, or social?
    c. Is it shared or idiosyncratic?
    d. Are there maternal and/or paternal effects?
    e. Are there sibling interaction effects?
  3. Are there gender effects?
  4. Is transmission horizontal (e.g., among peers) and/or vertical (e.g., parent to child)?
B. Genetic sources of variance
  1. To what extent is the trait influenced by genetic factors?
  2. What kind of gene action is involved?
    a. Additive?
    b. Dominant?
    c. Epistatic?
  3. How many loci are involved?
  4. Is there sex-limitation or sex-linkage?
  5. Are chromosomal effects involved?
C. To what extent is the variation due to chance, or to chaotic processes approximating chance?
D. Joint genetic and environmental influences
  1. Are there any genetic × environmental interactions?
  2. Are there gene-environment correlations?
    a. Passive?
    b. Evocative?
    c. Active?
E. Developmental influences including aging
  1. Do different genes come into play during development?
  2. Do different environmental influences come into play during development?
  3. Does the variance due to various categories of influence change over time?
F. Assortative mating
  1. Is assortative mating, if present, due to active phenotypic assortment or social homogamy?
  2. Are there sex differences in mate preference for the trait?
G. Selection
  1. What sort of selective factors were at work during the original evolution of the trait?
  2. Are there current selective factors at work?
  3. Is the trait an adaptation?

hensive enough to illustrate that heritability estimates are only the first step in the quantitative behavior genetic research program. An examination of the research literature in light of the questions posed in the table makes it clear that for most psychological traits few of these questions have ever been investigated.

## Quantitative Behavior Genetic Methods—Interpreting Kinship Correlations

Many of the questions in Table 1 are studied using standard behavioral genetic methods involving kin correlations and covariances. These methods typically assume that the variance in a quantitative phenotype ($V_P$) can be decomposed into an additive function of genetic effects ($V_G$), shared environmental effects ($V_C$), and nonshared environmental effects ($V_E$), or
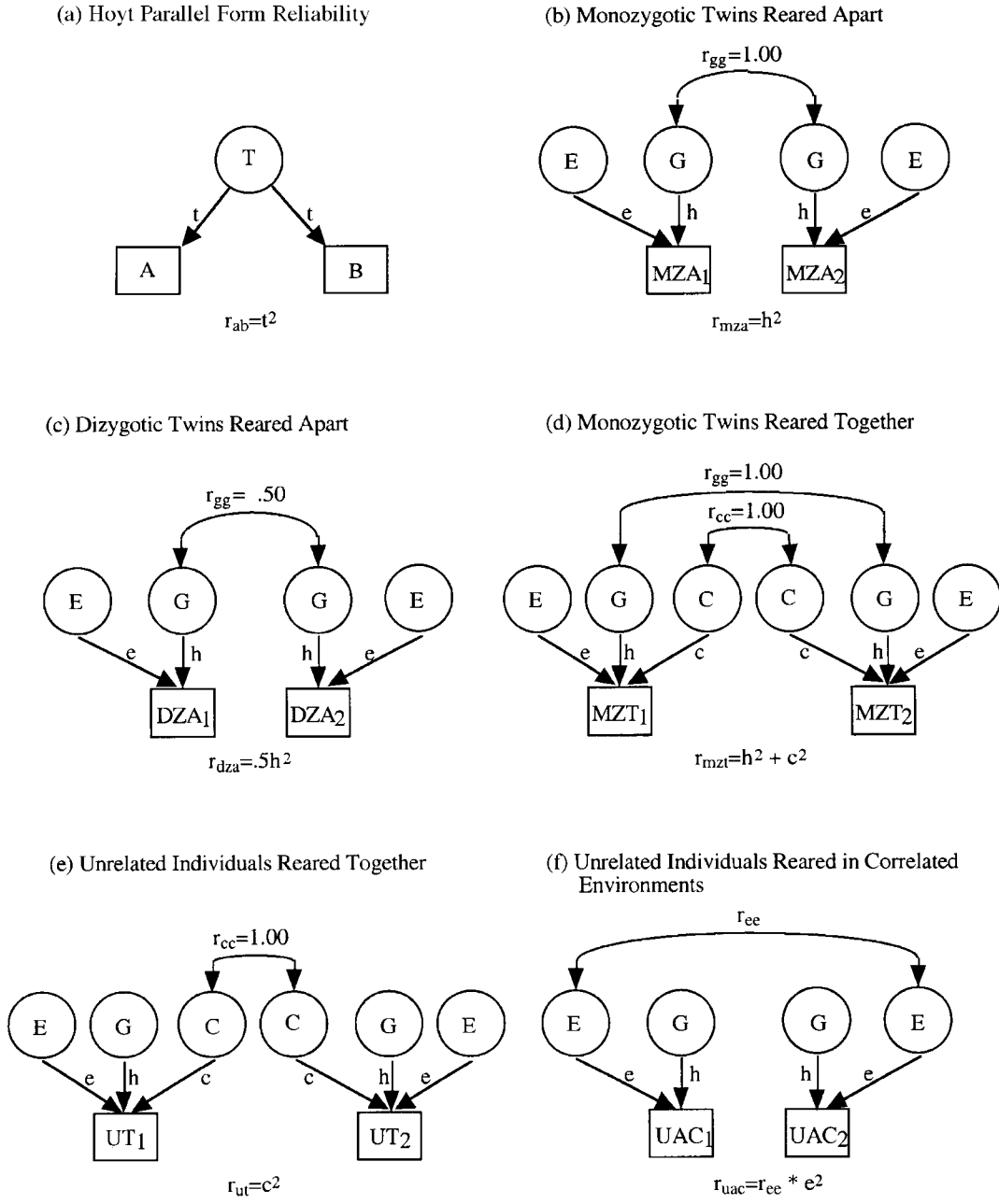
$$V_P = V_G + V_C + V_E \qquad (2)$$

Shared environmental effects refer to those environmental factors that are shared by reared-together relatives (e.g., parenting practices, parental income) and are thus a source of their phenotypic similarity, while nonshared environmental effects refer to those environmental factors that are not shared by reared-together relatives (e.g., peer group, accidents) and are thus a source of their phenotypic differences.

A standard injunction taught in introductory statistics courses is that correlation does not allow one to infer causation. This injunction is based on the fact that correlations are most often based on nonexperimental observations and any observed relationship could arise for a large number of reasons. Indeed, such correlations do not even allow an inference regarding direction of causation without additional knowledge such as temporal sequencing of events. Correlations based on experiments, such as a study that examines the relationship between drug dosage level and illness response level (or some indicator of illness such as number of white blood cells), are, however, regularly used to infer causation. The reason cause can be inferred in the latter case is because the manipulation (dosage and sequence of treatment, measurement, and randomization) was controlled by the investigator. Quantitative behavior genetic studies make use of the same logic. Genes come in various dosages (twins, parent $\times$ offspring, etc.) as do environments (reared together, reared apart). The former are experiments of nature and the latter are experiments of society. Figure 1 illustrates how these experiments can be utilized to draw inferences about genetic and environmental contributions to phenotypic variance.

Figure 1(a) shows a simple standardized path diagram widely used in psychology. It illustrates the correlations between two parallel psychological tests [technically a Hoyt (1941) reliability]. The paths (the $t$'s) tell us that psychologists understand that the correlation between test A and test B (measured phenotypes are shown in boxes) is due to a latent psychological construct (unmeasured latent constructs are shown in circles) called the individual's "true score". The true scores of the individuals who took the tests are the "cause" of the correlation between them (for unstandardized paths we would be speaking about covariances instead of correlations). The rules of path analysis specify that the correlation between the phenotypes is computed by multiplying the paths that link them. The correlation is thus ($t \times t$) or $t^2$ and indexes a squared value (a variance) called, in this instance, the true score variance. The idea that correlations need to be squared in order to account for variance (in which case they are called coefficients of determination) is widely taught and generally correct. It is not, however, always correct as this example and the ones to follow (kinship correlations) should make clear. Figure 1(b) is a simple generalization of the Hoyt reliability. Monozygotic twins (MZ twins, often misleadingly called identical twins) are known, from genetic theory and empirical study, to have identical or nearly identical genotypes (for the exceptions see Machin, 1996; Gringras and Chen, 2001), thus their genotypes are shown as being correlated at the value of 1.00. This could also be shown as a single G in a circle. The $h$ path (source of genetic influence on the phenotype) links each individual in a pair to the genotype, and because these MZ twins are reared apart (MZA twins) this is the only link between them. Thus the correlation is ($h \times 1.00 \times h$) or $h^2$. $h^2$ is the classic symbol for heritability or variance accounted for by genetic factors. MZ twins share all genetic factors that influence a phenotype including both additive and nonadditive factors. Additive factors are transmitted directly from generation to generation (often called breeding values) whereas nonadditive factors (dominance and epistasis) are not. Consequently, it is important to distinguish between broad heritability, $h^2_b$, which contains nonadditive genetic factors, and narrow heritability, $h^2_n$, which does not. The MZA correlation reflects all genetic factors and is thus a measure of $h^2_b$. Figure 1(c) illustrates the correlations between dizygotic twins reared apart (DZA twins, often misleadingly called fraternal twins). Because DZ twins share one-half of their genes identical by descent the genotype is connected by a value of .5. This figure assumes all additive genetic effects. Nonadditive genetic effects would not all be shared by DZ twins thus reducing the genetic similarity between DZ twins. This could be illustrated by dividing the latent genetic factor into two parts, an additive component (A) correlated .5 and a nonadditive component (D) correlated .25. The models discussed below easily incorporate such complexities. Figure 1(d) illustrates

(a) Hoyt Parallel Form Reliability

(b) Monozygotic Twins Reared Apart



(c) Dizygotic Twins Reared Apart

(d) Monozygotic Twins Reared Together



(e) Unrelated Individuals Reared Together

(f) Unrelated Individuals Reared in Correlated Environments



**Figure 1**    Path diagrams for (a) reliability, (b) monozygotic twins reared apart, (c) dizygotic twins reared apart, (d) monozygotic twins reared together, (e) unrelated individuals reared together, (f) unrelated individuals reared in correlated environments.

the correlation between MZ twins reared together (MZT twins). In this model we have added an additional latent trait to represent influences that make the twins similar due to their being reared in the same family, namely C (for common or shared environment). Obviously these models can be generalized to any kinship of any size.

Contemporary researchers fit models to observed kinship correlations using standard computer pro-grams (Mx, LISREL, etc.). The models incorporate specific effects of the sort listed in Table 1 that are of theoretical interest. Such models make a variety of assumptions, some of which are testable and others of which are not. Generally, the more kinships, the larger the sample, the better the measures, and the better the sampling of genotypes and environments, the less dependent the results will be on the underlying assumptions. Many worked examples can be found in

Neale and Cardon (1992), and model fitting in general is discussed in Loehlin (1998). This variance-covariance approach can also be applied to the search for quantitative trait loci (QTL's) (Neale, 1997; Blangero et al., 2000).

## Some Caveats on Twin and Adoption Methods

Twin and adoption studies have been widely used in behavioral genetics to resolve the separate contributions of genetic and environmental factors to phenotypic variation. The twin method has been both praised as "the perfect natural experiment" (Martin et al., 1997) and attacked as largely worthless (Layzer, 1976). It is, of course, neither. The adoption study has also been characterized as an ideal method for separating genetic and environmental influences, even though it also has some important limitations. Research designs that do not involve total experimental control by the investigator (organisms being bred for specific experimental reasons—inbred strains, participants being fully randomized to treatments, etc.) are always problematic in that alternative explanations of a finding are invariably possible. This is certainly the case with human twin and adoption studies. This does not, however, rule them out as useful research designs. It simply requires that findings be scrutinized closely and particular findings be subjected to replication and possible refutation using varied samples and methods (cf., Scarr, 1981, p. 528–529). On the environmental side, Stoolmiller (1998, 1999) has made forceful arguments that adoption studies have seriously under-sampled high-risk environments (restriction of range) and thus underestimate shared family environmental influences (see, however, Loehlin and Horn, 2000).

With respect to twin studies, concerns have been raised about both the representativeness and differential treatment of MZ and DZ twins. It is often claimed that twins are somehow not representative of human populations and that the circumstances of growing up as a twin are so different from ordinary circumstances that behavioral findings based on twin studies cannot be expected to generalize to "normal" individuals. This general argument can be reformulated into so many variations that it is not in principal refutable and therefore lacks force. Specific versions of the argument and its implications for specific traits can, however, be tested and the degree to which they may or may not bias findings estimated (cf., various chapters in Bouchard and Propping, 1993). It has often been suggested that the simple fact of being an MZ twin might influence the personality development of such twins, yet MZ twins do not differ much from DZ twins in personality nor do either of them differ much from singletons (Johnson et al., 2002). Twins do appear to have slightly lower IQs than singletons (Breland, 1974), but this certainly is not always true (Posthuma et al., 2000) and may be changing due to improved medical care of high-risk pregnancies.

The inference that genetic factors account for the greater phenotypic similarity of MZ as compared to DZ twins clearly depends on the assumption that differences in phenotypic similarity are not due to differences in environmental similarity. Behavioral geneticists call this assumption the "equal environmental similarity assumption", a term that is somewhat misleading in that the issue is not whether MZ twins experience more environmental similarity than DZ twins, but rather whether they are more likely to share trait-relevant features of their environments. For example, MZ twins are more likely than DZ twins to share friends and parental treatment in adolescence. However, this difference does not invalidate the equal environmental similarity assumption, because similarity of friends or parental treatment is not associated with twin similarity in personality, interests, or abilities (Loehlin and Nichols, 1976, Chap. 7). Tests of the equal environmental similarity assumption have repeatedly shown that it is valid in most instances (Scarr, 1968; Lytton, 1977, 2000; Scarr and Carter-Saltzman, 1979; Kendler et al., 1993; Xian et al., 2000; Borkenau et al., 2002). Good scientific practice, however, requires that the assumption be repeatedly tested for each trait under investigation and particular findings that depend on the assumption be replicated in designs that do not make the assumption. Large studies with multiple kinships can specifically test such assumptions.

The applicability of the equal environmental similarity assumption extends to the prenatal as well as the postnatal environment. In utero, twins can be distinguished in terms of whether they share a chorion, and thus have a single placenta. MZ twins can be monochorionic (MC) or dichorionic (DC) depending on the timing of their division; DZ twins are always DC. MC twins almost always share the same placenta and if this makes them more similar than DC and DZ twins we may have a specific example of violation of the trait-relevant equal environment assumption (Prescott et al., 1999). A small number of studies comparing very small numbers of MC and DC twins suggested that MC twins are more similar than DC twins on some, but not all, measures of mental abilities (Melnick et al., 1978; Rose et al., 1981). A greater number of small-sample studies (Brown, 1977; Welch et al., 1978; Sokol et al., 1995; Gutknecht et al., 1999;

Riese, 1999), however, failed to replicate these chorion effects. Moreover, a recent, large epidemiological study (Derom et al., 2001) using a near-representative sample from the East Flanders Prospective Twin Survey could not replicate the specific effects previously reported and found no chorion effect on total IQ ($r_{MC} = .83$, $n = 175$ pairs; $r_{DC} = .82$, $n = 95$ pairs; $r_{DZ} = .44$, $n = 181$ pairs). This latter study did report a chorion effect for two different mental ability measures, but the effects were very small, prompting the authors to emphasize caution and the need for replication. Nevertheless, careful assessment of twin placentation at birth would be highly desirable and significantly improve the quality of twin studies. It would also be very useful to parents and physicians as some rare physical disease processes occur in MC twins that do not occur in DC twins (Machin, 2001). For these diseases chorion type is a trait-relevant environmental variable. Generally speaking, however, twins do not differ in terms of their disease related characteristics (Andrew et al., 2001), but see Phelps et al. (1997) for arguments regarding viral influences on schizophrenia, Phillips (1993) for arguments regarding placentation and the fetal origin of disease hypothesis (i.e., that adult-onset disorders are affected by in utero stress and trauma), and subsequent defense of the twin method by a number of investigators (Braun and Caporaso, 1993; Duffy, 1993; Leslie and Pyke, 1993; Macdonald, 1993; Christensen et al., 1995). An entire issue of the journal *Twin Research* was devoted to the fetal origin of disease hypothesis (Lambalk and Roseboom, 2001), but none of the articles dealt with behavioral phenotypes.

