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Genes, Evolution, and Personality

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There is abundant evidence, some of it reviewed in this paper, that personality traits are substantially influenced by the genes. Much remains to be understood about how and why this is the case. We argue that placing the behavior genetics of personality in the context of epidemiology, evolutionary psychology, and neighboring psychological domains such as interests and attitudes should help lead to new insights. We suggest that important methodological advances, such as measuring traits from multiple viewpoints, using large samples, and analyzing data by modern multivariate techniques, have already led to major changes in our view of such perennial puzzles as the role of "unshared environment" in personality. In the long run, but not yet, approaches via molecular genetics and brain physiology may also make decisive contributions to understanding the heritability of personality traits. We conclude that the behavior genetics of personality is alive and flourishing but that there remains ample scope for new growth and that much social science research is seriously compromised if it does not incorporate genetic variation in its explanatory models.

KEY WORDS: Personality; heritability; evolutionary psychology; twins.

INTRODUCTION

This paper is a mix of the empirical, the theoretical, and the speculative. Enough empirical evidence, some of which is reviewed below, has now been gathered to convince anyone but the most extreme skeptic that virtually all human psychological traits are influenced by genetic factors to a significant degree. The primary consequence of this finding is that much contemporary social science research is uninterpretable if it does not incorporate the influence of genetic variation into its explanatory models (Harris, 1998; Rutter *et al.*, 2001; Scarr, 1997; Turkheimer, 2000). A great deal has been learned about the role of genetic influences on personality in recent years. Nevertheless, the research, like all good research, has raised many additional questions in the course of answering a few. Much of the work has been descriptive and atheoretical. We argue that behavior genetic researchers in the personality domain should embed their research programs in a richer and broader framework, one that encompasses epidemiology and evolutionary psychology, as well as more traditional personality and social psychology. In addition, behavior genetic researchers should include measures that probe to a greater level of detail than the typical three to five major personality traits; they should improve the quality of their instruments; and they should measure traits from multiple perspectives.

We have little to say about the genetics of temperament (Nigg and Goldsmith, 1998), mental abilities (Bouchard, 1998) or developmental behavior genetics (Turkheimer and Gottesman, 1996) in spite of their strong relevance to personality writ large. We also only touch on the relationship of personality to the genetics of psychopathology (Cardno *et al.*, 1999). These omissions are due simply to space limitations.

PERSONALITY TRAITS

Psychologists adopt a wide variety of approaches to the problem of how personality should be conceptualized. One strategy, popularized by Hall and Lindzey

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(1970), is to give multiple theories equal time, devoting a chapter to each point of view—a chapter on psychoanalysis, a chapter on learning theory, a chapter on the trait approach, a chapter on the humanistic approach, etc. (Funder and Ozer, 1997). Many of these chapters are literary treatments of personality. They are often fascinating and sometimes reflect profound insights into human motives and actions. As such they provide a source of hypotheses and content for personality measurement. They do not, however, constitute empirical evidence for or against any particular theory or approach to personality. Empirical scientific work requires that the constructs of a particular theoretical approach be measured so that hypotheses proposed by the theory can be tested. The measurement of personality constructs has been more vigorously pursued by trait theorists than anyone else. The trait approach to personality research has a long history (Goldberg, 1971) but was first formulated systematically by the founders of modern personality research, Gordon Allport (1937), Henry Murray (1938), and Raymond Cattell (1943). Cattell was also a pioneer in the application of heredity–environment analysis to personality traits (Cattell *et al.*, 1955); his multivariate abstract variance analysis was a forerunner of the model-fitting methods popular today (Cattell, 1960). Although vigorously attacked during the 1960s by researchers with a strong behaviorist penchant (Mischel, 1968), the trait approach has survived and prospered in both the theoretical domain (Funder, 1992; Pervin and John, 1999) and the applied domain (Hough, 1992; Schmidt and Hunter, 1998); much current research on personality and psychopathology takes a trait approach (Mathews and Deary, 1998; Watson and Clark, 1994; Widiger and Snakis, 2000). This does not mean that we believe that trait theory encompasses all there is to know about personality; we do not. Nor does it mean that there is full agreement among trait theorists regarding how many important personality traits there are, at what level they should be measured, or whether observer reports are preferable to self-reports. We argue for multiple levels and a combination of perspectives.

Personality traits, in our view, are major reflections of "causal agency" embedded in humans by evolutionary processes (Dennett, 1995). Humans, like all other higher organisms, have been shaped by the two major determinants of evolution, natural selection and sexual selection. They have been designed to transact actively with their environments in order to survive and reproduce. Personality traits in this view have distal causes but are influenced, triggered, and moderated by proximal internal and external stimuli. This causal active view of personality is not new. In Allport's (1937, p. 49) view, "Personality is something and personality does something." Recent theoretical and empirical work in personality research, behavior genetics, and evolutionary psychology has put considerable meat on the bare bones of this perceptive argument, and molecular genetics is poised to make its own contributions.

Animal Studies of Individual Differences in Personality

One compelling kind of evidence for genetic influence on behavior, including personality, is, as Darwin (1871, pp. 110–111) pointed out, the successful domestication of animals.

> So in regard to mental qualities, their transmission is manifest in our dogs, horses, and other domestic animals. Besides special tastes and habits, general intelligence, courage, bad and good tempers, etc. are certainly transmitted. With man we see similar facts in almost every family; and we know through the admirable labours of Mr. Galton that genius, which implies a wonderfully complex combination of higher faculties, tends to be inherited; and on the other hand, it is too certain that insanity and deteriorated mental powers likewise run in the same families.

The domestication of animals was probably not as simple a process as is sometimes assumed (Trut, 1999; Zeder and Hesse, 2000). For example, the human–dog relationship may reflect the evolution of wolves to fit a new niche, rather than deliberate domestication by humans (Morey, 1994). Scott and Fuller's (1965) classic work, *Genetics and the Social Behavior of the Dog*, dramatically illustrates Darwin's argument. Indeed, the dog genome is under intensive study, in part because of the dramatic difference in behavioral traits between breeds (Mellersh *et al.*, 2000; Ostrander *et al.*, 2000). Following Darwin's suggestion, geneticists have been selecting and breeding for behavioral traits and creating inbred strains of various organisms in their laboratories for many years. The mouse has been a particular favorite and that work has now gone molecular (Flint, 1999; Tang *et al.*, 1999; Young *et al.*, 1999).