Fortunately, inferences about the nature and existence of genetic and environmental influences on individual differences in behavior do not rest solely with twin studies. In particular, the adoption study design provides the opportunity for constructively replicating findings from twin studies. Thus, if heritable influences are important, we would expect to observe significant correlations between biological parents and adopted children even though they never contributed to the rearing of those children past the very earliest stages of life. Alternatively, if shared environmental factors are a major source of similarity among relatives, then we should observe significant correlations among adoptive relatives even though they are not biologically related.

The major limitation of adoption studies is that, owing to adoption practices, the homes in which adopted individuals are reared may be overly homogeneous, leading to an underestimation of shared environmental effects. For example, if adopted children were only placed in high-income families and never reared in poverty, then environmental effects associated with family income and poverty would never be revealed in an adoption study. Stoolmiller (1998, 1999) has forcefully argued that for certain traits (especially those related to socioeconomic status), adoption studies have seriously under-sampled environments (restriction of range) and therefore underestimated shared family environmental influences.

## HUMAN COGNITIVE ABILITIES

### Nature of "g" and Special Mental Abilities

In the last 20 years there has been a dramatic change of opinion amongst psychologists regarding the structure of human cognitive abilities. The theory of a hierarchical structure with a general cognitive factor, more accurately called "the g factor", at the apex has returned to prominence if not dominance after a long hiatus. The role of specific mental abilities—broad second-order factors—while not entirely eclipsed has become much less important. This is true both in the research domain (Bouchard, 1999) and in applied settings (Gottfredson, 1997b). An example of such a hierarchical structure is shown in Figure 2.

At the bottom of the hierarchy are specific psychological tests (Concept Formation, Incomplete Words, Picture Vocabulary, etc.) that are positively intercorrelated. These intercorrelations can be accounted for by a smaller number of "first-order latent factors" (Fluid Reasoning, Comprehensive Knowledge, Processing Speed, etc.), which in turn are correlated. General intelligence (g) is at the apex of the hierarchy and is needed to account for the correlations among the first-order factors. In this type of analysis the g factor generally accounts for the most variance (often more than all the specific variance in the first-order factors combined). A variety of factor methods are available for computing; all give essentially the same results (Jensen and Weng, 1994).

Even though there remain some critics of the concept of general intelligence (Horn, 1998), there is a strong consensus within psychology that g can be measured objectively using appropriate methods of factor analysis. Moreover, as a higher-order construct, g is entirely compatible with theories that posit the existence of multiple correlated abilities. The g factors derived from different well-chosen arrays of tests, often specified by a competing theory, are so highly correlated that they are essentially identical. A large number of replicated empirical findings supporting this conclusion are reported in detail in Carroll
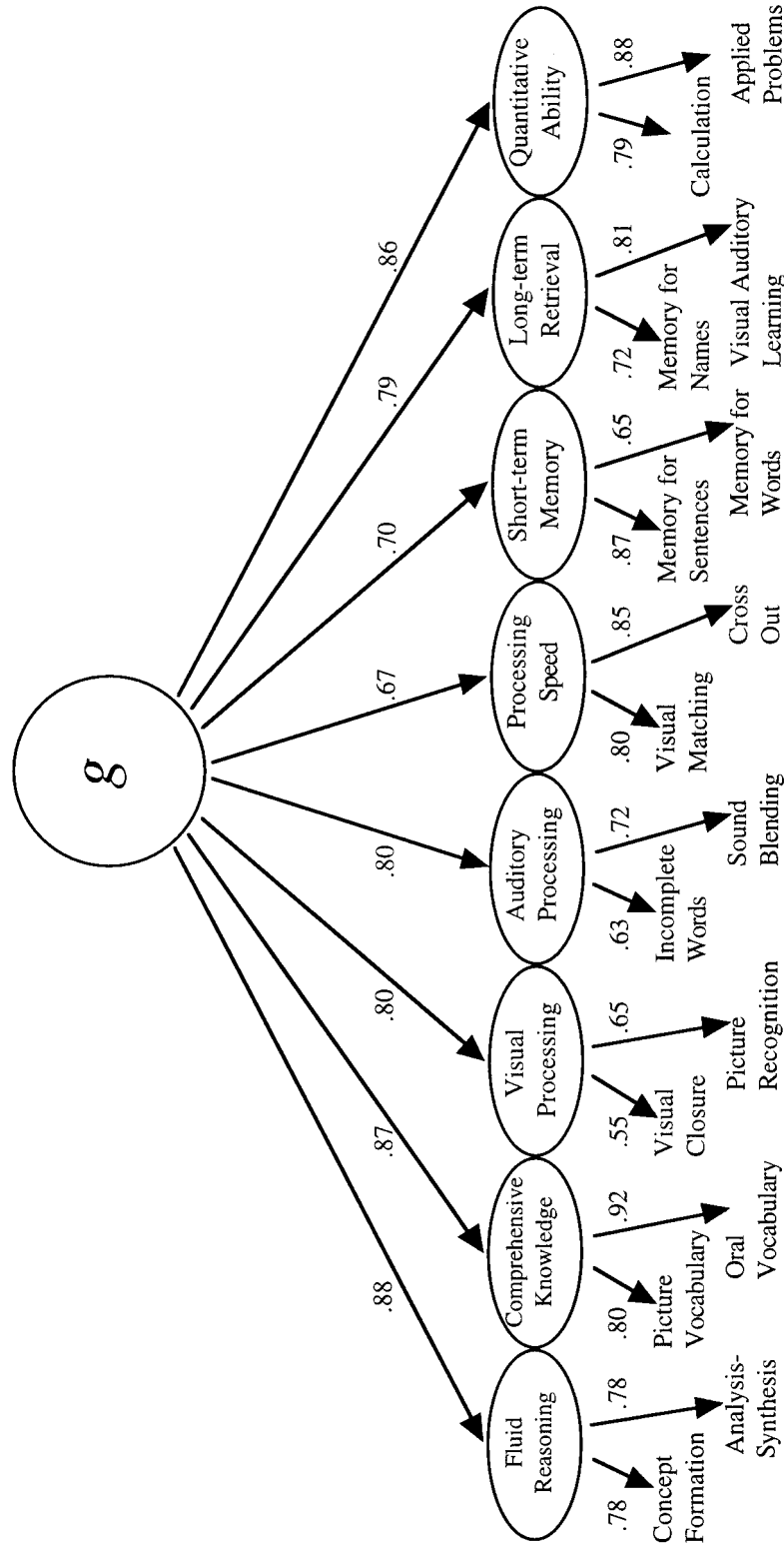
**Figure 2** Standardized factor loadings for the three-stratum theory of intelligence (from Bickley et al., 1995).

(1993). More recent studies include Bickley et al. (1995), Aluja-Fabregat et al. (2000), and Neuman et al. (2000). The hierarchical factor structure is highly similar if not identical across diverse ethnic groups within industrialized populations as well as across the two sexes (Carretta and Ree, 1995).

Although the nature of *g* remains elusive, progress is being made. Jensen (1998) has probably given this problem more thought than any other psychologist since Spearman (1904), and he has captured the unstated conception of *g* and primary factors (special mental abilities) that many researchers, particularly those with a biological orientation, actually carry in their minds. "Unlike any of the primary, or first-order, psychometric factors revealed by factor analysis, *g* cannot be described in terms of the knowledge content of mental test items, or in terms of skills, or even in terms of theoretical cognitive processes. It is not fundamentally a psychological or behavioral variable, but a biological one. We know that *g* reflects certain properties of the human brain because of its correlations with individual differences in a number of brain variables, such as size, metabolic rate, nerve conduction velocity, and the latency of evoked electrical potentials" (Jergen, 1998, p 578).

Importantly, the neurological basis for *g* is beginning to be explicated. Garlik (2002) has proposed a connectionist model that incorporates recent advances in neuroscience (having properties of neural systems) that is compatible with Jensen's description. He argues that individual differences in neural plasticity demand a *g* factor. Mackintosh (1998) has pushed the existing data a bit further, asserting that there is "reason to believe that the planning and monitoring functions attributed by cognitive psychologists and neuropsychologists to a central executive may constitute the basis of *g* or general intelligence" (p 325).

As we will see shortly, studies of IQ and brain structure in twins converge on a similar conclusion.

## Practical Importance of *g*

Before turning to the behavior genetic evidence regarding *g* we note that measures of *g* in the applied domain carry much if not almost all of the predictive validity of any battery of mental ability tests, and these findings generalize across both sexes and ethnic groups within industrialized populations (Sackett et al., 2001). In addition, unlike research in the 1960s and early 1970s, which was based on numerous very small samples and which led applied psychologists to believe that the predictive utility of cognitive tests did not generalize from one situation to the next, meta-analytic summaries and individual studies utilizing

huge samples document that general ability measures predict a wide-range of real-world criteria (Carretta and Doub, 1998; Ree and Carretta, 1998; Schmidt and Hunter, 1998; Schmidt, 2002). Extensive discussion of these issues can be found in Carroll (1997), Gordon (1997), and Gottfredson (1997a).

## Estimates of Genetic and Environmental Influence on *g*

An informative place to begin the discussion of studies of genetic influence on *g* is a recent reanalysis, by Devlin et al. (1997a), of a slight update of kinship correlations for IQ originally summarized and published by Bouchard and McGue (1981). Devlin has written critically of the behavioral genetic literature (Devlin et al., 1995, 1997b), so that we may expect him to report a conservative estimate of genetic influence. The most important findings in their article are that: the broad heritability of IQ is about 50% (additive variance = .34, nonadditive genetic variance = .15); twin maternal (i.e., in utero) effects account for 20%, and sibling maternal effects account for 5% of IQ variance; and shared environmental factors account for 17% of IQ variance. As McGue (1997) notes in an accompanying commentary, the result of this article is to center the debate on whether IQ is 50 or 70% heritable. This is a remarkable shift from the previous view, asserted by numerous critics, that the heritability of IQ is near zero.

While we regularly use modeling in our own work, we would like to repeat the caveat we placed at the end of our original presentation. "Although the data clearly suggest the operation of environmental effects, we found no evidence for two factors sometimes thought to be important—sex-role effects and maternal effects. That the data support the inference of partial genetic determination for IQ is indisputable: that they are informative about the precise strength of this effect is dubious. Certainly the large amount of unexplained variability within degrees of relationship, while not precluding attempts to model the data, suggests that such models should be interpreted cautiously" (Bouchard and McGue, 1981, p 1058).

Given the need for cautious interpretation, it is especially noteworthy that the Devlin et al. (1997a) estimate of 49% for the heritability of IQ is in close agreement with the estimate of 51% reported by Chipuer et al. (1990) and the estimates of 47 and 58% reported by Loehlin (1989), who fit different although clearly converging models to the IQ correlations. Where the different analyses disagree is in terms of apportioning environmental, and not genetic, effects. Devlin et al. (1997a) conclude that the prenatal envi-

ronment exerts a significant influence on IQ, increasing the IQ similarity of twins over other relative pairings. In contrast, Chipuer et al. and Loehlin conclude that the postnatal rather than the prenatal environment is most important.

The Devlin et al. (1997a) conclusion that the prenatal environment contributes to twin IQ similarity is especially remarkable given the existence of an extensive empirical literature on prenatal effects. Price (1950), in a comprehensive review published over 50 years ago, argued that almost all MZ twin prenatal effects produced differences rather than similarities. As of 1950 the literature on the topic was so large that the entire bibliography was not published. It was finally published in 1978 with an additional 260 references. At that time Price reiterated his earlier conclusion (Price, 1978). Research subsequent to the 1978 review largely reinforces Price's hypothesis (Bryan, 1993; Macdonald et al., 1993; Hall and Lopez-Rangel, 1996; see also Martin et al., 1997, box 2; Machin, 1996).

Consideration of features of kinship similarity for IQ not incorporated into the analyses reported by Devlin et al. and the other modelers can help to further elucidate the nature of environmental influences on IQ. In particular, kinship correlations for IQ vary with age and failure to take this into account may have resulted in an overestimate of maternal environmental effects. For example, the kinship that provides the most direct test for postnatal environmental effects is the correlation between nonbiologically related, reared-together (i.e., adoptive) siblings (unrelated together or URTs). Devlin et al. did not include this kinship in the analysis they report "because the observed correlations are extremely variable" (p. 469). In fact, and as shown in Figure 3, a major contributor to the heterogeneity in the adoptive sibling correlation is the age of the sample. The childhood data are from Burks (1928), Freeman et al. (1928), Leahy (1935), Skodak (1950), Scarr and Weinberg (1977), and Horn et al. (1979). The adult data are from Scarr and Weinberg (1978), Teasdale and Owen (1984), Scarr et al. (1993), Loehlin et al. (1997), and Segal (2000).

The adoptive siblings assessed in childhood or adolescence, when they were presumably still living together, had an average IQ correlation of .26, suggesting that common rearing accounts for 26% of IQ variance. The adoptive sibling pairs assessed in adulthood, however, had an average IQ correlation of only .04, suggesting that common rearing effects do not endure once the siblings no longer live together. Importantly, failure to observe significant IQ similarity in adult adoptive sibling pairs does not appear to be a consequence of biased sample selection. Teasdale and

Owen (1984) reported an IQ correlation of .02 for 24 pairs of adopted, adult brothers obtained through Danish conscription board files. Because evaluation for conscription is mandatory for Danish males (regardless of medical status), and because the researchers had access to the complete Danish adoption register, this sample can be considered one of the most, if not the most, representative adoption study in the literature. While the representativeness of the sample does not solve the problem of restriction of range, it certainly makes it less salient.

Twin studies also suggest that genetic and environmental contributions to IQ vary with age. Wilson (1978) was one of the first to explore changes in kinship correlations for IQ in a longitudinal study, and his findings are shown in Figure 4.

Prior to age 2, the phenotypic assessments used in this study are best characterized as indicies of mental development, and not IQ. The content of these mental development assessments is quite different from the, primarily verbal, content of the IQ tests used in the later years. In any case, if we use the Falconer formula $2(r_{mz} - r_{dz})$ as an estimate of genetic influence we see that in the early months there is minimal genetic influence but that by the age of 1 genetic factors begin to express themselves and they get much larger from 4 years of age and on. The same influences are expressing themselves in the sib-twin and midparent-offspring correlations. These longitudinal data thus suggest that with age, genetic factors increase while environmental factors decrease in importance.

Building on the work by Wilson, McGue et al. (1993) plotted twin IQ correlations by age. The IQ variance estimates derived from comparing the age-specific MZ and DZ correlations are shown in Figure 5.

Again we see the growing expression of genetic influence and decreasing manifestation of shared environmental influence. Not shown in Figure 5 is the extreme paucity of adult twins in studies of IQ—the younger twins swamp the data base. It is far easier to recruit twins in school settings than it is to recruit adult twins and bring them to a laboratory. Nevertheless, these data are highly consistent with the URT data and also suggest that heritability is contingent on age.