In spite of the obviousness of personality differences within and between animal species, very little work was carried out in this domain because of the fear of being accused of anthropomorphism (Griffin, 1992). Times have changed, and Gosling and John (1999) were able to review 19 factorial studies of personality in 12 species of nonhuman animals (17 based on factor

analyses of individual animals, 2 based on ratings of breeds) and organize the data according to the five-factor model (FFM)—Neuroticism, Agreeableness, Extraversion, Openness, Conscientiousness, and the two additional dimensions Dominance and Activity. Their findings are summarized in Table I.

In a later section we ask whether dimensions of personality represent evolved adaptations. The animal studies imply that natural selection on humans most likely reshaped portions of phylogenetically old mechanisms rather than creating new ones. This is, of course, the standard view of evolution as a processes of tinkering (Jacob, 1977). This view raises the possibility of contrasting the genetic architecture of traits that are phylogenetically old with that of those that might be relatively new. In their summary of work on the inheritance of personality and attitudes (discussed below), Eaves and his colleagues (1999, p. 79) conclude,

> The consistent differences between attitudes and personality may point to different roles in human adaptation. The absence of any assortment and vertical cultural inheritance for personality may indicate that personality is the manifestation of phylogenetically early properties of the nervous system that predates the evolution of the human brain. . . . On the other hand, it is impossible to conceive of attitudes without a culture depending on learning and social interaction for its maintenance and requiring, therefore, those aspects of the brain which are more fully differentiated in humans.

Interestingly enough a glance at Table I shows Neuroticism, Agreeableness, Extraversion, and Dominance are well represented among the social mammals. The more uniquely human traits Openness and Conscientiousness are more sparsely represented. The infrequent appearance of Activity across species strikes us as an anomaly. It is also an underrepresented trait in most human research. Gosling (2001) provides an enlightening and in-depth discussion of what we can learn about personality from animal research.

The Structure of Human Personality Traits

Personality trait theorists have long been divided into lumpers (a few broad traits) and splitters (many more narrowly specified traits). Modern factorial research has largely resolved this problem: there is now a considerable consensus that the structure of personality can be conceptualized in a hierarchical fashion, with a set of global traits at the top of the hierarchy and more specialized traits below. Figure 1 illustrates some of the strong commonalities across various schemes for organizing traits and points out where some important conceptual differences remain. (Of course, it does not include all such attempts!)

For many years Eysenck championed the idea of three global traits—Extraversion, Neuroticism, and Psychoticism (Eysenck and Eysenck, 1985; Nyborg, 1997)—and a great deal of behavior genetic research has utilized this model (Eaves *et al.*, 1989). The three global traits and nine facets of each (representing the content of the trait measures, not individual scales) are given in the first column in Fig. 1.

Table I. Animal Personality Factors Based on Factor Analyses of Individual Animals Classified According to the Five-Factor Model and Two Additional Factors[a]

	Five-factor model trait names					Additional dimensions	
Species	Neuroticism	Agreeableness	Extraversion	Openness	Conscientiousness	Dominance	Activity
Chimpanzee	x	x	x	x	x	x	x
Gorilla	x	x	x			x	
Rhesus monkey	x	x	x	x		x	
Vervet monkey		x		x		x	
Hyena	x	x		x		x	
Dog	x	x	x		x	x	
Cat	x	x	x		x		
Donkey		x	x				
Pig		x	x	x			
Rat	x	x					
Guppy	x		x				
Octopus	x		x				x

[a]Adapted from Gosling and John (1999); an x indicates that the factor or a combination of two factors was found for that species.