Recent longitudinal family and adoption data from the Texas Adoption Project (TAP) and Colorado Adoption Project (CAP) confirm these findings. In the TAP, researchers reported that estimates of IQ heritability increased (from .38 to .78) while estimates of shared environmental influence decreased (from .19 to .00) as the adopted children in the families aged
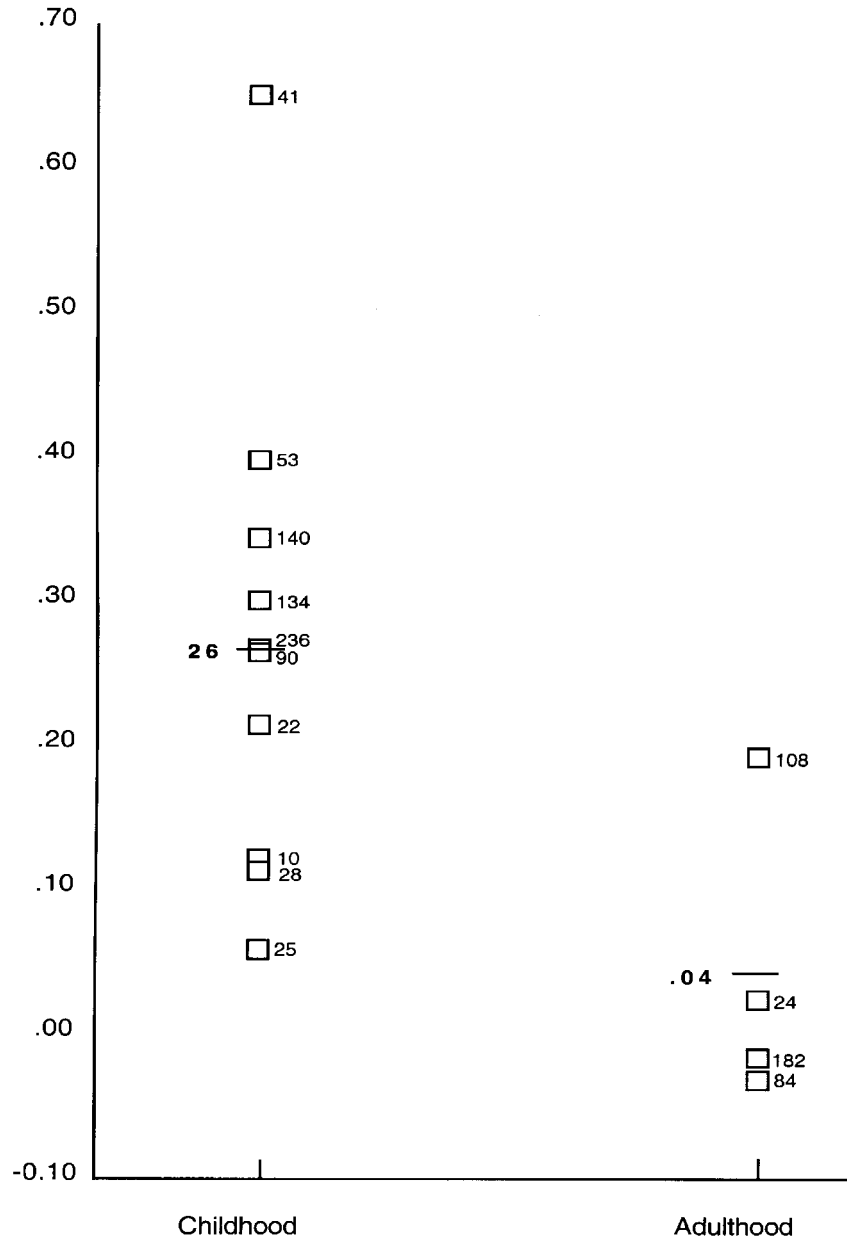
**Figure 3** IQ correlations (open boxes), sample sizes (associated numbers), and weighted mean correlations (bars) for unrelated individuals reared together organized as pairs measured in childhood and pairs measured in adulthood.

from adolescence to young adulthood (Loehlin et al., 1997). A notable feature of TAP is that test reliability was incorporated into the model so that parameter estimates refer to true score rather than observed score variance. In CAP (Plomin et al., 1997), parent-offspring IQ correlations (weighted average for mothers and fathers) for adoptive and control (matched biological) families were assessed at 1, 2, 3, 4, 7, 12, and 16 years of age. These findings are shown in Figure 6. The correlations are modest and in about the same

range for both types of families until about age 5, after which they diverge dramatically, with the adoptive family correlations reaching an asymptote of zero at age 12. Model fitting to the data yielded a heritability estimate of .56, an environmental transmission value of .01, an assortative mating value of .21, and a genotype-environment correlation of .01. A very similar trend, of adopted children becoming more similar to their biological than their adoptive parents over time, was reported by Honzik (1957).
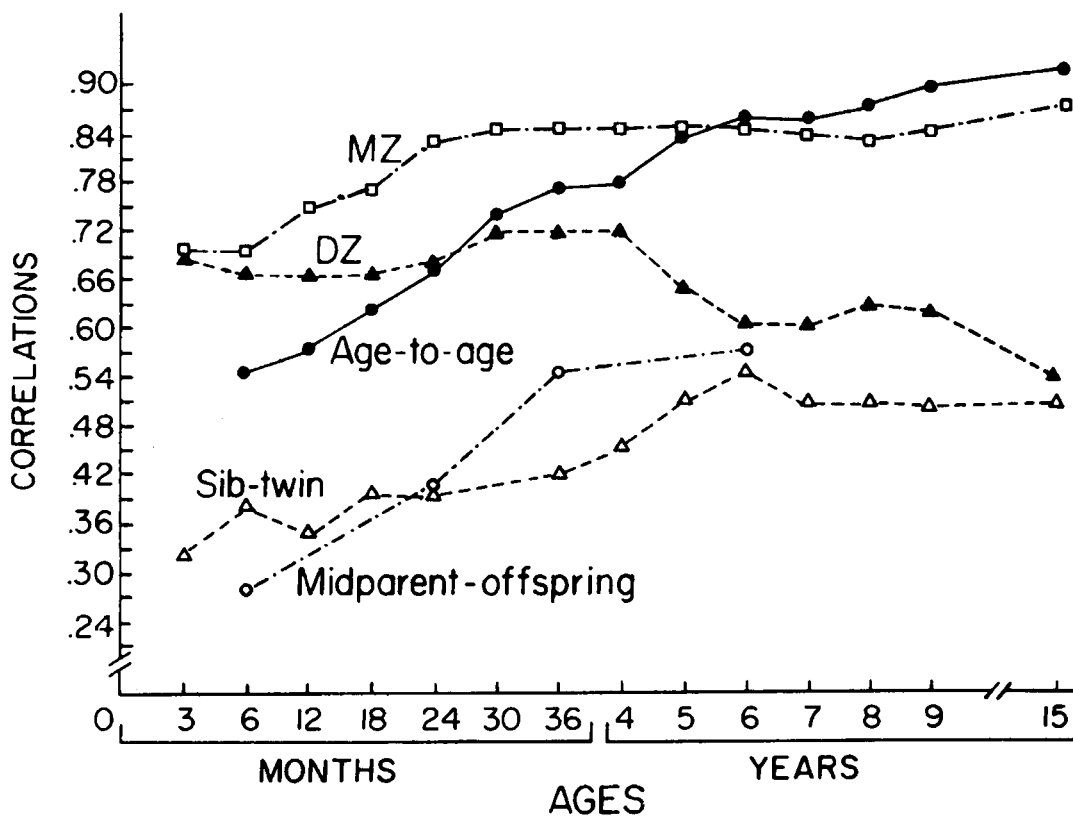
**Figure 4** Mental development correlations for MZ twins, DZ twins, twin-sibling sets, parent-offspring sets, and individual children from age to age (from Wilson, 1983).

Boomsma et al. (1999) have recently published estimates of heritability and shared environmental influence for IQ by age (5, 7, 10, 16, 18, 27 years of age) from a sample of Dutch twins. To these estimates we add data extending the Dutch sample to age 50 that were kindly provided to us by Prof. Boomsma (see



**Figure 5** IQ variance component estimates derived from published IQ twin correlations. Estimates are based on the standard assumptions used with the Falconer heritability formula (from McGue et al., 1993).

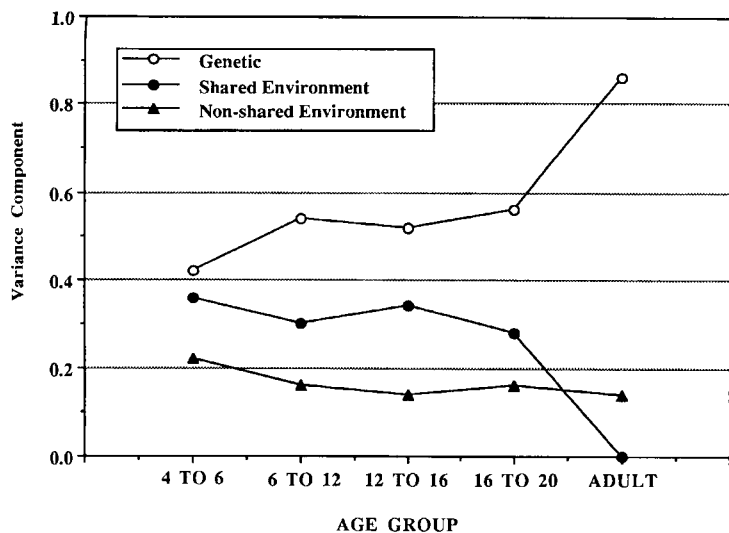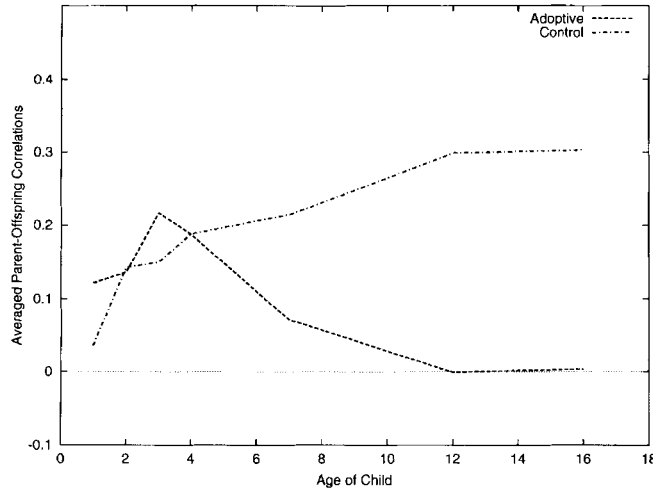**Figure 6** Correlations between parents' IQ and children's IQ in adoptive and control (i.e., biologically related) families at 1, 2, 3, 4, 7, 12, and 16 years (from Plomin et al., 1997).

also Posthuma et al., 2002a, Figure 12.1). The results are shown in Figure 7.

Interestingly, the heritability of general cognitive ability may decline in late life. McClearn et al. (1997) reported estimates of heritability and shared environmental influence in a sample of 117 twins age 80 years or older. For the first principal component of the seven cognitive tests, an index of *g,* heritability was estimated at .62 (95% CI, .29–.73), and shared environment was estimated at .11 (95% CI, .00–.47). If a

short form of the Weschler Adult Intelligence Scale was used to estimate *g,* heritability was estimated at .55 (95% CI, .19–.76), and shared environment was estimated at .20 (95% CI, .00–.47). The influence of shared environment could have been dropped from the model in both instances as indicated by the confidence intervals. McGue and Christensen (2001) recently replicated McClearn et al.'s findings by reporting a heritability estimate of .54 (95% CI, .27–.63) for a general cognitive ability measure, in a sample of
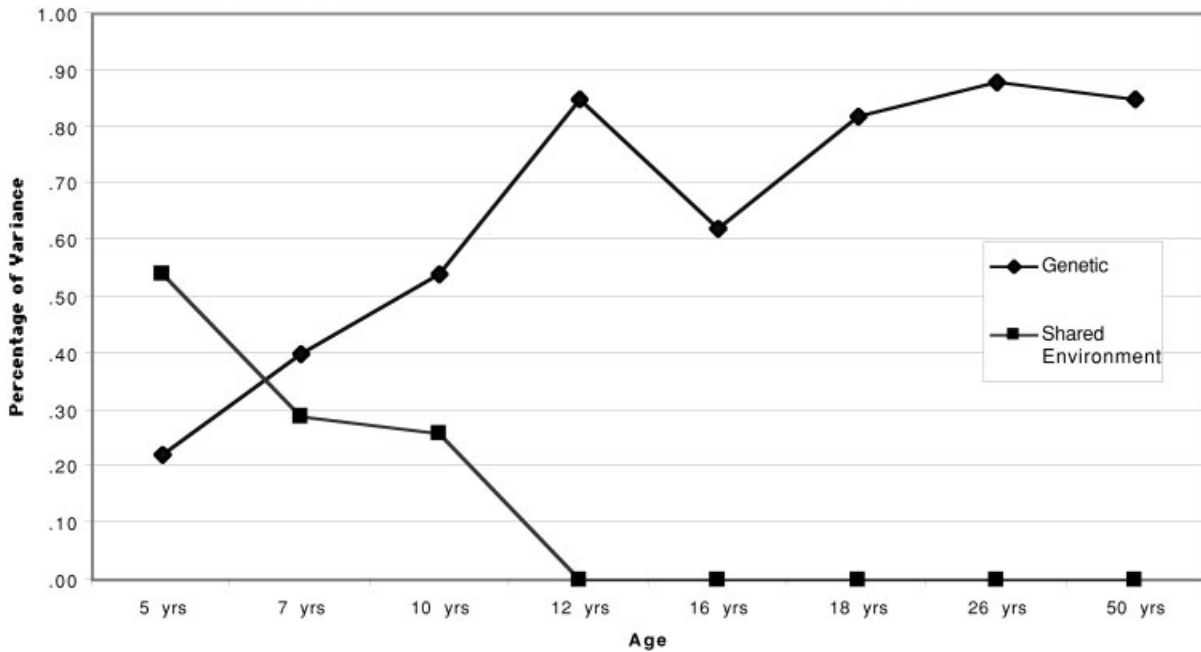


**Figure 7** Estimates of genetic ($h^2$) and shared environmental ($c^2$) variance for IQ by age for Dutch samples.

Danish twins 75 years and older. These heritability estimates are a bit lower than in younger adult data (Plomin et al., 1994), and suggest that heritability decreases in older cohorts. This conclusion is also supported by longitudinal studies of older twins (Finkel et al., 1995, 1998).

In summary, twin, adoption, and longitudinal family studies of IQ all converge on the conclusion that genetic factors increase while shared environmental factors decrease in importance with age, at least until middle age. Summary estimates of heritability from Devlin et al. (1997a), Chipuer et al. (1990), and Loehlin (1989) all fail to take these age effects into account.

## Implications of IQ Heritability: Neurogenetics

One of the most unfortunate misinterpretations of the heritability coefficient is that it provides an index of trait malleability (i.e., the higher the heritability the less modifiable the trait is through environmental intervention). Research on IQ provides an effective counter example to this false conception. As reviewed above, all available evidence converges on a moderate (in adolescence and childhood) to strong (in adulthood) heritability for IQ. Nonetheless, there is an equally strong convergence of evidence indicating that the population average IQ has increased substantially over the past 50 years (Flynn, 1998). The observation of significant trait heritability along with substantial secular increases, although perhaps paradoxical, is not a peculiar feature of IQ. Adult height is also highly heritable, but yet mean height appears to also have increased substantially in the same populations and over the same time period for which increases in IQ have been observed (Fernández-Bellesteros et al., 2001). Because the populations studied must be relatively genetically stable, environmental influences must constitute the source of secular increases in IQ. Dickens and Flynn (2001) recently proposed a theoretical model that would account for the high within-population heritability of IQ while allowing for environmental modifiability of the sort that would produce strong secular changes.

Of what utility is a heritability estimate if it does not implicate trait (non)modifiability? The finding that IQ is heritable has motivated some psychologists to search for the brain origins for these heritable influences, focusing initially on simple brain size. Paleo-anthropologists have long operated on the assumption that bigger brains reflect greater cognitive capacity.

This assumption is based on the fact that human brains are about three times larger than would be expected for a primate of our weight and consume an inordinate amount of our entire energy intake—20 to 25% of resting metabolism compared to 8% for anthropoid primates and 3–4% for most mammals (Leonard and Robertson, 1994). Principles of evolutionary ecology would immediately lead one to presume that such an organ had been under intense selection. The interesting question is whether selection is aimed at specific mental abilities, *g,* or something else. A fascinating study of the evolution of the mammalian brain by Finlay and Darlington (1995) concluded that "the most likely brain alteration resulting from selection for any behavioral ability may be a coordinated enlargement of the entire nonolfactory brain" (p. 1578). If Jensen is correct in his hypothesis that *g* is a biological feature of the entire brain, then brain size and other features of the brain should be somewhat related to *g,* in a diffuse manner reflecting many different parts of the brain. A recent MRI study of the brains of 97 healthy elderly men lends strong support to this hypothesis. The authors concluded that "the relationship between specific cognitive tests and regional brain volumes could best be summarized by a significant positive relationship between a general brain size factor and a general cognitive factor, and not by associations between individual tests and particular brain regions" (MacLullich et al., 2002, p. 169). The correlation between the two factors was .42.