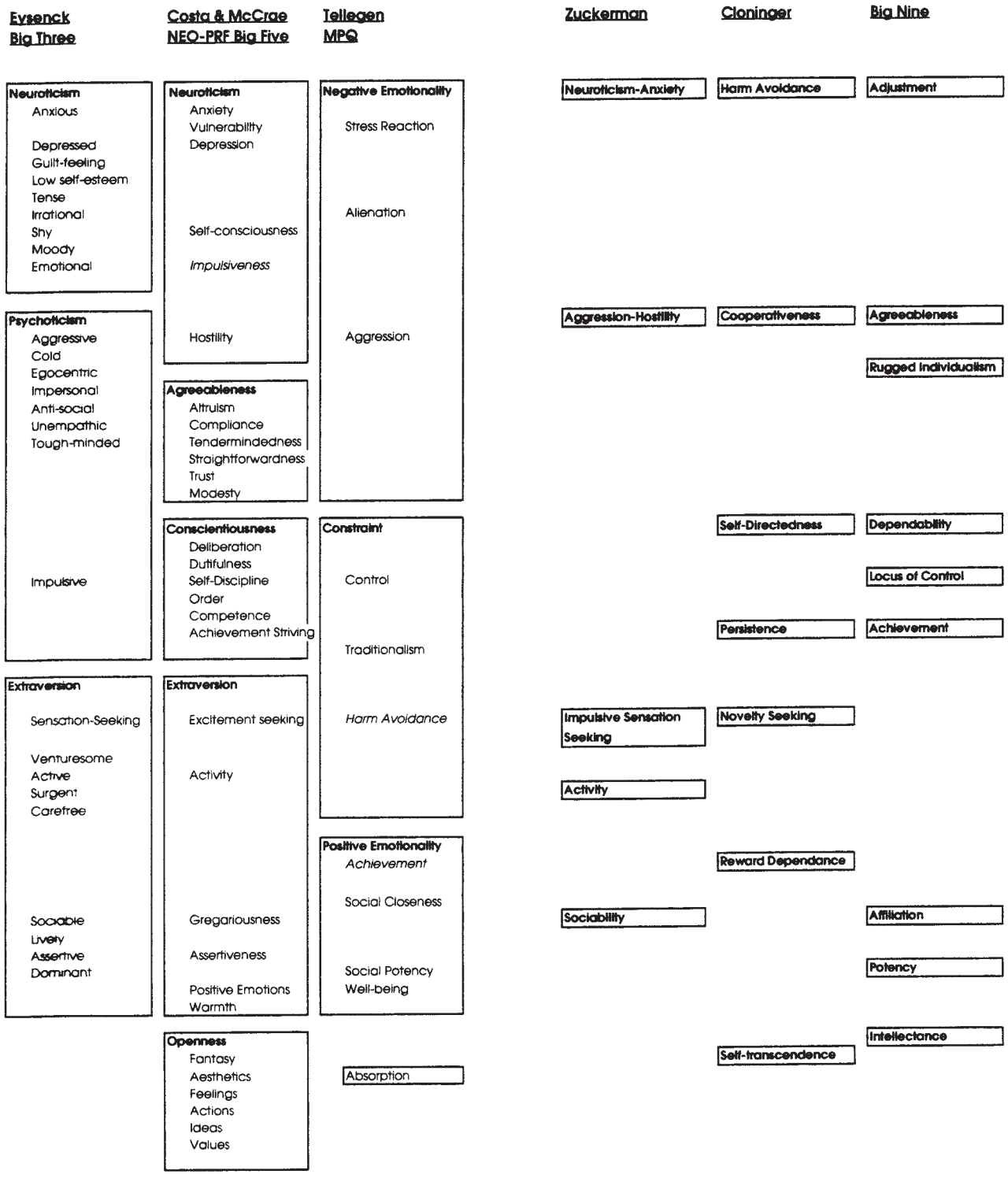

Fig. 1. Some schemes for organizing personality traits.

Of recent popularity in behavior genetic research is the FFM—Extraversion, Neuroticism, Agreeableness, Conscientiousness, and Openness (Goldberg, 1993; John and Srivastava, 1999; McCrae and Costa, 1997). These five global traits and the facet scales used to measure them are listed in column 2 in Fig. 1.

A competing three-factor model proposed by Tellegen (1985) is given in column 3. In this instance the global factors represent the sum of particular scales which are given in the boxes. Church (1994) provides an empirical and theoretical comparison of the Tellegen and FFM schemes. For criticisms of the FFM see Eysenck (1992), Block (1995, 2001), Butcher and Rouse (1996), and Carroll (2001).

To extend the picture we have included the models of Zuckerman (1996) and Cloninger *et al.,* (1996), as well as a model proposed by Hough (1992), shaped largely by applied considerations. This model subdivides both Extraversion (into Affiliation and Potency) and Conscientiousness (Achievement and Dependability) and adds two additional traits, Rugged Individualism and Locus of Control.

On the whole, the rough equivalences across the rows in Fig. 1 suggest that the various schemes can be seen as dividing up much the same pie in slightly different ways, with some unevenness in the density of coverage at the lower levels. A few instances of traits classified differently in other schemes are shown in italics in Fig. 1. Thus the impulsiveness facet is included under Neuroticism in the Big Five, but its opposite, Control, goes with Constraint in the Tellegen scheme; achievement is variously located under Positive Emotionally and Conscientiousness; and harm avoidance, with Neuroticism and with Constraint.

It should be clear that one can slice the trait hierarchy at different levels. For many purposes, choice of level is simply a practical question. In personnel selection it reflects a bandwidth-fidelity dilemma—Ones (1996), for example, argues that a higher level rather than a lower level is the most practical one in applied work. For theoretical work (see the Darwinian argument below) it is probably important to look well beyond a few global traits. That personality has a hierarchical structure is a theoretical formulation that must in itself be explained or refuted. The general argument that evolution hones numerous specialized solutions to problems rather than producing general purpose mechanisms (Buss *et al.,* 1998) raises the question why general factors exist. The animal studies may answer that question. Evolution must work with what is at hand. The narrower traits (facets) may have been developed more recently around the more primitive traits. This raises the question mentioned earlier, whether the facets might have a somewhat different genetic architecture than the more global traits.

Sources of Variance in Personality

One common criticism of behavior genetic studies is that estimation of heritability is largely uninformative because estimates may vary from population to population and from one type of environment to another. This argument makes little sense to us. Average stature varies from population to population and from one environment to another. Would one use this argument as grounds for not studying the stature of populations? The degree to which the heritability of various traits varies from population to population is an empirical question that remains to be answered—and a potentially informative one. One reason this type of criticism is made is because of the lack of a widely accepted systematic framework from which behavior genetic research on human individual differences can be assessed. Eaves (1982) has provided the beginnings of such a framework and we have provided a somewhat expanded version in Table II. The questions listed would be considered perfectly reasonable for any anthropometric character. Why not for psychological traits?

We use the term "epidemiological questions" in the title of Table II for two reasons. First, we would like to focus the reader on the need for population representative samples for accurate portrayal of the effect size of the causal mechanisms listed in the table (Loehlin and Horn, 2000; Newman *et al.,* 1998; Stoolmiller, 1998, 1999). Second, we agree with Krueger, Caspi and Moffitt (2001), who present compelling evidence that personological research has reached the point at which personality questionnaires should be included in major epidemiological studies that address pressing social problems such as mental illness and crime. A convincing case has also been made that the behavior genetics of personality should be an integral component of behavioral medicine (Koopmans, 1997; McClearn *et al.,* 1996; Turner *et al.,* 1995; Vogler *et al.,* 1997).

It should not be assumed that all the items in the long list given in Table II are quantitatively about equally important. As we shall see in the case of broad personality traits, the answer to item B.1 is often a figure in the range 40 to 80%, dominating all other individual components. The variance B.1, as a proportion of the total variance, is the so-called broad heritability,

Table II. 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–environmental 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?

the proportion of individual differences attributable to genetic differences between individuals.

Estimating Variance Components by Model Fitting

How are variance components, such as those in Table II, to be estimated empirically? Nowadays this is usually done by fitting models to the observed variances and covariances between pairs of individuals of different kinds (e.g., MZ and DZ twins or parents and children), using computer programs such as Mx and LISREL. Models are specified, often using path diagrams, that embody the assumptions the modeler wishes to make. For example, observed variation on a trait might be assumed to stem from three sources—additive genetic variation, via a path *h*, environmental variation which is shared with family members, via a path *c*, and a category of residual effects, via a path *e*—the latter would include any effects on the trait of nonshared environment, errors of measurement, developmental accidents, genotype × environment interactions, etc. Observed covariation on the trait between pair members would be modeled as due to additive genes correlated 1.0 between MZs and .5 between DZs, and shared family environment correlated 1.0 in both groups. The program would

then fit the model to the observed variances and covariances, adjusting the values of *h*, *c*, and *e* by trial and error to achieve the best possible fit to the data. At the end the program prints out the values of *h*, *c*, and *e* that produced the best fit and a chi-square value indicating how good the fit was; if this chi-square value is large relative to the degrees of freedom involved, it means that this model does *not* fit the data—something is wrong with the assumptions. Comparison of the fit of models with and without a particular path can be used to test the statistical significance of that path; confidence intervals can also be established.