As the MacLullich et al. study shows, correlations between IQ and brain size, originally established using crude measures of brain size, have recently been replicated using powerful brain imaging methods to estimate brain structure and volume. This work has been extended to twins. In a study of 10 MZ and 10 DZ twins, Thompson et al. (2001) reported that many specific brain structures were strongly heritable (i.e., 80% or more), and that frontal gray matter volume correlated significantly with IQ (.37–.45). Although the Thompson et al. study is small and warrants cautious interpretation, their findings were quickly replicated and extended by Posthuma et al. (2002b), who also reported a strong heritability for brain volume (greater than 80%), and a significant correlation between brain volume and IQ (.25, a value lower than the correlations reported by Thompson et al. and MacLullich et al.). Taken together, these two studies illustrate the potential power of using brain-imaging technology in genetically informative designs to uncover the genetic and neural origins of high cognitive functioning (Plomin and Kosslyn, 2001).

## Genetic and Environmental Influence on Specific Mental Abilities

While the bulk of behavioral genetic research on human cognitive ability has focused on general cognitive ability, research on specific mental abilities (i.e., abilities at one level below the general factor in the hierarchical model) also implicates the importance of genetic influences. The CAP has reported parent-offspring correlations by age for Verbal Ability, Spatial Ability, Speed of Processing, and Memory. The results are similar to those in Figure 6 in that the adoptive correlations hover about zero and the biological correlations climb with age, although for the specific mental abilities (SMA) they are not as high. Bouchard et al. (1990b) updated the meta-analysis by Nichols (1978) of the world twin literature for SMAs, adding data from MZA and DZA twins. They showed that, much like the IQ literature, there was great heterogeneity in the data. The mental ability data were summarized into the same four factors as the CAP data. Bouchard (1998) attempted to model the data but none of the models fit, and he presented the results for the models that fit the data best. Those results are given in the first row under each of the four mental abilities summarized in Table 2. The next group of studies is of young people and we utilized the same grouping of mental abilities. The third grouping is based on adult data, again using the same SMA grouping, while the final grouping is for late-life samples (i.e., 75 and older).

Table 2 illustrates much the same age effect on heritability (increasing through middle age until it begins to drop in old age) as we showed for $g$, except for memory. These results are not a surprise as these mental abilities are highly intercorrelated and constitute the vehicles with which we derive a measure of $g$. It is possible to fit what is known as a common pathway model, shown in Figure 8, to such data and estimate the percentage of genetic and variance contributed by $g$ and by each specific test. As the figure shows, genetic (G), shared (C), and nonshared (E) sources of variance are estimated for the cognitive factor ($g$), which is made up of the variance common to the SMAs, and then separately estimated for the remaining variance that is not shared. The solution for the Minnesota Twin Study of Adult Development and Aging is shown in Table 3. The total heritability (under Total) is now divided into that which is shared with $g$ (General) and that which is specific to each test. The same is true for the nonshared environmental variance, which is largely specific to the tests. Note that the model allows for shared environmental variance at both the general and specific level.

None is necessary at the general level to fit the data. The heritability of the general factor $g$ for this data set is .81.

## Specific Genes for $g$

The effort to identify "genes for cognitive ability" has begun, although no such genes have yet been discovered. A number of candidate genes have come to the fore in initial studies, but none of the findings have been replicated. Posthuma et al. (2002a) provide a brief review, Plomin et al. (2001) and Hill et al. (2002) provide an example of the difficulties of replication in this domain. It has been extremely difficult to identify the genes involved in polygenic traits even in animal studies, but progress is being made (Morley and Montgomery, 2001; Toma et al., 2002) and there is reason for optimism as more and more powerful tools come online.

## PERSONALITY

## Nature and Importance of Human Personality

Personality research has enjoyed a strong resurgence over the past decade. This resurgence is due in part to the recognition that personality is predictive of a wide range of behavioral and social outcomes. Barrick and Mount (1991) and Hough and Oswald (2000) reviewed the use of personality assessments in work settings and concluded that they contribute importantly to the prediction of worker performance and productivity. Dawis (1992) in counseling and Harkness and Lilienfeld (1997) in clinical psychology have shown how personality factors can play a critical role in advising and treatment planing. Personality is also strongly associated with mental health problems ranging from depression to substance abuse (Widiger and Snakis, 2000), and there is a growing recognition that some mental health problems might actually represent the extreme of normal range variation in personality (Krueger et al., 1998). Another contributor to the revitalization of personality research is the growing recognition that the domain of personality, like human abilities, is hierarchically organized. Unlike human abilities where a single super-ordinate dimension is generally agreed on, there is some dispute over the number of higher-order traits needed to span personality. The major alternative hierarchical schemes for organizing personality, as given by Bouchard and Loehlin (2001) in a recent review of the genetics of

**Table 2  Estimates of Genetic and Shared Environmental Influence on Verbal Ability, Spatial Ability, Perceptual Speed and Accuracy, and Memory from a Comprehensive Meta-Analysis and Studies of Young, Adult, and Old Twins**

| Study or Source of Information | Genetic Influence | Shared Environment |
|---|---|---|
| Verbal Ability | | |
| Meta-analysis of multiple sources of data* | .48 | .21 |
| Young participants | | |
| Colorado Adoption Project (age 16), (Plomin et al., 1997)[†] | .54 | .02 |
| Young twins (Jacobs, 2001)[‡] | .44 | .23 |
| Young twins (Wilson, 1975)[‡] | .60 | .21 |
| Young twins (Segal, 1985)[‡] | .82 | .00 |
| Young twins (Labuda et al., 1987)[‡] | .54 | .17 |
| Mean of young participants | .59 | .13 |
| Adult participants | | |
| Swedish adult twins (Pedersen, et al., 1992)[§] | .58 | .09 |
| Minnesota Twin Study of Adult Development and Aging[‡] | .77 | .00 |
| Mean of adult participants | .68 | .05 |
| Very old cohorts | | |
| Swedish very old cohort[‖] | .32 | .13 |
| Danish twins (McGue and Christensen, 2001) | .37 | .00 |
| Mean of very old participants | .35 | .07 |
| Spatial Ability | | |
| Meta-analysis of multiple sources of data* | .60 | .00 |
| Young participants | | |
| Colorado Adoption Project (age 16), (Plomin et al., 1997)[†] | .39 | .01 |
| Young twins (Jacobs, 2001)[‡] | .70 | .00 |
| Young twins (Wilson, 1975)[‡] | .50 | .18 |
| Young twins (Segal, 1985)[‡] | .84 | .00 |
| Young twins (Labuda et al., 1987)[‡] | .24 | .43 |
| Mean of young participants | .53 | .12 |
| Adult participants | | |
| Swedish adult twins (Pedersen et al., 1992)[§] | .46 | .07 |
| Minnesota Twin Study of Adult Development and Aging[‡] | .73 | .27 |
| Mean of adult participants | .60 | .17 |
| Swedish very old cohort[‖] | .32 | .13 |
| Perceptual Speed and Accuracy | | |
| Meta-analysis of multiple sources of data* | .64 | .00 |
| Young participants | | |
| Colorado Adoption Project (age 16), (Plomin et al., 1997)[†] | .22 | .06 |
| Young twins (Jacobs, 2001)[‡] | .61 | .05 |
| Young twins (Wilson, 1975)[‡] | — | — |
| Young twins (Segal, 1985)[‡] | .56 | .12 |
| Young twins (Labuda et al., 1987)[‡] | .47 | .26 |
| Mean of young participants | .47 | .12 |
| Adult participants | | |
| Swedish adult twins (Pedersen et al., 1992)[§] | .58 | .00 |
| Minnesota Twin Study of Adult Development and Aging[‡] | .62 | .38 |
| Mean of adult participants | .60 | .19 |
| Swedish very old cohort[‖] | .62 | .00 |

**Table 2   (*Continued*)**

| Study or Source of Information | Genetic Influence | Shared Environment |
|---|---|---|
| Memory | | |
| Meta-analysis of multiple sources of data* | .48 | .00 |
| Young participants | | |
| Colorado Adoption Project (age 16), (Plomin et al., 1997)[†] | .26 | .05 |
| Young twins (Jacobs, 2001)[‡] | .34 | .19 |
| Young twins (Wilson, 1975)[‡] | — | — |
| Young twins (Segal, 1985)[‡] | — | — |
| Young twins (Labuda et al., 1987)[‡] | — | – |
| Mean of young participants | .30 | .12 |
| Adult participants | | |
| Swedish adult twins (Pedersen et al., 1992)[§] | .38 | .00 |
| Minnesota Twin Study of Adult Development and Aging[‡] | .55 | .45 |
| Mean of adult participants | .47 | .23 |
| Very old cohorts | | |
| Swedish very old cohort[‖] | .52 | .00 |
| Danish twins (McGue and Christensen, 2001) | .43 | .03 |
| Mean of very old participants | .48 | .02 |

  * Based on model-fitting data from a four group design (MZA, DZA, MZT, and DZT). See Bouchard (1998), Table 4 for details.

  [†] Data base is made up of factor scores.

  [‡] We followed Finkel and McGue (1995) and chose the Information subtest to measure verbal ability, the Block Design subtest to measure spatial ability, the Digit Symbol (or Coding on the WISC) to measure perceptual speed, and Digit Span to measure memory.

  [§] Based on model-fitting data from a four group design (MZA, DZA, MZT, and DZT), mean of subtests.

  [‖] All scores based on two tests except for perceptual speed measured by the Symbol Digit test.

personality, are shown in Table 4. The table illustrates that even though alternative schemes differ in terms of the number of higher-order dimensions (ranging from 3 to 9), there is broad agreement on how specific personality traits are hierarchically organized.

The Eysenck "Big Three" of Neuroticism, Psychoticism, and Extraversion have dominated European personality theory and a large block of behavioral genetic research for many years (Eaves et al., 1989; Nyborg, 1997). Eysenck's construct of Psychoticism (Eysenck et al., 1985) is the only one of the three that is controversial (Claridge and Birchall, 1978), and, interestingly, behavioral genetic studies have been instrumental in throwing doubt on its validity (Heath and Martin, 1990). Because of this uncertainty, the three-factor model proposed by Tellegen (1982) and used as the basis for the development of the Multidimensional Personality Questionnaire (MPQ) is currently preferred among three-factor models. These three factors are Positive Emotionality (comparable to aspects of Eysenck's Extraversion factor and referring to the tendency to be positively and actively engaged with one's environment), Negative Emotionality (comparable to aspects of Eysenck's Neuroticism factor and referring to the tendency to experience negative mood states), and Constraint (the ability and willingness to inhibit behavioral response impulses). The Big Five model, championed by Costa and McCrae (1992), retains the Eysenkian higher-order dimensions of Neuroticism and Extraversion, but distinguishes two aspects of Eysenck's Psychoticism, Agreeableness (i.e., being cooperative and friendly) and Conscientiousness (similar to Tellegen's Constraint factor), and adds a fifth dimension, Openness (i.e., willingness to welcome diverse intellectual and cultural experiences), that are not well captured in the three-factor models. The Zuckerman and Cloninger (1996) schemes are quite similar, with the latter enjoying wide popularity in psychiatric research. The Big Nine is based on years of research in the world of work (Hough, 1992), but appears to be deficient in measures of stimulus seeking and impulsivity even though these traits have been of critical importance to investigators who study addictive behavior (substance abuse, alcoholism, gambling, etc.) (Konner, 1990; Zuckerman, 1994; Bevins, 2001). Although the alternative models of personality are not without their critics (Block, 1995a,b, 2001), their commonalties provide an important scheme for organizing what might otherwise be a bewildering array of findings on individual personality scales (Konner, 1990; Hough, 1992; Goldberg, 1993; Zuckerman, 1994; Zuckerman and Cloninger, 1996; Bevins, 2001).
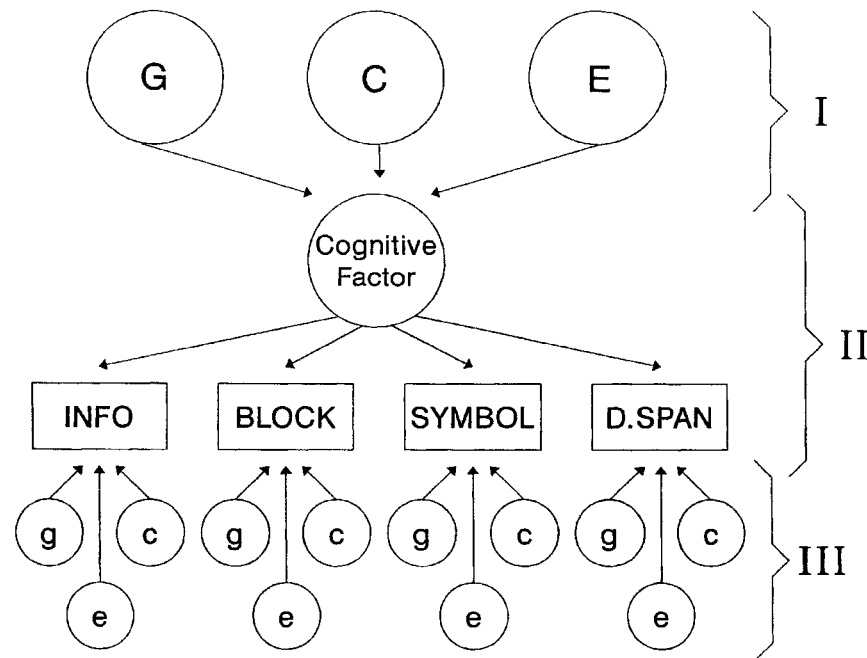
**Figure 8** Common pathway model. Level I denotes the genetic (G), shared environmental (C), and nonshared environmental (E) influences on the cognitive ability factor. Level II denotes the factor structure of four cognitive measures combined into a single factor; the path coefficients represent factor loadings. Level III denotes the specific genetic (g), shared environmental (c), and nonshared environmental (e) influences on the individual cognitive tests. INFO, information; Block, block design; Symbol, digit symbol; D. Span, digit span. From Finkel et al. (1995).

## Estimates of Genetic and Environmental Influence on Personality

The best evidence for genetic influence on personality, as Darwin (1871, pp. 101–111) noted, is the successful domestication of animals. For many years, psychologists avoided studying personality in nonhuman animals for fear of being accused of anthropomorphism. This is no longer the case. Gosling and John (1999) have shown how the Big Five dimensions of personality can be applied in animal studies, and Gosling (2001) has described animal models of personality in a major review. Behavioral genetic studies of animal behavior related to personality have been underway for years (DeFries et al., 1966, 1978). Animal models of anxiety (a key feature of Neuroticism) are particularly popular, but because of the limited repertoire of measures generally used in animal studies, there is always a question regarding how well the animal model represents the human condition. Turri et al. (2001) provide a nice example of how complex this problem is.

Early meta-analyses of twin studies of personality can be found in Nichols (1978), Eaves et al. (1989), and Bouchard (1997). In addition to these, Bouchard and Loehlin (2001) organized findings from four recent large studies of adult twins according to the Big

**Table 3   Estimated Variance Components Resulting from the Best Fit Model in Figure 8 to Minnesota Twin Study of Adult Development and Aging Data**

| Variable | Heritability | | | Nonshared Environment | | |
|---|---|---|---|---|---|---|
| | General | Specific | Total | General | Specific | Total |
| Information | .39 | .38 | .77 | .03 | .20 | .23 |
| Block design | .49 | .24 | .73 | .04 | .23 | .27 |
| Digit symbol | .24 | .38 | .62 | .02 | .36 | .38 |
| Digit span | .16 | .39 | .55 | .01 | .44 | .45 |

Data from Finkel et al. (1995).