The particular model just described would allow one to estimate 3 variance components of the 20 or so listed in Table II: namely, h^2, the contribution of additive genes, c^2, the contribution of shared environment, and e^2, everything else. How does one estimate other components? The answer: Complicate the models, and use additional informative groups. Thus if one wants to look at sex differences, one could use variances and covariances from male–male, female–female, and opposite-sex twin pairs and see whether models assuming *h*, *c*, and *e* to be the same for males and females will or will not fit the data. Or one could add parents to the design to look at maternal and paternal effects or assortative mating. Or one can specify models that have patterns of genetic correlations appropriate to dominance (nonadditive effects at a single locus) or epistasis (nonadditive effects involving multiple loci) or that include contrast effects between siblings. Or one can look at the effects of particular environmental variables (e.g., parental warmth) by incorporating explicit measures of them into the model. On the genetics side, a variance–covariance approach to the search for quantitative trait loci (QTLs) is described by Blangero *et al.* (2000).

The procedure is very flexible. One can extend it in a multivariate direction by modeling several traits at once or in a longitudinal direction by using models incorporating repeated measures of the same individuals. A wealth of particular behavior genetic models may be found, with worked examples, in a handbook by Neale and Cardon (1992); for more on model fitting in general, see Loehlin (1998).

PERSONALITY IN AN EVOLUTIONARY PSYCHOLOGY PERSPECTIVE

No one doubts that personality traits exist functionally. That is, they predict behavior in applied settings (Barrick and Mount, 1991; Hough and Oswald, 2000), they are useful in clinical research and practice (Dawis, 1992; Harkness and Lilienfeld, 1997; Lubinski, 2000; Widiger and Snakis, 2000), and they are deeply embedded in natural languages (John *et al.*, 1988). The question remains, In what sense are they real? They could be real but arbitrary, and simply shaped by the particular child rearing practices of a given culture. In this instance quite different traits would likely characterize cultures that differed from each other in significant ways. Those who take this position view the human brain as largely a general purpose learning machine (tabula rasa). It is easy to see why one might take this view. Humans undergo a long period of growth and development and they demonstrate a great deal of behavioral plasticity. The polar opposite approach to this question is the "modular view" of many evolutionary psychologists. In this view different parts of the brain are highly specialized to solve specific problems faced by humans during their evolution (Barkow *et al.*, 1992; Buss, 1999; Buss *et al.*, 1998; Cartwright, 2000; Pinker, 1997). This period of evolution is generally considered to be the Pleistocene and is called the environment of evolutionary adaptation (EEA). It is important to distinguish between the contemporary environment and the EEA because modules shaped by the EEA need not be adaptive in the contemporary environment (Bock and Cardew, 1997). A Darwinian adaptation in this view is "a phenotypic trait with purposeful/functional design created by past selection" (Thornhill, 1997, p. 5). The emphasis on the EEA and "past selection" are designed to counter critics who object that particular phenotypes are not currently related to fitness and, therefore, cannot be "adaptive." In the parlance of evolutionary psychology, organisms are adaptation executors not fitness maximizers (Tooby and Cosmides, 1992, p. 54). It is only in populations that fitness is maximized by the selective development of adaptations, and it usually takes a very long time. In summary, the plasticity view sees the contemporary environment as the primary driving or "causal" or shaping force, while the modular view sees the environment as triggering specific modules that were tuned to specific features of the environment by evolution in the EEA. Of course, many intermediate views are possible (Nesse, 2000).

Evolution and Individual Differences

How might personality traits be related to humans' evolved psychology under a modular view? The following is a speculative account, perhaps a little closer

to the position of Tooby and Cosmides (1990) that personality traits (as such) are unlikely to be adaptations than to that of Wilson (1994) that they may very well be. However, we place a great deal more emphasis on the importance of individual differences than do Tooby and Cosmides (cf. Segal and Macdonald, 1998).

At the most basic level, we find the primitive emotion–motivation systems shared by humans with other mammals—anger, fear, sex, nurturance, curiosity, and so on (Plutchik, 2001). We may suppose that these systems have trigger points and damping points that vary from individual to individual, held only within a broad range by stabilizing selection, that is, selection against extremes at either end. Genetic variation in these parameters persists because their taking on particular values within the acceptable ranges does not lead systematically to reproductive advantage or disadvantage. This may be because of different individuals occupying different environmental niches at a given time, or environments changing over time, or frequency dependencies—the value of a trait may depend on how many other people have it. Contingencies among traits may also be a factor—a hair-trigger temper may be advantageous if one is big and strong but not if one is small and weak. The apparent overresponsiveness of some human defense mechanisms (both physiological and psychological) has led many researchers to look for "flawed genes." A signal detection analysis of such mechanisms, embedded in evolutionary theory, makes it clear that "when the cost of expressing an all-or-none defense is low compared to the potential harm it protects against, the optimal system will express many false alarms (Nesse, 2001).

Individual differences in these emotion–motivation systems are not themselves adaptations, but to the extent that the environment of early humans was largely social, it provided a basic landscape within which adaptations developed, adaptations which in many cases required the correct recognition of the characteristics of individuals: Is this particular person trustworthy or a cheater, aggressive or compliant, stable or erratic, dumb or smart? As Buss (1995a, p. 22) puts it, "Perceiving, attending to, and acting upon differences in others has been (and likely still is) crucial in solving adaptive problems." In his view, higher-order traits such as the Big Five represent basic dimensions of the social adaptive landscape: who is good company (Extraversion), who is kind and supportive (Agreeableness), who puts in sustained effort (Conscientiousness), who is emotionally undependable (Neuroticism), and who has ideas that pan out (Openness/Intellect).

To say that evolution has shaped personality does not mean that direct study of the brain is irrelevant. Just the opposite is true. The modularists have leaned heavily on the localized effects of brain damage to support their views (Duchaine *et al.*, 2001). It may well be that the results from imaging studies in humans (Canli *et al.*, 2001), in conjunction with experimental lesioning, hormonal manipulation, and microelectrode recording in lower animals, will provide the decisive evidence to resolve modularity–plasticity issues (Allman, 2000; Panksepp and Panksepp, 2000).

It is important to recognize that individual differences on personality traits tend to be quite large relative to other differences, such as sex differences, that are viewed as substantial and important by evolutionary psychologists—and by laypersons. Neuroticism is an example of a personality trait that consistently shows a relatively large sex difference, rivaled only by agreeableness and sometimes openness among the Big Five. Women, on average, tend to score higher on neuroticism scales than men do. Three estimates of this difference, based on large studies of adults in Finland and the United States (Costa *et al.*, 1986; Viken *et al.*, 1994), were .29, .17, and .29 within-group standard deviations, an average of about .25. This implies a correlation of .14 between sex and the trait, in a population with equal numbers of the two sexes (Cohen, 1977). This means that about 2% ($.14^2$) of the total variation in neuroticism scores is predictable from knowledge of a person's sex.

Now, of course, part of the 98% of the variation in neuroticism not predicted by sex reflects temporary trait fluctuations and measurement error, but in the Finnish study about 61% of the within-sex variation was stable over a 6-year interval. A reasonable estimate, then, is that individual differences play a role about 30 times greater than sex differences in variation on this trait.

Experience-Producing Drives

Many of the emotion–motivation systems underlying these dimensions can be considered *experience-producing drives* (Bouchard, 1997a; Hayes, 1962). During an individual's development such a drive leads to the acquisition of a system of related knowledge, attitudes, and values. The content comes from the surrounding culture, but it has been selected by a biased system and the individual has created his/her own experiences. That is, to a considerable extent individual differences come about because of inherent propensities (experience-producing drives) to engage in differ-

ent types of activities that result in extended practice and the formation of stable psychological structures. Bouchard *et al.* (1996) have extended the theory along the lines proposed by Scarr (1992), arguing that such drives have evolved for individuals to fit appropriate environmental niches. In this sense individuals create their own environments. The process of humans learning the content underlying a trait is seen as no different from the way in which other organisms learn about the world to which they are adapted (beavers learning about water, logs, etc.). In each instance the learning mechanisms have built in motivational biases. "The alcoholic engages in self-destructive behavior, the sociopath surrounds himself with like minded peers, and the extrovert seeks social stimulation" (McGue *et al.,* 1991, p. 401), and these behaviors significantly influence their proximal environmental input (experiences). These kinds of "created effects" can in principle be significantly attenuated, perhaps even eradicated, if one has total control over an individual's environment. But in practice, one does not, and the environment usually reflects the genes. The behavioral effects of genes are never totally environment-free. The gene that enhances learning and memory in mice (Tang *et al.,* 1999) requires that the mouse have experience with the tasks for the gene to manifest its phenotypic effect. A comment by Dawkins (1982, p. 197) makes the point succinctly.

> A geneticist colleague has argued that there are virtually no behavior genetic traits, because all those so-far discovered have turned out to be 'by products' of more fundamental morphological or physiological effects. But what on earth does he think any genetic trait is, morphological, physiological or behavioral, if not a 'by product' of something more fundamental? If we think the matter through we find that all genetic effects are 'by products' except protein molecules.

In the spirit of Dawkins' geneticist colleague, some psychologists argue that genetic influences on personality are mediated largely if not entirely by external physical characteristics:

> For example, similar interests and values in identical twins may reflect their similar physiques, constitutions, and physical characteristics rather than any personality specific genes. These physical qualities may lead other people to treat them similarly even when the twins live in different environments. A shared interest in becoming a photography fashion model, for example, may say more about the inheritance of faces than of personality. Unquestionably one's physical endowment has extensive influence on one's life and personal development. The degree to which there are specific genes for particular psychological characteristics is much less evident. (Mischel, 1986, p. 184)

This is a specific testable hypothesis about how genes may influence personality. Feingold (1992) carried out an extensive meta-analysis of the literature relating physical attractiveness to personality and found almost nothing, thus the title of his paper: "Good-Looking People Are Not What We Think"; but see also Langlois *et al.* (2000). As evolutionary psychologists would have predicted, the physically attractive do have somewhat of an advantage gaining access to sexual partners. Bouchard (1993) has reviewed the relevant evidence on this issue and shown that Mischel's explanation could at best account for only a trivial portion of the similarity between twins. Loehlin and Nichols (1976) long ago showed that similarity in treatment could not account for twins' similarity in personality. They found differences in treatment and early experience of twins to be related to differences in their personalities at a level too low to be of any predictive value.

Assortative Mating for Personality

Assortative mating, if it is of a sizable magnitude, introduces complexity into the analysis of the genetic architecture of a trait. For example, high assortative mating for a trait strongly under the influence of additive genes accounts for a considerable amount of the phenotypic variation in a population characterized by such a mating practice. Consequently it is important to estimate the size of the assortative mating coefficient for the various personality traits. Contrary to widespread belief, such coefficients for most personality traits are often found to be very modest and many are near zero. The mean correlation for 10 MPQ scales (excluding Traditionalism) based on 583 spousal pairs is .13 (Lykken and Tellegen, 1993). Waller and Shaver (1994) report a similar finding for a 33-item version of the MPQ. The mean correlation for the 20 folk scales of the California Psychological Inventory (CPI) is .22 (Bouchard *et al.,* 1998). In multiple large-sample studies the spousal correlations for Psychoticism, Neuroticism, and Extraversion are near zero (Eaves *et al.,* 1999). Adjusting for unreliability of measurement would not raise these figures much. Attitudinal measures, on the other hand, show sizable correlations (Eaves *et al.,* 1999; Waller *et al.,* 1990). The very low personality correlations may seem somewhat of a puzzle given the strong preferences shown in the mate choice literature (Buss, 1994). But on traits where the preferences of males and females differ, one would not expect agreement. Moreover, Lykken and Tellegen (1993) studied 738 twin couples and found that "characteristics both of the

chooser and the chosen constrain mate selection only weakly" and argue that it is "romantic infatuation that commonly determines the final choice from a broad field of potential eligibles and that this phenomenon is inherently random, in the same sense as is imprinting in precocial birds" (p. 56). Note that the "broad field of potential eligibles" refers to the modern world, not to the EEA. In the EEA the number of eligibles at any given time would likely have been quite limited, and forming a romantic bond on short notice might have been a good idea.