**Table 4   Major and Minor Schemes for Organizing Personality Traits**

| Major Schemes | | | Minor Schemes | | |
|---|---|---|---|---|---|
| Eysenck Big Three | Costa & McCrae NEO-PRF Big Five | Tellegen MPQ Big Three | Zuckerman (5) | Cloninger (7) | Hough Big Nine |
| Neuroticism | Neuroticism | Negative emotionality | Neuroticism-anxiety | Harm avoidance | Adjustment |
| Anxious | Anxiety | | | | |
| | Vulnerability | Stress reaction | | | |
| Depressed | Depression | | | | |
| Guilt-feeling | | | | | |
| Low self-esteem | | | | | |
| Tense | | | | | |
| Irrational | | Alienation | | | |
| Shy | Self-consciousness | | | | |
| Moody | | | | | |
| Emotional | Impulsiveness | | | | |
| Psychoticism | | | Aggression-hostility | Cooperativeness | Agreeableness |
| Aggressive | Hostility | Aggression | | | |
| Cold | | | | | |
| Egocentric | | | | | Rugged individ-ualism |
| Impersonal | Agreeableness | | | | |
| Antisocial | Altruism | | | | |
| Unempathic | Compliance | | | | |
| Tough-minded | Tendermindedness | | | | |
| | Straightforwardness | | | | |
| | Trust | | | | |
| | Modesty | | | | |
| | Conscientiousness | Constraint | | Self-directedness | Dependability |
| | Deliberation | | | | |
| | Dutifulness | | | | |
| Impulsive | Self-discipline | Control | | | Locus of control |
| | Order | | | | |
| | Competence | | | | |
| | Achievement striving | | | Persistence | Achievement |
| | | Traditionalism | | | |
| Extraversion | Extraversion | | | | |
| Sensation-seeking | Excitement seeking | Harm avoidance | Impulsive sensation seeking | Novelty seeking | |
| Venturesome | | | | | |
| Active | Activity | | | | |
| Surgent | | | Activity | | |
| Carefree | | | | | |
| | | Positive emotionality | | | |
| | | Achievement | | Reward dependance | |
| | | Social closeness | | | |
| Sociable | Gregariousness | | Sociability | | Affiliation |
| Lively | | | | | |
| Assertive | Assertiveness | | | | |
| Dominant | | Social potency | | | Potency |
| | Positive emotions | Well-being | | | |
| | Warmth | | | | |
| | Openness | | | | Intelligence |
| | Fantasy | | | Self-transcendence | |
| | Aesthetics | Absorption | | | |
| | Feelings | | | | |
| | Actions | | | | |
| | Ideas | | | | |
| | Values | | | | |

Some traits are scored in reverse.
Modified from Bouchard and Loehlin (2001).

**Table 5   Broad Heritabilities of Self-Report Measures of the Big Five Factors Based on Four Recent Twin Studies, a Comprehensive Review of Twin, Adoption, and Biological Kinships (Loehlin, 1992), and a Summary of the Earlier Twin Literature (Bouchard, 1997)**

| | Recent Twin Studies | | | | | Reviews | |
| | | | | | | Loehlin (1992) | Bouchard (1997) |
| Trait | Jang et al. (1996) (Canada) | Waller (1999) (US) | Loehlin et al. (1998) (US) | Riemann et al. (1997) (Germany) | Mean of the Four Recent Studies | Review of Kinships | Summary of Literature |
|---|---|---|---|---|---|---|---|
| Extraversion | .53 | .49 | .57 | .56 | .54 | .49 | .54 |
| Agreeableness | .41 | .33 | .51 | .42 | .42 | .35 | .52 |
| Conscientiousness | .44 | .48 | .52 | .53 | .49 | .38 | .40 |
| Neuroticism | .41 | .42 | .58 | .52 | .48 | .41 | .58 |
| Openness | .61 | .58 | .56 | .53 | .57 | .45 | .52 |
| MZ pairs | 123 | 313 | 490 | 660 | | | |
| DZ pairs | 127 | 91 | 317 | 304 | | | |

Five model and compared these results to a synthesis of twin, adoption, and family studies conducted by Loehlin (1992), as well as to Bouchard's (1997) reanalysis of the Nichols (1978) data. A summary of this comparison, given in terms of estimates of broad heritability, is given in Table 5. The Jang et al. (1996) and Reimann et al. (1997) studies used versions of the NEO (Costa and McCrae, 1992), a widely used self-report measure of the Big Five. Findings from both the Waller (1999) and Loehlin (1998) studies used different instruments, but the findings could be organized according to the Big Five (1998). In all of these studies shared environmental influence was estimated as zero or near zero.

Analyses based solely on twin samples (i.e., the four individual studies and the Bouchard review) consistently yield higher estimates of personality heritability than analyses based on twin, adoption, and family data (i.e., the Loehlin review). The difference may be due to nonadditive genetic effects (which contribute to the similarity of MZ twins but not to parent-offspring pairings; Plomin et al., 1998), although a variety of methodological and measurement problems (age at measurement, comparability of measures, sampling, etc.) cannot be ruled out. Combined model fitting of multiple kinships using the same instrument can help determine whether nonadditive genetic factors are operative.

## Combined Model Fitting and Sex Differences

Finkel and McGue (1997) fit models to MPQ data gathered from 12 sets of kinships (male-male, female-female, and male-female MZ and DZ twins and sib-ling pairs, and the four gender-specific parent-offspring pairings) totaling 4298 pairs. The participants were aged 17 years or older and drawn from the Minnesota Twin Family Registry (Lykken et al., 1990). A variety of sources of variance were tested for, including sex-limitation. The results are presented in Table 6 by sex, and nonadditive variance ($d^2$) is estimated for each trait. We have also added the mean broad-sense heritability (weighted mean of males and females), the simple correlation for MZT twins (weighted mean of males and females), and the MZA correlation from the MISTRA (made up of male and female twins) for comparative purposes. No significant shared environmental variance was detected for any scale and there was no evidence to suggest that different genetic factors influenced personality in males and females. There was, however, evidence for sex differences in heritability for three specific scales, Alienation, Control, and Absorption (shown in bold), but for none of the higher-order factors. For most of the specific and higher-order scales, estimates of nonadditive genetic effects were considerable (typically accounting for from 10 to 20% of the variance) and statistically significant.

Looking first at the higher-order factors, we see that Positive Emotionality (Extraversion) has a heritability of .50, virtually identical to Loehlin's (1992) synthesis estimate and only slightly below the mean of the four recent twin studies and the Bouchard (1997) summary. A recent study based on nearly 30,000 individuals in 80 distinct kinships (the Virginia 30,000) reported by Eaves and his colleagues (1999) reported heritability estimates for Extraversion of .50 in females and .43 in males, figures very close to those in Table 6. Notice that the MZT correlation

**Table 6   Variance Components and Broad Heritability for the Scales and Higher-Order Factors of the Multidimensional Personality Questionnaire and Intraclass Correlations for Monozygotic Twins Reared Together (MZT) and Apart (MZA)**

| | $a^2$ | $d^2$ | $e^2$ (Plus Error) | $h_b^2$ | Mean* $h_b^2$ | Intraclass Correlations MZT ($n = 626$ Pair) | MZA ($n = 74$) |
|---|---|---|---|---|---|---|---|
| Well-being | | | | | .40 | .45 | .50 |
| Females | .22 | .18 | .60 | .40 | | | |
| Males | .33 | .07 | .60 | .40 | | | |
| Social potency | | | | | .54 | .59 | .54 |
| Females | .30 | .24 | .46 | .54 | | | |
| Males | .38 | .15 | .47 | .53 | | | |
| Achievement | | | | | .36 | .37 | .33 |
| Females | .21 | .17 | .62 | .38 | | | |
| Males | .10 | .22 | .68 | .32 | | | |
| Social closeness | | | | | .46 | .49 | .44 |
| Females | .35 | .12 | .53 | .47 | | | |
| Males | .21 | .23 | .56 | .44 | | | |
| Stress reaction | | | | | .44 | .45 | .47 |
| Females | .23 | .22 | .55 | .45 | | | |
| Males | .39 | .03 | .58 | .43 | | | |
| **Alienation** | | | | | .47 | .45 | .35 |
| Females | .16 | .23 | .61 | .39 | | | |
| Males | .25 | .36 | .39 | .61 | | | |
| Aggression | | | | | .38 | .38 | .40 |
| Females | .18 | .21 | .61 | .39 | | | |
| Males | .05 | .30 | .65 | .35 | | | |
| **Control** | | | | | .38 | .41 | .46 |
| Females | .20 | .13 | .67 | .33 | | | |
| Males | .02 | .45 | .53 | .47 | | | |
| Harm avoidance | | | | | .45 | .47 | .45 |
| Females | .22 | .23 | .55 | .45 | | | |
| Males | .27 | .19 | .54 | .46 | | | |
| Traditionalism | | | | | .54 | .61 | .52 |
| Females | .47 | .08 | .45 | .55 | | | |
| Males | .52 | .00 | .48 | .52 | | | |
| **Absorption** | | | | | .38 | .42 | .56 |
| Females | .29 | .15 | .56 | .44 | | | |
| Males | .11 | .15 | .74 | .26 | | | |
| Mean | .25 | .19 | .57 | .44 | .44 | .46 | .46 |

**Higher-Order Factors**

| | $a^2$ | $d^2$ | $e^2$ (Plus Error) | $h_b^2$ | Mean* $h_b^2$ | MZT | MZA |
|---|---|---|---|---|---|---|---|
| Positive emotionality | | | | | .50 | .55 | .43 |
| Females | .31 | .17 | .52 | .48 | | | |
| Males | .29 | .24 | .47 | .53 | | | |
| Negative emotionality | | | | | .44 | .44 | .47 |
| Females | .25 | .17 | .58 | .42 | | | |
| Males | .29 | .19 | .52 | .48 | | | |
| Constraint | | | | | .52 | .56 | .58 |
| Females | .44 | .09 | .47 | .53 | | | |
| Males | .23 | .27 | .50 | .50 | | | |
| Mean | .30 | .19 | .51 | .49 | .49 | .52 | .50 |

Note: $a^2$ = additive genetic variance; $d^2$ = dominance variance; $h_b^2$ = broad heritability ($a^2 + d^2$); $e^2$ = nonshared environmental variance including measurement error.

Traits in boldface show a significant sex difference in heritability.

\* Weighted mean of males and females.

Adapted from Finkel and McGue (1997), with permission.

**Table 7   Comparison of Genetic and Environmental Parameter Estimates for Neuroticism in the Virginia 30,000, the Combined Virginia and Australia Extended Family Kinships, and the Minnesota Extended Kinship**

| Sources of Variance | Virginia 30,000 | | Combined Virginia and Australia | | Minnesota | |
|---|---|---|---|---|---|---|
| | Males | Females | Males | Females | Males | Females |
| Genetic | | | | | | |
|   Additive | .169 | .274 | .25 (.21–.29) | .28 (.24–.31) | .29 | .25 |
|   Assortment* | .001 | .002 | .06 (.04–.08) | .06 (.04–.08) | | |
|   Nonadditive | .258 | .228 | .10 (.04–.15) | .13 (.09–.18) | .19 | .17 |
|   Total | .423 | .504 | .41 | .47 | .48 | .42 |
| Environment | | | | | | |
|   Parental | .050 | .000 | | | | |
|   Shared | .019 | .000 | | | | |
|   Nonshared | .553 | .490 | .65 (.61–.69) | .58 (.56–.61) | .52 | .58 |
| G-E covariance | .019 | .006 | | | | |

    * Assortment variance is included in the additive variance.

alone only slightly overestimates the heritability and the MZA correlation only slightly underestimates it. The MZT minus MZA correlation would suggest some shared environmental influence on Extraversion, but for the large confidence interval around the MZA correlation (.23 to .60), which precludes drawing any claims of statistical significance for this difference. Absence of a shared environmental effect on Extraversion is indicated both by the model fitting of the entire data set and shared environment estimates of .00 (females) and .02 (males) in the large Eaves et al. (1999) study.

Negative Emotionality (Neuroticism), the second higher-order dimension, yields a heritability of .44, which is identical to the MZT correlation. The slightly, but not significantly, higher MZA than MZT correlation suggests that, like Extraversion, there is no shared environmental influence on Negative Emotionality, a result consistent with the full analysis. The results are very similar to the estimate from the Loehlin synthesis and the mean for the four recent twin studies, but somewhat below the estimate provided by the Bouchard (1997) twin meta-analysis. There are two other very informative comparisons available for Neuroticism. One is from the Eaves (1999) study cited above, and the other is from a combined sample that included the Virginia 30,000 used by Eaves et al. and a large extended twin kinship sample from Australia (20,945 members) (Lake et al., 2000). There was no heterogeneity between the two samples, and a simple model fit the combined data set very well. The results of the three studies are shown in Table 7.

Table 7 illustrates how difficult it is to accurately estimate nonadditive genetic variance. Even though the various studies agree on the overall contribution of genetic factors, there are clear differences in the proportion of genetic variance accorded to nonadditive

effects. The 95% confidence intervals for nonadditive genetic variance based on the very large combined sample do not include the estimates from the Virginia 30,000 sample (which makes up about half the sample from which the estimates are derived) nor those from the Minnesota male sample, and they barely include the estimate from the Minnesota female sample. Notice also that parental environmental and G-E covariance effects, which can be estimated with these large kinships, are estimated to be very small. Combined data on the Extraversion factor have not been published. Turning finally to Constraint (Table 6), the third MPQ factor, we see that the heritability estimate is close to the MZT correlation, which, when compared to the MZA correlation, suggests no shared environmental influence, a result consistent with the model fitting. We do not believe that a direct comparison of Constraint with either Psychoticism or Conscientiousness is appropriate.

A comparison of the overall heritabilities, and the MZT and MZA correlations for the specific scales of the MPQ, is also informative. The fact that the MZT correlations very closely approximate the heritabilities flows directly from the failure to find shared environmental influence and is a consistent finding for most personality traits. The similarity between the MZT and MZA correlations independently confirms the lack of shared environmental influence and the broad heritability estimates. In the personality domain the MZT correlation alone provides an excellent approximation to the heritability of a trait.

## What Are the Specific Genetic Factors that Influence Personality?

Given that personality is both an essential feature of mental health and has consistently been shown to be

heritable, there has been considerable effort directed at identifying the specific genes that contribute to individual differences in personality. To date, these efforts have not produced confirmed and replicable findings. The most widely studied genetic polymorphism in personality is a variable repeat sequence in the third exon of the dopamine D4 receptor (DRD4; Tarazi and Baldessarini, 1999). DRD4 has been targeted both because of the role of dopamine in brain reward and approach systems, and because the variable number of tandem repeat (VNTR) polymorphisms may be biologically functional. Both Benjamin et al. (1996) and Ebstein et al. (1996) reported significantly higher scores on novelty seeking (NS) for individuals carrying at least one copy of the 7-repeat allele than those with no copies of this allele. Although this initial evidence appeared very promising, there followed a bewildering array of studies that both replicated and failed to replicate the original reports. McGue (2002) reported a meta-analysis of association studies of DRD4 and NS and concluded that, although the overall effect of carrying the 7-repeat allele was statistically significant, there is substantial and unexplained heterogeneity in findings reported by different investigators.

A second polymorphism that has received considerable attention in the personality field is a functional variant (long versus short) in the promoter region of the gene that codes for the serotonin transporter (5-HTTLPR). Lesch et al. (1996) reported that individuals with at least one copy of the short allele scored significantly higher on a measure of neuroticism than individuals who carried two long alleles. As was the case with DRD4, the initial positive finding was followed by a series of replications and failures to replicate the initial association. In his meta-analysis, McGue (2002) reports that the overall effect of the 5-HTTLPR polymorphism on neuroticism is not statistically significant, although again there is marked heterogeneity of findings across studies.

At this point, it is difficult to determine why association studies in personality have yielded a heterogeneous set of findings. Failures to replicate may reflect the limitation of standard genetic association studies when there are ethnic stratification effects. Alternatively, failures to replicate could be due to nonadditive genetic influences such that the effect of a single polymorphism may depend on genetic background, and the latter may vary from sample to sample. Despite the failure to find confirmed associations, the search for specific genes in personality is likely only to intensify as additional behaviorally relevant genes are identified through the Human Genome Project.

## What Are the Nonshared Environmental Influences on Personality?

Analyses of twin, family, and adoption data are overwhelmingly consistent in implicating both genetic and environmental contributions to personality. These studies are also consistent in indicating that the predominant sources of environmental influence correspond to factors that produce personality differences (i.e., nonshared) rather than similarities (i.e., shared) between reared-together relatives. Surprisingly, the specific nonshared environmental factors that influence personality have been extremely difficult to identify. A recent meta-analysis led to the conclusion that nonshared environmental influences are probably quite complex and will "remain outside the domain of systematic scientific investigation for a long time" (Turkheimer and Waldron, 2000, p. 93).