Behavior genetic studies have also thrown light on other facets of marriage. There is now considerable evidence that divorce is heritable (McGue and Lykken, 1992; Trumbetta and Gottesman, 2000; Turkheimer *et al.,* 1992) and that this effect is, in part, mediated by personality (Jockin *et al.,* 1996). Marriage, on the other hand, appears to act as a protective factor against liability for depression (Heath *et al.,* 1998). The evidence also suggests that initial assortment is the cause of what spouse similarity there is, rather than phenotypic convergence (Caspi and Herbener, 1993). Finally, there appears to be assortment for antisocial behavior (Krueger *et al.,* 1998).

The theory of mate selection is an important intersection of behavior genetics, evolutionary psychology, and sociology (Buss, 1999, Part 3; Eaves *et al.,* 1984; Mascie-Taylor and Boyce, 1988), and it possibly deserves even more empirical and theoretical attention than it has received. The modest spouse correlations for personality, in contrast to the sizable correlations for intelligence (Bouchard and McGue, 1981) and social attitudes and values (D'Onofrio *et al.,* 1999), strike us as a paradox that has not been explained by evolutionary psychologists.

OVERVIEW OF EARLIER BEHAVIOR GENETIC FINDINGS ON PERSONALITY

Twin Studies of Broad Personality Traits

The behavior genetic literature on personality has been reviewed a number of times in recent years (Bouchard, 1993, 1997b; Loehlin, 1992; Sherman *et al.,* 1997). We briefly summarize the earlier literature on twins to provide a background against which to discuss recent findings.

A meta-analysis of twin data collected before 1976 for Eysenck's three factors (P, E, and N) was carried out by Eaves *et al.* (1989). In the same book they analyzed P, E, and N from a London twin study (both sexes) and the Australian Twin Study (by sex). The twin meta-analysis yielded a value of near zero for shared environment for all three traits. The DZT correlations were almost exactly half the MZT correlations and the Falconer heritabilities were .58 for Extraversion ($n = 36$ studies), .44 for Neuroticism ($n = 22$ studies), and .46 for Psychoticism ($n = 15$ studies). The smaller London twin study replicated the previous work, although the DZT correlations were lower than found in the past. The larger Australian twin study also found, for both sexes, minimal shared environmental effects and heritabilities similar to those reported above. Extraversion and Psychoticism showed some nonadditivity (.32 for E and .11 for P).

Bouchard (1997b) organized the twin correlational data for personality reported by Nichols (1978) according to the Big Five and eight of the Big Nine. The DZ correlations were generally half the MZ correlations (Big Five, mean $r_{MZ} = .48$, mean $r_{DZ} = .22$; Big Eight, mean $r_{MZ} = .47$, mean $r_{DZ} = .26$), suggesting a simple additive model with heritabilities of the order of .52 and .42 and minimal shared environmental variance. One study of Locus of Control (Miller and Rose, 1982) can be added to this summary to complete the Big Nine, and the results are consistent with the above conclusions.

Recent twin studies of the Big Five have yielded similar results, summarized in Table III. These are from twin studies in the United States (Waller, 1999), Canada (Jang *et al.,* 1996a), and Germany (Riemann *et al.,* 1997), and they are compared with a review of the earlier literature on twin and familial correlations (Loehlin, 1992). The most striking characteristic of these studies is that they provide roughly equal broad heritabilities across the Big Five factors, mostly in the range $.48 \pm .10$. Shared environment was fairly consistently estimated as at or near zero. In the majority of the analyses, the genetic variance of one or more of the traits appeared to have a substantial nonadditive component, but which dimensions these were varied from sample to sample. This might reflect differences in content between scales purporting to measure the same trait, or sampling fluctuations affecting the relative sizes of correlations, or, conceivably, genuine differences between the populations sampled. Large studies with multiple measurement of traits, carried out across several populations, should clarify matters.

Thus for broad traits at the level of Eysenck's three factors or the Big Five, the twin data have fairly consistently suggested heritabilities in the .40–.60 range and little or no effect of shared family environment. The remaining 40 to 60% of the variance is typically

Table III. Broad Heritabilities of Self-Report Measures of the Big Five Factors[a]

	Loehlin review	Jang *et al.* (Canada)	Waller (U.S.)	Loehlin *et al.* (U.S.)	Riemann *et al.* (Germany)
Extraversion	0.49	0.53	0.49	0.57	0.56
Agreeableness	0.35	0.41	0.33	0.51	0.42
Conscientiousness	0.38	0.44	0.48	0.52	0.53
Neuroticism	0.41	0.41	0.42	0.58	0.52
Openness	0.45	0.61	0.58	0.56	0.53
MZ pairs		123	313	490	660
DZ pairs		127	91	317	304

[a] Loehlin (1992) review, model fitting to twin and family correlations from the literature with widely varying sample sizes; Jang *et al.* (1996a) and Riemann *et al.* (1997), versions of the McCrae and Costa NEO: Waller (1999), factors resembling the Big Five; Loehlin *et al.* (1998), latent factors from three derived measures, data of Loehlin and Nichols (1976).

labeled "nonshared environment," but it really comprises a conglomerate of residual causes—nonshared environmental influences as such, G × E interactions, chance factors in development, and errors of measurement. On the whole, the genetic variance appears to be mostly additive, but some nonadditivity is often found.