The challenges in identifying nonshared environmental influences are nicely illustrated by the NEAD (Nonshared Environment in Adolescent Development) study, which was designed specifically to study this question (Reiss et al., 2000). It utilized a national sample, six genetically informative kinship designs, and multiple sources of data on most variables, thereby creating "consensus trait measures" for six traits. The average heritability of these traits was .68 and the nonshared environmental contribution was .17. The mean reliability of the six measures was, however, .81, and thus measurement error could account for all of the nonshared environmental variance. The average effect associated with shared environmental factors was .15, although this effect was large for only two of the six traits (see Bouchard and Loehlin, 2001, Table 6). It is important to recognize that the NEAD consensus traits involved self-report and observational measures gathered over different time periods (days if not weeks apart) to avoid specific sources of variance in each measure and specific sources of irrelevant influence (unreliability) present at particular points in time. Composites thus formed likely yield more valid, more reliable, and more heritable measures than single-time self-reports. Another possible source of nonshared environmental variance in ordinary personality traits is chance or developmental noise (Molenaar et al., 1993; Finch and Kirkwood, 2000). It may well be that so many minor chance and idiosyncratic factors intervene between conception and the full manifestation of psychological phenotypes that a meaningful portion of the variance will never be accounted for by specific effects that generalize across individuals.

In summary, these studies paint a rather simple picture of genetic and environmental influence on

personality. Genetic influences account for approximately 40–55% of the variance in personality. Some of the genetic effects appear to be nonadditive genetic variance, although it is difficult to precisely estimate such effects. There appear to be sex differences in heritability, but they are infrequent and probably not large, and the same genes appear to operate on all traits in both sexes. To date, no confirmed associations of personality with specific genetic polymorphisms have been reported, although there are several promising leads.

These conclusions can be generalized, of course, only to new populations exposed to a range of environments similar to those studied. There is a tendency to characterize the range of environments in which ordinary people spend most of their lives as benign and nontraumatic. This is simply untrue, as a brief chat with any practicing clinical psychologist or psychiatrist will quickly reveal. Nevertheless, the extremes of poverty and affluence are not being sampled as adequately as they should by many if not most research studies, and therefore it is not possible to generalize findings to the entire population of whatever group of people is being studied. Better sampling of populations would be highly desirable, but the extent to which better studies would change the findings is an empirical question, not one to which we can assume the answer.

## SOCIAL ATTITUDES

### Genetic Influence on Social Attitudes

Attitudes are often defined as learned predispositions, positive or negative, towards some object (person, institution, situation). Social attitudes generally refer to predispositions towards socially relevant policies (abortion), institutions (the Federal Government), types of people (Blacks, Jews, Arabs, Democrats, Communists, Socialists), and so forth. Unlike the domains of abilities, personality, and interests, there is no widely agreed upon core set of variables, instruments, or approaches in the social attitude field (Eagly and Chaiken, 1993). Given the widespread belief that attitudes are learned, the notion that they might be genetically influenced will strike some readers as highly unlikely. Indeed, several early behavioral genetic studies included measures of social attitudes as control variables for which no genetic influence was expected. These expectations were not met, as social attitudes were found to be partly heritable (e.g., Scarr, 1981), leading prominent attitude researchers to entertain the possibility that social attitudes might be

genetically influenced (Eagly and Chaiken, 1993, p. 3). Today, rather than being dismissed as preposterous, the hypothesis of heritable influences on attitudes has been embraced by some social psychologists engaged in basic research on the nature and origins of social attitudes (Tesser, 1993; Crelia and Tesser, 1996). The first published report on the heritability of social attitudes was by Eaves and Eysenck (1974). Eysenck's Public Opinion Inventory, which measures two factors, Radicalism versus Conservatism (R) and Toughmindedness versus Tendermindedness (T), and an 80-item personality inventory, were administered to 451 MZ and 257 DZ twin pairs (preponderantly females). R yielded a heritability of .65 and was entirely independent of the personality measures. T yielded a heritability of .54 and had a small correlation with the personality trait of Extraversion. This groundbreaking report was virtually ignored and had very little influence on the field. About the time of the Eaves and Eysenck report, Scarr and Weinberg (1978) were carrying out the adoption study cited earlier. These investigators included the California F-scale (F for Fascism) in their assessment of adoptive and biological families. The F-scale had been developed by a team of researchers interested in the rise of Nazism, who published their findings in a landmark book—*The Authoritarian Personality* (Adorno et al., 1950). The F-scale had been the focus of a very large body of research but eventually grew to be disfavored, primarily because of its very high negative correlation with IQ (Christie and Jahoda, 1954; Stone et al., 1993). In their adoption study, Scarr and Weinberg (1981) also found that individuals who were high in verbal ability tended to score low on the F-scale. When she adjusted statistically for the F-scale's association with verbal ability, however, the heritability of the F-scale remained statistically significant, even if diminished. Remarkably and unexpectedly, heritable influences on authoritarianism could not be accounted for by heritable influences on cognitive ability.

Martin et al. (1986) administered the 50-item version of the Wilson-Paterson Conservatism scale (WPCS) to a large sample of MZ, like-sex DZ, and unlike-sex DZ twins from the Australian twin registry. The WPC scale uses a "catch phrase" format, whereby respondents are asked to indicate whether they agree with various topics (e.g., death penalty, X-rated movies, women's liberation, foreign aid, abortion, etc.) by simply circling YES, ?, or NO. The heritability of the WPC scale was estimated at .62 in a model-fitting analysis that took into account assortative mating as estimated in a separate husband-wife sample. Perhaps even more remarkable than the estimate of significant heritable influences was the find-

**Table 8   Intraclass Correlations for Right Wing Authoritarianism (RWA) for MZA, DZA, MZT, and DZT Samples without Controlling for General Cognitive Ability (GCA), and the MZA and DZA Correlations with GCA Partialed Out**

| | Kinship Group | | | |
|---|---|---|---|---|
| Variables | MZA | DZA | MZT | DZT |
| RWA | .69 | .00 | .63 | .42 |
| | (.48–.82) | (−.31–.33) | (.57–.68) | (.34–.49) |
| RWA partialing out GCA | .59 | −.09 | NA | NA |
| | (.34–.76) | (−.39–.23) | | |

NA: Not available for reared-together twin sample.
95% Confidence intervals for intraclass correlations.
MZA, $n = 39$; DZA, $n = 38$; MZT, $n = 423$; DZT, $n = 434$.

ing that shared environmental factors exerted no influence on social attitudes once the effects of assortative mating had been taken into account. Unfortunately, because these investigators did not include measures of personality and ability in their assessment battery, they could not determine the extent to which heritable influences on social attitudes were independent of heritable influences in these other domains. Nonetheless, their findings led them to the strong prediction that the conservatism scores of reared-apart MZ twins would be correlated .62. Martin et al.'s conclusion that social attitudes are moderately to strongly heritable has been supported in several subsequent investigations. In the MISTRA, the intraclass correlation for a 28-item version of the WPCS was .59 for MZA ($n = 54$ pairs) and .21 for DZA ($n = 46$ pairs) twins. The spouse ($n = 93$ pairs) correlation was .60. Model-fitting analyses yielded a highly significant heritability estimate of .56 (95% confidence interval .38–.70). This heritability is quite close to the value of .62 reported by Martin et al. (1986) using a longer and therefore more reliable version of the WPCS. Interestingly, statistical refinement of the WPCS measure in the MISTRA sample (dropping a few items that did not load on the first principal component) yields an MZA correlation of .62, a DZA correlation of .29, and a spouse correlation of .60 (heritability estimate of .60–95%, confidence interval of .43–.73; Bouchard, 2002). Eaves et al. (1999) also replicated the Australian twin study findings in a model-fitting analysis of data from the Virginia 30,000 (using 80 kinships of twins and their relatives). They reported an average heritability of .55 (.65 for males, .45 for females) for the same 28-item version of the WPCS used by Bouchard et al. (2002).

A study by McCourt et al. (1999) that assessed Right Wing Authoritarianism (RWA) in both a sample of reared-apart twins from the MISTRA and a sample of reared-together twins from the Minnesota Twin Registry provided an opportunity to test Martin et al.'s conclusions concerning both the existence of heritable and the lack of shared environmental influences on a social attitude measure other than the WPCS. The twin correlations from this study are given in Table 8. Because RWA was correlated .37 with IQ in the MISTRA sample (IQ was not assessed in the reared-together twin sample), the reared-apart twin correlations were adjusted for IQ. This adjustment had only a modest effect on the MZA and DZA correlations.

There is little difference in the MZA and MZT correlations for RWA, suggesting little shared environmental influence. Among DZ twins, however, reared-together twins were more similar than reared-apart twins, suggesting that shared environmental influences are important for this class of relatives. Sample sizes were, however, small, and associated confidence intervals were wide, suggesting the need for caution in interpreting individual correlation coefficients. Optimal weighting of the combined data was achieved through model-fitting analyses that took into account assortative mating effects. Two models were found to fit the combined data equally well: a model that posited that all twin similarity was owed to genetic factors ($h^2 = .64$); and a model that posited the existence of both genetic ($h^2 = .50$) and shared environmental ($c^2 = .16$) contributions. Thus, while this study of reared-apart and reared-together twins cannot entirely rule out the existence of modest shared environmental influences, it provides further confirmation of the trends first noted by Martin and his colleagues. An important feature of social attitudes is that they can vary substantially by age. Figure 9 shows the mean WPC scores as a function of age and educational attainment for the Virginia 30,000 sample.

Beginning after age 30, mean WPC scores increase independently of educational level. In a large twin study, Eaves et al. (1997) investigated the implication of age changes in the WPC for estimates of genetic
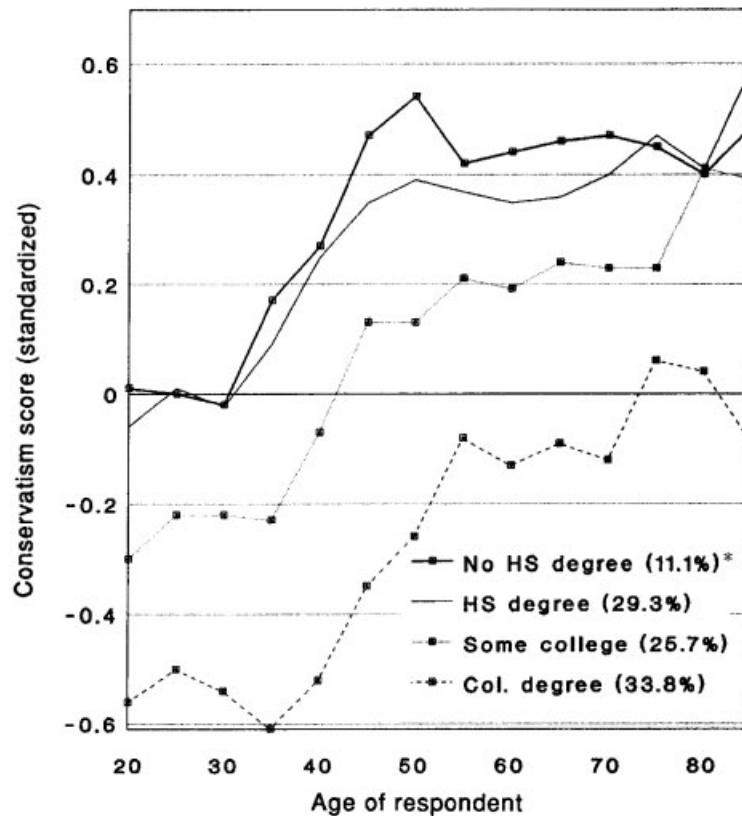
**Figure 9**   Mean conservatism scores, for all respondents, grouped by age and educational level. HS, high school; col, college. *Percent of sample within education level. From Truet (1993).

and shared environmental influences. Their findings are summarized in Figure 10. This figure illustrates two important points. First, genetic factors have little influence on WPC scores prior to age 20, but substantial influence after age 20. Second, there is considerable variation in the size of the differences between the MZ and DZ twins from point to point, reflecting both the effect of varying sample sizes and chance differences in the populations sampled.

## Religious Affiliation and Religiousness

Twin studies of religious affiliation (e.g., Christian, Jewish, Muslim) have shown that variance in this trait is nearly completely environmental in origin, thus demonstrating that model-fitting is not intrinsically biased and can indeed show no genetic effects when that is the case (Eaves et al., 1990). Alternatively, frequency of church attendance, an aspect of religiousness, appears to be genetically influenced. Using the Virginia 30,000 sample (Americans representing 80 distinct kinship pairings), Maes et al. (1999) reported that 25 to 42% of the variance (depending on sex) in religious attendance was heritable, while 14 to

34% of the variance was associated with shared environmental effects.

D'Onofrio et al. (1999) have summarized much of the behavioral genetic literature on *adult* religiousness through 1998. After updating reports for MISTRA based on our recent results, we have reproduced their findings for strictly religious measures in Table 9. The Martin et al. (1986) assessment of religiousness is based on single items. Items are known to be less reliable than scales composed of multiple inter-related items. Despite limitations in the assessment of religiousness, Martin et al. (1986) still found, on average, significant genetic (.28) and shared environmental (.25) influence. The MISTRA scales all suggest a moderate heritability for religiousness (mean = .47). For the three scales from which data were available from both reared-together and reared-apart twins, heritability estimates based on the reared-together data only [i.e., $h^2 = 2(MZ − DZ)$] and heritability estimates based only on the MZA correlation led to precisely the same conclusion. Although the DZA correlations appear inconsistent, they are not statistically incompatible with the summary heritability estimates due to the small DZA sample size.
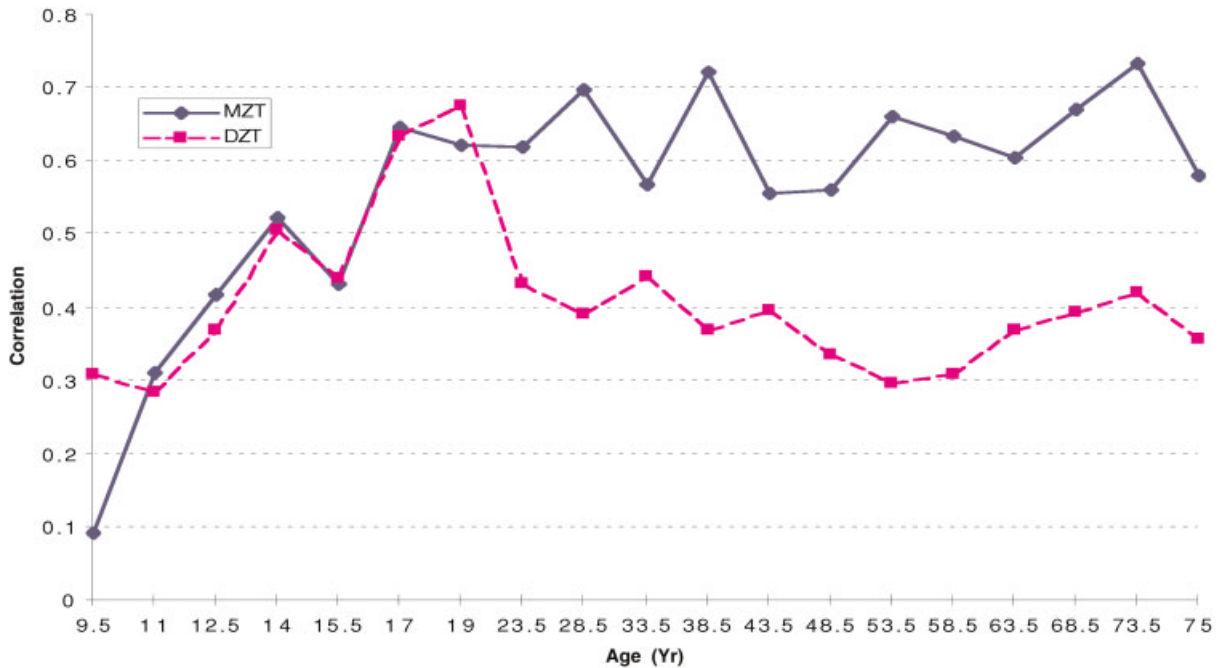
**Figure 10**  Twin correlations for conservativism by age (pooled over sexes). From Eaves et al. (1997).

This is an excellent context in which to address one of the cautions mentioned in the introduction; namely that the correlation between child rearing practices and offspring characteristics can be confounded with genetic factors. Participants in MISTRA are asked to complete a number of "environmental measures", one

**Table 9  Twin Correlations and Parameter Estimates for Measures of Religiousness Based on Adult Twin Studies and One Adoption Study**

| Measure | MZT | DZT | MZA | DZA | $h^2$ | $c^2$ | $e^2$ | Reference |
|---|---|---|---|---|---|---|---|---|
| Items from WPC | | | | | | | | |
| Sabbath observance | .51 | .35 | — | — | .35 | .18 | .47 | Martin et al. (1986) |
| Divine law | .49 | .38 | — | — | .22 | .26 | .52 | Martin et al. (1986) |
| Church authority | .48 | .35 | — | — | .29 | .20 | .51 | Martin et al. (1986) |
| Bible truth | .58 | .46 | — | — | .25 | .34 | .41 | Martin et al. (1986) |
| MISTRA-scales (# of items) | | | | | | | | |
| Religious leisure time interests (6) | .58 | .28 | .50 | .12 | .57 | — | .44 | Bouchard (2002) |
| Religious occupational interests (4) | .43 | .23 | .55 | .09 | .44 | — | .56 | Bouchard (2002) |
| MMPI religious fundamentalism (10) | .56 | .32 | .55 | .01 | .54 | — | .46 | Bouchard (2002) |
| Intrinsic religiousness (9) | — | — | .37 | .20 | .43 | * | .57 | Bouchard et al. (1999) |
| Extrinsic religiousness (11) | — | — | .24 | .38 | .39 | * | .61 | Bouchard et al. (1999) |
| SCII religious interests | — | — | .42 | .32 | .43 | * | .57 | Bouchard (2002) |
| AVL religious value | — | — | .50 | .13 | .46 | * | .64 | Bouchard (2002) |
| Religious orthodoxy (adoption data) | — | — | — | — | .28 | .26 | — | Beer et al. (1998) |
| Personal devotion | .52 | .40 | — | — | .29 | .24 | .47 | Kendler et al. (1997) |
| Religious salience | .72 | .56 | — | — | .29 | .42 | .29 | Carver and Ury (1997) |

Abbreviations are: MZT = monozygotic twins reared together, DZT = dizygotic twins reared together, MZA = monozygotic twins reared apart, DZA = dizygotic twins reared apart, $h^2$ = additive genetic variance estimate, $c^2$ = shared environmental variance estimate, $e^2$ = unshared environmental variance estimate plus error, WPC = Wilson-Patterson Conservatism, MISTRA = Minnesota Study of Twins Reared Apart, MMPI = Minnesota Multiphasic Personality Inventory, SCII = Strong-Campbell Interest Inventory, AVL = Alport, Vernon, Lindsey Study of Values.

* $c^2$ cannot be estimated with only twins reared apart; the estimate of $c^2$ in these studies reflects both shared and nonshared influences.

of which is the Moral Religious Emphasis (MRE) scale of the Family Environment Scales (Moos and Moos, 1994). This instrument requires a retrospective report of family/parental behavior while the respondent was growing up (Hur and Bouchard, 1995). For study participants who were reared by their biological parents (mostly spouses of the twins) the correlation between MRE and Intrinsic Religiousness is .53. For adopted individuals the correlation is only .10. This comparison is supportive of strong genetic and weak environmental influence on the trait. Although there was a tendency for MZA twins to be placed in rearing homes with similar levels of MRE ($r = .32$), this placement effect contributes only .003 to the MZA correlation because of the weak correlation between the MRE and Intrinsic Religiousness in adoptive families. The focus in the previous section has been on adult religiousness. As with social attitudes, genetic influence on religiousness is attenuated in younger samples. Winter et al. (1999), using the MMPI Religious Fundamentalism scale, reported heritabilities of .11 and .22 and shared environmental effects of .60 and .45 for Finnish *adolescent* girls and boys (16 years olds), respectively. Boomsma et al. (1999) also reported little genetic influence on three measures of religion [Religious Upbringing (no/yes), Religious Affiliation, Participation in Religious Activities (none, am religious but do not participate, am an active member of the church)] in a sample of adolescent (18 year old) twins. These measures do not directly address religiousness as a trait, but the last one should have at least a modest correlation with direct measures, and these results with adolescents stand in clear contrast with findings on adults. It will be interesting to see what happens when direct measures of religiousness are gathered on these individuals in adulthood.

The most recent twin study (MZ = 195 pairs, DZ = 141 pairs) of genetic influence on attitudes (Olson et al., 2001) utilized 30 heterogeneous items (not selected to measure one or more underlying factors). They found a mean item $h^2$ of .32, with 26 of the items yielding significant estimates of genetic influence. A factor analysis of the items yielded nine factors. Three factors had heritabilities of zero. The remaining six factors had a mean heritability of .50 and a mean shared environmental influence of .04. The item "Organized Religion" had a heritability of .45 and a shared environmental influence of .00. A few of these heritabilities were mediated by personality factors (measured by very brief self-report), as it was possible to demonstrate that some attitudes and some personality measures shared genetic variance. The authors concluded that their findings were consistent with previous behavior genetic studies of attitudes.

As Eaves et al. (1999) have pointed out: "The significant contribution of genetic factors to social attitudes means that virtually no measurable aspect of human behavioral variation is so far removed from the impact of events at the genetic level as to be considered in complete isolation from the emerging theory and knowledge in genetics and sociobiology" (p. 79). Critics of this kind of work argue that there simply cannot be genes for attitudes or genes for religiousness. We argue, why not? As Dawkins (2000) has pointed out, it is easy to restate the idea of a gene for religion as a "gene for developing the kind of brain that is predisposed to religion when exposed to a religious culture". Genes guide the construction of brains and we know that transcranial magnetic stimulators can elicit religious feelings even in the brains of the nonreligious (Ramachandran and Blakeslee, 1998, Chapter 9).
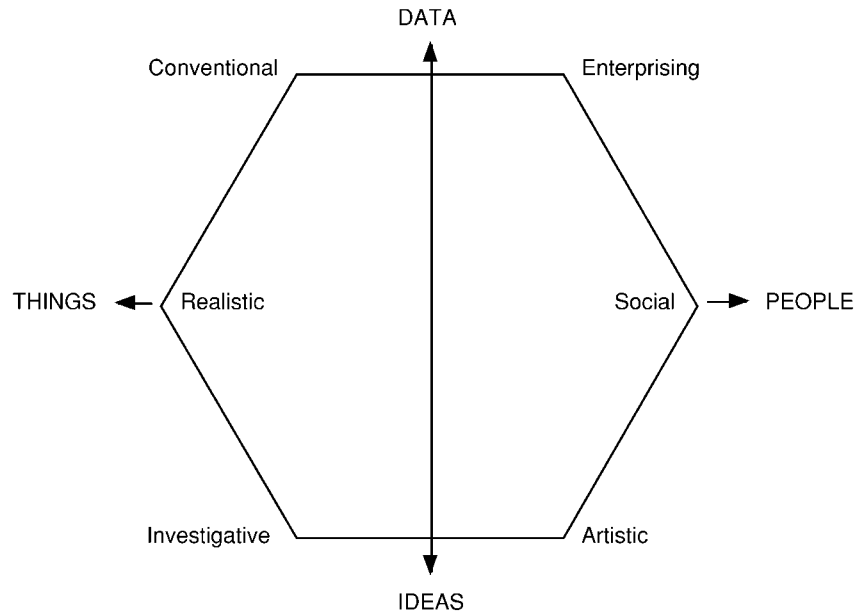
## PSYCHOLOGICAL INTERESTS

### Interests and Work Values

In industrialized societies people spend a very significant percentage of their lives at work. In order to aid people in the making of a satisfying vocational/occupational choice, psychologists have developed a number of elaborate technologies and theories of vocational interests (Lubinski, 2000).

Many psychologists, as well as most lay people, believe that vocational interests are no more than a manifestation of personality traits. To make matters worse the major theorist in this domain calls his theory a "Theory of Vocational Personalities" (Holland, 1997). In actuality, there is little overlap between interests and personality. Waller et al. (1995) reported correlations between the 11 primary scales of the MPQ and 17 occupational interest scales from the Minnesota Occupational Interest Inventory (discussed below) in a sample of 4000 adults. Of the 187 correlations reported, only nine were equal to or greater than .30. Hansen (1984), on reviewing the larger literature concluded that "for the most part, correlational studies between interest scores and personality scores have been extremely disappointing" (p. 117). The domains of personality measurement and vocational interest measurement are quite distinct.

### Structure of Vocational Interests

There is not the same degree of consensus regarding the structure of vocational interests as there is in the

DATA

Conventional                    Enterprising

THINGS ◄─── Realistic          Social ───► PEOPLE

Investigative                   Artistic

IDEAS

**Figure 11**   The Holland Hexagon (which characterizes six types of people) shown against two broad dimensions of interests (Things vs. People; Data vs. Ideas).

domains of ability and personality. The dominant view is that of Holland's hexagonal model, shown in Figure 11. In simple terms, Holland's theory purports that there are six major general interest factors (often called Themes), and the correlations between the various types are "inversely proportional to the theoretical relationships between them" (Holland, 1985, p. 5). The two broad dimensions of Things versus People and Ideas versus Data, widely discussed by vocational interest researchers (Lippa, 1998) and implicated in sex differences in interests, are also shown in Figure 11. The competing model of vocational interests is a hierarchical one much like that found in the ability and personality domains (Gati, 1991). The hierarchical model has attracted much less empirical support than the Holland hexagonal model (Rounds, 1995; Day and Rounds, 1998; Day et al., 1998).

## Twin Studies of Vocational Interests

Early twin studies of vocational interests were few in number, generally had modest sample sizes, and used a variety of instruments and statistical methods for estimating genetic influences. These studies were nicely organized and subjected to a meta-analysis by Nichols (1978). His results are shown in Table 10.

Nichols grouped interest measures in a manner that is quite similar to the Holland themes (Practical = Realistic, Science = Investigative, Business = Enterprising, Clerical = Conventional, Helping = Social).

The mean intraclass correlation is .48 for MZT twins and .30 for DZT twins. The associated mean Falconer heritability estimate [i.e., $2(r_{MZ} - r_{DZ})$] is .36 (Falconer, 1960).

The Minnesota Twin Registry (1990) includes in its assessment battery the Minnesota Occupational Interest Inventory and the Minnesota Leisure Time Inventory. The two inventories yield 17 occupational interest scales and 18 leisure interest scales that are highly inter-related (Lykken et al., 1993; Waller et al., 1995, Table 3). Waller et al. (1995) have reported boxplots of intraclass correlations for both instruments, for both MZ and DZ twins by sex. They are shown in Figure 12. The results are remarkably similar to those reported by Nichols, although the DZ

**Table 10   Mean Intraclass Correlations from Twin Studies for Vocational Interests by Twin Type**

|                              | Twin Type | |
| --- | --- | --- |
| Interest Dimension           | MZ  | DZ  |
| Practical (Conventional)     | .50 | .37 |
| Science (Investigative)      | .54 | .29 |
| Business (Enterprising)      | .45 | .30 |
| Clerical (Conventional)      | .44 | .26 |
| Helping (Social)             | .48 | .30 |
| Artistic (Art)               | .50 | .32 |
| Mean of all interests        | .48 | .30 |

From Nichols (1978).
Holland names are in parentheses.
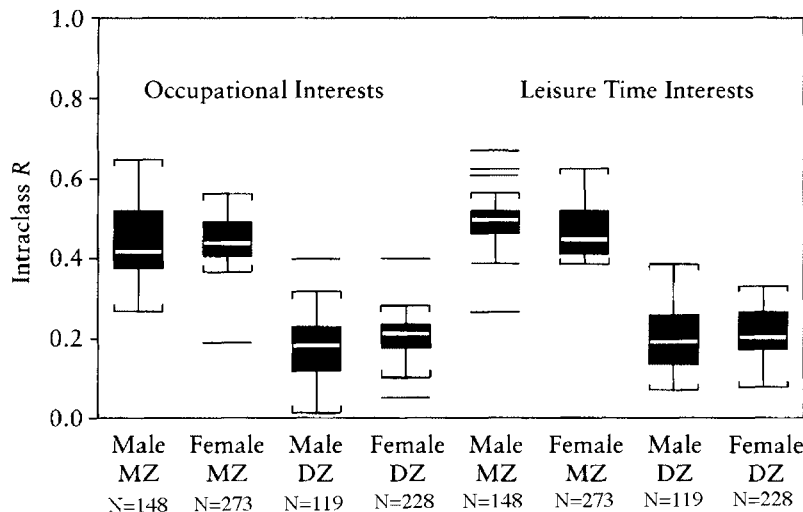
**Figure 12**   Box plots of MZ and DZ intraclass correlations for 17 occupational interest and 18 leisure time interest scales by sex. From Waller et al. (1995). Modified and reproduced by special permission of the Publisher Davies-Black Publishing a division of CPP, Inc., Palo Alto, CA 94303 from *Assessing Individual Differences in Human Behavior* by David Lubinski & Rene V. Dawis, Editors. Copyright 1995 by Davies-Black Publishing, a division of CPP, Inc. All rights reserved. Further reproduction is prohibited without the Publisher's written consent.

correlations are a little lower, implying somewhat higher heritabilities (between .40 and .50).

## Combining Twin, Adoption, and Family Studies

There are only a few adoption and family studies of vocational interests, but all of them have used various versions of the Strong Interest Inventory (Campbell, 1971; Hansen and Campbell, 1985; Harmon et al., 1994). The Strong is one of only a few psychological inventories that has undergone regular revision to keep it up to date. As a result, item content on this scale has changed somewhat over the years. It is, however, possible to use the overlapping items on the various forms to score a common set of scales (Hansen, 1982). It is important to recognize that correlations with the common scales can be expected to be attenuated because these scales are shorter and thus somewhat less reliable than the full-length scales. Betsworth et al. (1994) combined data from multiple twin, adoption, and family studies, and scored and analyzed the common interest scales. The twin and family correlations from their analyses are given in Table 11. Their model-fitting results are given in Table 12. The sample of twins reared together (from the National Merit Scholarship Twin Study) dominates the data set in terms of sample size. The sample of reared-apart twins has the smallest sample sizes (particularly the DZA sample). Nevertheless, on av-

**Table 11   Interest Scale Correlations for Twins Reared Apart, Twins Reared Together, Adoptive Families, and Biological Families**

|  | MZA $n = 59$ | DZA $n = 33$ | MZT $n = 1960$ | DZT $n = 1212$ | Adopted Parent*Off $n = 283$ | Adopted Siblings $n = 63$ | Biological Parent*Off $n = 332$ | Biological Siblings $n = 60$ |
|---|---|---|---|---|---|---|---|---|
| Realistic | .20 | .15 | .49 | .20 | .17 | .17 | .11 | −.10 |
| Investigative | .39 | .00 | .46 | .24 | .11 | .05 | .19 | .12 |
| Artistic | .23 | .17 | .51 | .29 | .09 | −.11 | .19 | .33 |
| Social | .42 | .04 | .45 | .20 | .08 | .10 | .15 | .17 |
| Enterprising | .41 | .04 | .41 | .25 | .07 | .23 | .14 | .11 |
| Conventional | .24 | .10 | .49 | .22 | .12 | .17 | .00 | .20 |
| Mean | .32 | .08 | .47 | .23 | .11 | .10 | .13 | .14 |

From: Betsworth et al. (1993).