Adoption and Family Studies of Personality

Adoption studies and family studies have typically yielded lower heritability estimates. Loehlin *et al.*, (1981) reviewed earlier family studies using personality questionnaires and found an average sib–sib correlation of .17 and parent–child correlation of .14. If shared environmental effects are negligible, as the twin studies suggest, and assortative mating is slight (as discussed above), heritability estimates can be obtained simply by doubling these figures, yielding heritabilities of .34 and .28, respectively. The latter figure might be an underestimate, since it does not allow for parent and child being measured at different ages, as is typical in family studies. A recent very large (20,415 unique sib pairs) population-based study (Martin *et al.*, 2000) utilizing the Eysenck EPQ yielded correlations of .118 for P, .164 for E, and .171 for N, results quite similar to those in the earlier studies.

Adoption studies usually include biological relatives as control or comparison groups. In studies in Minnesota and Texas, the median correlations across sets of personality inventory scales in several samples averaged about .20 for biological siblings and about .15 for biological parent–offspring pairs (Loehlin *et al.*, 1985, 1987; Scarr *et al.*, 1981). In the Colorado Adoption Project, correlations on the scales of the EASI were lower still. At age 16 they averaged about .08 for biological parents and offspring and about .05 for biological siblings (Plomin *et al.*, 1998). In these various studies correlations for adoptive relatives tended to be quite low, with the average correlations lying in the range −.03 to .08, consistent with the finding from twin studies that shared family environment has little effect on personality.

In the large Hawaii Family Study of Cognition, subsamples of families were given various personality questionnaires, including the Eysenck Personality Inventory, the 16PF, the Comrey Personality Scales, Gough's Adjective Check List, and scales for Sensation-Seeking, Locus of Control, and Anomie. For parents and children, the average correlation over all scales was .12; the average sibling correlation was .10 (Ahem *et al.*, 1982).

It would be difficult to assign an average figure much above .15 for the parent–child and sibling biological correlations in these studies, implying a typical heritability of .30 or so for personality scales, in contrast to the heritabilities of about .50 obtained in the twin studies. Why the difference? One plausible interpretation is that nonadditive genetic variance may be playing a larger role in personality than was suggested by the MZ and DZ correlations alone. The genetic correlation between parent and child is based only on the additive component of genetic variance, and that between sibs mostly so, especially when epistasis is involved. MZ twins share all their genetic variance, additive and nonadditive, so that when nonadditive genetic variance is present, estimates based on MZs alone or on the difference between MZs and DZs will be larger than those based on parent–child and sibling correlations. The two kinds of estimates are sometimes

referred to as heritability in the broad sense and in the narrow sense. The former refers to the totality of genetic variance; the latter to just the additive part, that portion transmissible, on average, across generations. The presence of nonadditivity is of some interest from an evolutionary perspective, as it suggests that the traits have been under selection—presumably, in the case of personality traits, largely stabilizing selection against extreme values at either end.

Combined Model Fitting

There have been a number of studies in which models have been fit jointly to personality data from twins and other relatives. Sometimes these represent combinations of different data sets; sometimes they are based on the families of twins. Eaves *et al.* (1989) reported examples of each kind for the Eysenck scales. In the analysis based on combined data sets, two of the three Eysenck dimensions showed substantial nonadditive genetic variance (Extraversion did not); in the twin-family analysis, which involved measures of Extraversion and Neuroticism, a simple additive model fit the data well for both dimensions.

Loehlin (1992), in reviewing the behavior genetic literature on personality, classified scales roughly according to the Big Five and fit models to reported correlations for various groups, including twins reared together and apart and adoptive and biological family correlations. A model involving additive genes and epistasis fit the combined data reasonably well, estimating shared environment as zero and broad heritabilities in the range .35 to .49, of which something like a third was nonadditive for all dimensions except Openness (the heritabilities were given in Table III).

Finkel and McGue (1997) fit models to data from Tellegen's MPQ for twins and their families from the Minnesota Twin Family Registry. Mean heritabilities across sexes lay in the range .35 to .54 for the individual scales of the MPQ and .45 to .52 for the three higher-order factors. Significant nonadditivity was found for all the scales except Traditionalism and Absorption.

Finally, Eaves and his colleagues (1998) fit models to measures of Extraversion and Neuroticism in large samples of U.S. twins and their families—spouses, parents, siblings, and children (20,554 individuals altogether)—plus large twin samples from Australia and Finland (3806 and 7144 pairs, respectively). They found that when allowances were made for different levels of reliability, reflecting the fact that different length scales were used in the different studies, the same model fit all three sets of data. This model included additive and nonadditive genetic effects and nonshared environment—there was no evidence of effects of shared environment. Males showed higher broad heritabilities than females did for Extraversion (.57 versus .38); the two sexes were very similar for Neuroticism (.52 and .51). Assortative mating was nonexistent for both traits (spouse correlations of .00 and .01). A substantial proportion of the genetic variance for both traits was nonadditive—a model involving epistasis fit a little better than one involving dominance, but the two are not easily distinguishable in data such as these. Similar results were reported for Neuroticism when the U.S. twins and their families were compared to a comparable Australian sample (N = 20,945) (Lake *et al.,* 2000).

In short, a simple picture emerges, at least with respect to broad personality dimensions as measured by self-report questionnaires: a substantial genetic contribution to individual differences, in the 40 to 60% range, part of it nonadditive; no contribution of shared family environment; and a large remainder attributable to nonshared environment plus whatever else may be in the residual term.

Multivariate Behavior Genetic Analyses of Personality Traits

Nearly always, behavior genetic studies of personality that make overall estimates of nonshared environmental variance find a lot of it. Objectively measured nonshared environmental variables have, however, proven to be a very difficult to operationalize. Turkheimer and Waldron (2000) carried out a meta-analysis of the relevant literature and concluded that that the prospects in this domain are "gloomy," emphasizing the possibility that the process underlying nonshared environmental variance are sufficiently complex that they will "remain outside the domain of systematic scientific investigation for a very long time" (p. 93). As we point out later a consensus model of personality which incorporates multiple viewpoints shrinks the nonshared environmental variance considerably. Nevertheless, for personality variables assessed in the traditional fashion understanding and measuring the variables that influence the nonshared environment remains a challenge. One line of evidence does, however, suggest systematicity to this source of variance, namely, the fact that the higher order structure of the nonshared environmental variance is the same as the phenotypic and additive genetic structure. As shown in