**Table 12    Proportion of Variance in Interest Measures Attributed to Additive Genetic, Nonadditive Genetic Shared Environmental, and Nonshared Environmental Effects**

| Scale | Additive Genetic | Nonadditive Genetic | Shared Environmental | Nonshared Environmental and Error |
|---|---|---|---|---|
| Realistic | .00 | .36 | .12 | .52 |
| Investigative | .17 | .19 | .10 | .54 |
| Artistic | .23 | .16 | .12 | .49 |
| Social | .13 | .25 | .08 | .55 |
| Enterprising | .15 | .16 | .11 | .59 |
| Conventional | .00 | .38 | .11 | .51 |
| Mean | .11 | .25 | .11 | .53 |

From: Betsworth et al. (1993).

erage the MZA twin correlation (.32), which reflects both additive and nonadditive genetic variance (broad heritability), comes close to the overall mean estimate of .36 for the broad-sense heritability. The twins reared together, if taken alone, however, suggests a mean heritability of .47, additive genetic effects only, and no shared environmental effects. The apparent discrepancy between the results from the reared-together and reared-apart twins is due to two factors: there is consistent evidence for shared environmental effects (i.e., reared-apart twins are less similar than reared-together twins and adopted relatives have similar, albeit modestly similar, interest patterns); and once these shared environmental effects have been taken into account, there is evidence for nonadditive genetic effects (e.g., MZA twins are more than twice as similar as DZA twins).

Additional behavior genetic research on vocational interests is clearly needed. In particular, the offspring in the various adoption and family studies reviewed above were still teenagers at the time of their interest assessment. Interests have not fully crystallized by that age, and it may be that the heritability of interests, like the heritability of IQ, increases with age. It would be interesting to see data on the same kinships when everyone was measured at the same age.

## Twin Studies of Work-Related Behavior

Arvey et al. (1989) reported a study of intrinsic (i.e., perceived benefits from a job) and extrinsic (i.e., objective benefits of employment) job satisfaction using MZA twins (*n* = 34). They predicted that because intrinsic satisfaction most likely reflects personal and internal factors it would show a higher heritability than extrinsic satisfaction, which is generally thought to be controlled by external factors. The correlation for intrinsic satisfaction was .32 and

statistically significant, while the correlation for extrinsic satisfaction was .11 and not statistically significant, confirming the hypothesis. A follow-up study (Arvey et al., 1994) of 95 MZT and 80 DZT pairs yielded a broad heritability estimate of .23 for intrinsic satisfaction, while variance in extrinsic satisfaction could be explained entirely by environmental factors. Data previously gathered from the National Academy of Science and National Research Council (NAS-NRC) twin sample (MZT, *n* = 1152 pairs; DZT, *n* = 1055 pairs) included a single question ("How do you feel about the job you now have?") scored on a five-point response format. We take this measure to assess general satisfaction. Model fitting to these data yielded a heritability estimate of .27. It seems reasonable to conclude that about 25% of the variance in measures of job satisfaction is due to genetic factors. While this may not seem like a great deal, we are not aware of any other single source of influence (e.g., compensation, benefits) that explains this much of the variance in job satisfaction.

Another study in this domain used small samples of MZA (*n* = 23 pairs) and DZA (*n* = 20 pairs) and the Minnesota Importance Questionnaire to measure the work values of Achievement, Comfort, Status, Altruism, Safety, and Autonomy. It yielded estimates of genetic influence of .56, .31, .43, .18, .41, and .34 respectively (mean = .37) (Keller et al., 1992). Finally, the NAS-NRC sample discussed above included 15 job importance items in addition to the job satisfaction item. The mean heritability estimate for these *items* was .34.

While the number of studies of interests and work measures is much more limited than in the domains of abilities and personality, there are enough studies using different kinships to make a convincing case that reliable measures in this domain are significantly influenced by genetic factors.

**Table 13   Reared-Together Monozygotic (MZ) and Dizygotic (DZ) Twin Concordance for Adult and Childhood Behavioral Disorders**

| Disorder | MZ | | DZ | | Type | Source |
|---|---|---|---|---|---|---|
| | Concordance | Sample Size | Concordance | Sample Size | | |
| Adult disorders: | | | | | | |
| Schizophrenia | .38 | 279 | .11 | 461 | Compilation | Gottesman (2001) |
| Affective illness | .65 | 146 | .14 | 278 | Compilation | Berrettini (1997) |
| Alcoholism | | | | | | |
| Men | .41 | 413 | .22 | 617 | Compilation | McGue (1995) |
| Women | .34 | 155 | .31 | 154 | Compilation | McGue (1995) |
| Cannabis dependence | | | | | | |
| Men | .50 | 487 | .31 | 387 | Single study | Lynskey et al. (2002) |
| Women | .35 | 699 | .29 | 507 | Single study | Lynskey et al. (2002) |
| Major depression | | | | | | |
| Men | .31 | 861 | .25 | 656 | Single study | Kendler et al. (1999) |
| Women | .48 | 507 | .43 | 348 | Single study | Kendler et al. (1999) |
| Criminal conviction | .52 | 229 | .23 | 316 | Compilation | Gottesman and Goldsmith (1994) |
| Panic disorder | .24 | 67 | .11 | 55 | Single study | Kendler et al. (1993) |
| Bulimia nervosa | .23 | 35 | .09 | 23 | Single study | Kendler et al. (1991) |
| Childhood disorders | | | | | | |
| Attention deficit/hyperactivity | .58 | 69 | .31 | 32 | Single study | Sherman et al. (1997) |
| Tourette syndrome | .53 | 30 | .08 | 13 | Single study | Price et al. (1985) |
| Autism | .64 | 45 | .09 | 36 | Compilation | Smalley et al. (1988) |
| Juvenile delinquency | .91 | 55 | .73 | 30 | Compilation | Gottesman and Goldsmith (1994) |

## PSYCHOPATHOLOGY

Twin and adoption studies have consistently implicated the existence of genetic influences on a wide range of behavioral disorders including schizophrenia, affective disorder, and personality disorders, calling into question the purely psychosocial and dynamic theories that dominated psychological thinking for much of the 20th century. A thorough consideration of the extensive behavioral genetic literature on psychopathology is well beyond the scope of the present review. Recent reviews of this literature can be found in McGuffin et al. (2002) and Leboyer and Bellivier (2002). Here, we report in Table 13 reared-together MZ and DZ twin concordance rates for various forms of psychopathology. These rates are taken either from meta-analytic summaries of the available literature, or, when these are not available, a single large-scale study selected to be representative of the relevant literature. As is evident for the adult and childhood disorders listed, concordance rates are consistently higher among MZ than among DZ twins, consistent with the existence of genetic influences.

The strength of genetic and environmental influence on disorder risk has been quantified through application of liability-threshold models to twin and family concordance rates (Falconer, 1965). In the liability-threshold model, the disorder is assumed to be a manifestation of some unobserved, continuously distributed variable (termed liability) such that affected individuals have liability scores that exceed some fixed threshold along the liability continuum. Under this formulation, the inheritance of the disorder is due to the inheritance of the underlying liability, which, like that for any other quantitative phenotype, can be modeled using standard biometric principles (Neale and Cardon, 1992). For most disorders, biometric analysis of twin and family concordance data has yielded strong evidence for genetic influences. The heritability of liability has been estimated to be approximately 80% for schizophrenia (Gottesman, 2001), attention deficit/hyperactivity disorder (Sherman et al., 1997), autism (Smalley et al., 1988; Szatmari, 1999), and Tourette syndrome (Price et al., 1985), approximately 50–60% for alcoholism (McGue, 1991, 1995) and cannabis dependence (Lynskey et al., 2002), and approximately 40% for major depression (Kendler and Prescott, 1999). Genetic factors clearly exert a major and pervasive influence on risk of behavioral disorders.

As is the case with personality, for most forms of psychopathology the major source of nongenetic influence appears to be nonshared rather than shared environmental factors. Antisocial behavior appears,

however, to be an exception to this general rule. Twin studies have generally found that approximately 30% of the variance in adolescent conduct disorder can be attributed to shared environmental effects (e.g., Gottesman and Goldsmith, 1994; Jacobson et al., 2000). Of some interest is whether these shared environmental effects persist into adulthood. In a large twin study of U.S. male veterans who retrospectively reported their history of adolescent and adult antisocial behavior, Lyons et al. (1995) reported that the portion of variance in antisocial behavior attributable to shared environmental factors declined from 31% in adolescence to only 5% in adulthood. In contrast, the heritability of antisocial behavior increased from 7 to 43% over the same time period. A recent meta-analytic review of 51 twin and adoption studies of antisocial behavior confirmed the decrease in shared environmental effects with age, but failed to find the expected increase in genetic influences (Rhee and Waldman, 2002).

In all likelihood, the genetic basis underlying risk for developing any specific behavioral disorder is due to the operation of multiple genes rather than just one gene. The progress of the Human Genome Project has motivated large-scale, systematic efforts to identify the multiple genes that convey vulnerability to specific behavioral disorders. Two major strategies have been used: genetic linkage studies, and candidate-gene association studies. In linkage studies, researchers attempt to identify the chromosomal locations of disorder-vulnerability genes by identifying genetic markers that reliably cosegregate with disorder status within families. To span the human genome, initial linkage studies typically involve genome-wide searches using 300–400 markers distributed throughout the human chromosomes. Positive initial linkage findings can then be followed by targeted searches of the implicated region in an effort to narrow the region likely to contain a locus affecting disorder risk. The major advantage of a genome-wide linkage study is that it provides a systematic search of the human genome when there are no strong a priori hypotheses about the genes affecting risk.

Genome-wide linkage studies have been undertaken for most major behavioral disorders, including schizophrenia (e.g., Moises et al., 1995), bipolar disorder (e.g., Ginns et al., 1996), manic-depressive illness (e.g., Berrettini, 1997), alcoholism (e.g., Reich et al., 1998), anorexia (e.g., Grice et al., 2002), attention-deficit/hyperactivity disorder (e.g., Fisher et al., 2002b), reading disability (e.g., Fisher et al., 2002a), and autism (e.g., Lamb et al., 2000). Although these studies have not yet led to the discovery of any behaviorally relevant genes, several chromosomal re-

gions have been implicated in several studies of the same disorder. Thus, the short arm of chromosome 6 has been repeatedly implicated in linkage studies of reading disability, and is now subject to targeted searches to narrow the region of interest (Kaplan et al., 2002). Similarly, a recent meta-analysis of genome-wide linkage studies of schizophrenia and bipolar disorder found the strongest evidence for susceptibility loci on the short arm of chromosome 8, and the long arms of chromosomes 13 and 22 for schizophrenia, and the long arms of chromosome 13 and 22 for bipolar disorder (Badner and Gershon, 2002).

The second major strategy for gene identification is a candidate-gene association study. The logic of an association study is exceedingly simple. If a specific gene conveys risk for a specific disorder then the gene should be more common among those who are affected as compared to those who are unaffected by the disorder. Despite its simplicity, the genetic association remains controversial given both the limited number of candidate genes that are currently available (Sullivan et al., 2001) and the difficulty in ethnically matching cases and controls (Kidd, 1993). A handful of genes affecting the serotonergic, dopaminergic, and noradrenergic neurotransmission systems have been investigated with a broad range of behavioral disorders. While there are no unequivocally confirmed associations of neurotransmitter polymorphisms with behavioral disorders at this time, there are several promising leads. These include the COMT polymorphism and risk for schizophrenia (Egan et al., 2001), the dopamine transporter (DAT1) (Cook et al., 1995) and the DRD4 (Farone et al., 2001) genes and risk for attention-deficit/hyperactivity disorder, and the serotonin transporter gene and anxiety disorders (Veenstra-VanderWeele et al., 2000).

Interestingly, the one unequivocally confirmed association between a genetic polymorphism and a complex behavioral disorder involves genes that are expressed in the liver rather than the brain. The rate of alcohol metabolism is principally regulated by two liver enzymes, aldehyde dehydrogenase (ALDH) and alcohol dehydrogenase (ADH). A mutation in the mitochondrial form of ALDH, inherited by 50% of individuals of East Asian ancestry and resulting in reduced ALDH activity, conveys protection against the development of alcoholism (Harada et al., 1982), while a high-activity form of ADH is associated with reduced risk of alcoholism (Thomasson et al., 1991).

While the current yield of specific behaviorally relevant genes is meager, there are several reasons to be optimistic. First, the large number of candidate genes that are certain to emerge over the next 5 years through continued progress of the Human Genome

Project will provide a rich resource for genetic association studies. Second, the coupling of molecular genetic strategies with sensitive brain imaging approaches will provide more powerful tests for genetic effects. For example, Hariri et al. (2002) recently reported a significant association between neuronal activity in the human amygdala and a polymorphism in the serotonin transporter gene that had been previously implicated in anxiety. Finally, the identification of specific behaviorally relevant polymorphisms should provide the opportunity to powerfully test for the genotype-environment interactions widely believed to exist for behavioral disorders. Consistent with this proposition, Caspi et al. (2002) recently reported that a polymorphism associated with low activity of the enzyme monoamine oxidase A (MAOA) was associated with increased antisocial behavior among boys who had been maltreated in childhood but not among boys who had not experienced childhood maltreatment.

## SUMMARY

We have attempted to persuade the reader that while behavior genetic research with human psychological traits faces a number of difficulties—for example, undetected environmental influences due to lack of experimental control, restriction of range of environments, and unrepresentative samples—those difficulties can be largely overcome by the use of multiple designs, adequate measurement, and better sampling. A consequence of the behavior genetic findings is the realization that studies of environmental influence that make use of variation in ordinary biological families face comparable difficulties—such as correlations between variables that confound genetic and environmental influence and are therefore uninterpretable. It should be clear therefore that behavior genetic designs are necessary not only to address questions regarding genetic sources of variance, but also to estimate environmental and joint environmental and genetic sources of influence on psychological traits. As Table 1 illustrates, behavior genetic studies are about much more than estimating heritability.

We argued that the evidence strongly supports the idea that mental abilities are hierarchically organized with a *g* factor at the apex and specific mental abilities below it. The *g* factor is of considerable practical importance, more so than specific mental abilities, and is significantly influenced by the genes. Recent studies suggest that the heritability of the *g* factor increases from childhood through adolescence and into adulthood, with some decline at older ages. The find-

ings of increasing heritability with age are sufficiently counterintuitive that more research is highly desirable. Particularly desirable are longitudinal studies of twins and unrelated individuals reared together. Estimates of common (shared) family environmental influence on *g* appear to be far less than commonly believed.

Specific mental abilities are also influenced by the genes, but much of this influence is mediated by *g*. Genetic influence on specific mental abilities may also increase with age, but there is much less evidence in support of this proposition.

Personality is also best conceptualized in a hierarchical fashion, with various theories postulating between three and nine major factors and numerous specific traits. Personality measures are of considerable practical utility in a variety of domains. A number of reviews of earlier studies, many using small samples, and contemporary large-sample studies suggest that genetic influence on personality trait variation is in the 40–55% range. There is a strong consensus that common (shared) family influence on personality traits is very close to zero.

Social attitudes have only recently come under study by behavior geneticists as they were previously thought to be influenced almost entirely by family and cultural practices. Recent twin studies of adults, including twins reared apart, and one adoption study, suggest that social attitudes such as Religiousness, Conservatism, and Authoritarianism are perhaps as heritable as personality traits. Religious affiliation, on the other hand, is shaped almost entirely by environmental influences. A more limited number of studies suggest that work attitudes and values as well as job satisfaction are also influenced by genetic factors.

The domain of psychological interests was shown to be quite distinct from the domain of personality with which it is often confused. A sizeable number of twin studies have now demonstrated that interests are significantly influenced by the genes, but perhaps just a bit less than personality traits. A model-fitting analysis of the Strong Interest Inventory utilizing twins, adoptees, and biological family members confirmed these studies but, contrary to the twin studies, suggested that much of the genetic influence was nonadditive.

Various behavior disorders were shown to be significantly influenced by genetic factors, but the degree of genetic influence varied considerably from one disorder to another. With the exception of antisocial behavior, environmental influence on most forms of psychopathology is nonshared rather than shared. Most behavior disorders appear to be influenced by multiple genes. There are no unequivocally confirmed

associations between a genetic polymorphism expressed in the brain and a behavior disorder. Nevertheless, modern molecular genetic strategies, particularly when combined with new brain imaging techniques, appear very promising. The ability to characterize specific behaviorally relevant genetic polymorphism will also allow powerful tests of genotype-environment interactions long thought to be important for behavioral disorders.

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