Fig. 1, the phenotypic structure (structure of the raw correlations) of the MPQ consists of three higher-order factors, Negative Emotionality, Positive Emotionality, and Constraint. It is possible to ask if the genetic correlations and the nonshared environmental correlations have the same structures. This is an empirical question, as there is no mathematical constraint that requires the structure to match each other. The method of carrying out such an analysis is complex (involving numerous paths in a model called a triangular or Cholesky decomposition) but simple in conception. Recall that the estimation of genetic and environmental influence on a trait involves decomposition of the twin covariances on that trait. When a number of traits are available as with the 11 MPQ scales, cross-twin, cross-trait covariances (i.e., the covariance between Twin A scores on Well-Being and Twin B scores on Social Potency, the covariance between Twin A scores on Well-Being and Twin B scores on Achievement, and so on) for both MZ and DZ twins can be computed. One can then fit a Cholesky model to these phenotypic variances and covariances and decompose them into genetic and environmental covariances. These matricies can then be factor analyzed in the same manner as phenotypic correlations and covariances. Krueger (2000) carried out such an analysis on the MPQ using the large Minnesota Twin Registry Sample. Highly comparable factor structures were found for the three matricies. As Krueger (2000, p. 1066) points out, "If the differences between twins were due only to random measurement error, it would impossible for these random error components to covary in a psychologically meaningful way that resembles the phenotype. The structure of the nonshared environment thereby points both to its reality and its importance."

Other studies have yielded comparable results, although perhaps not quite such clean ones. In an analysis based on item clusters from the California Psychological Inventory, similar factors appeared in the genetic and unshared environmental portions of the covariance, factors resembling several of the Big Five, with a somewhat different structure appearing in the (modest) shared environment component (Loehlin, 1987). A similar study of the structure of Cloninger's Tridimensional Personality Scale confirmed that a fourth dimension shown in phenotypic factor analyses was present genotypically as well, at least for women, who constituted the bulk of the twin sample used (Stallings *et al.,* 1996).

McCrae and his colleagues (2001) used a similar strategy with the combined data from the Canadian and German twin samples (mentioned in connection with Table III), but they took the process a step further. Following a suggestion by Rowe (1982), they reasoned that the unshared environmental covariance could be divided into two portions, one due to the implicit personality theories of the raters (their beliefs as to what goes with what) and one due to genuine structure in the unshared environment. They used self and spouse ratings from a separate sample to disaggregate the two components of the unshared environment and factor-analyzed the four resulting matrices. They recovered a typical Big Five structure from the genetic and implicit personality theory matrices. They found little if anything in the shared environment matrix and a couple of factors in the residual unshared environment matrix—one in the Conscientiousness domain and one combining the warmth and sociability aspects of Extraversion with the altruism and tenderness facets of Agreeableness.

The multivariate approach has been used to address a variety of theoretical questions. For example, Eysenck's higher-order Psychoticism factor has always been controversial (Claridge and Birchall, 1978). Heath *et al.* (1989, 1990) threw additional doubt on its validity when they found that the items scored on the P scale went together phenotypically, in line with Eysenck's theory, but on a genetic factor, subsets of P scale items reflecting hostility/toughmindedness and suspiciousness actually loaded in opposite directions. Big Five supporters, who suspect that P may combine the negative poles of Agreeableness and Conscientiousness, may not be entirely surprised at this finding of its genetic heterogeneity.

On can also ask if two correlated traits, say anxiety and depression, are linked for genetic or for environmental reasons. Eley (1997) found that in a sample of 8- to 16-year-old twins in Great Britain, the association between these two traits was mostly genetic (80% of it), with 20% due to shared environmental factors and none to unshared environment.

Another way in which multivariate analyses have been employed in behavior genetics is to ask whether traits in normal and clinical populations have the same underlying genetic and environmental structures, which would be consistent with the idea that pathological traits are just extreme versions of the same traits that occur in nonclinical populations. For example, Livesley *et al.* (1998) analyzed samples of personality-disordered patients, general population subjects, and twins, using an 18-scale self-report inventory. They found highly similar phenotypic factor structures in all three samples, and these were the same as the genetic

and (unshared) environmental structures found in the twin sample. Multivariate genetic analysis can also be used to clarify the relationships between questionnaires as well as within them. In a study with the Australian twin sample, multivariate analyses based on the genetic components of the Eysenck and Cloninger personality questionnaires showed them to be in part overlapping but in part capturing different aspects of personality variation (Heath *et al.*, 1994). Markon *et al.* (2001) show a similar relationship between the MMPI and the MPQ scales.

Clearly, the use of multivariate behavior genetic analysis is becoming an important tool in examining the basis of personality structure.

Traits at Different Levels of Generality

There have been a number of attempts to compare the genetic and environmental variation of traits at different levels of generality, for example, broad traits and their facets, or even individual personality questionnaire items (reviewed by Loehlin, 1992, Chap. 4). In the Finkel and McGue (1997) twin-family study mentioned earlier, the average broad heritability of the scales was .44 and that of the three higher-order dimensions was .49. Clearly, this is not a huge difference, but perhaps one would not expect to find one, given that the factors are simply composites of the scales.

Jang and his colleagues (1998) took this approach a step farther, presenting heritability data for the facet-level scales of the Big Five after partialing out the relevant Big Five factor from each facet scale. That is, these are the heritabilities for what is distinctive to each facet, apart from the Big Five factor to which it belongs. The study was based on the combined Canadian and German twin samples (see Table III), both of which used versions of the Costa and McCrae (1995) NEO questionnaire. The results are listed in Table IV. Most of the residual factors showed genetic variance, although their heritabilities were typically lower than those of their parent factors, with the majority lying in the 20–35 range. This is not surprising since reliable variance has been removed from each scale in the partialing process, most of it genetic. The last columns in Table IV show that, by correcting for unreliability, the heritabilities are brought up to the usual levels for personality scales.

Of particular interest, four of the residual facets proved to be fit better by a model involving shared environment than by one involving genes. Two of these were in the Agreeableness domain (Altruism and Modesty), and two in Conscientiousness (Achievement Striving and Deliberation). It will, of course, be interesting to see if these particular findings hold up under replication. Altruism has shown shared environmental effects in some studies (Krueger *et al.*, 2001b) and not others (Rushton *et al.*, 1986). Jang *et al.*, (1998, p. 1556) conclude, "Facet-level traits are not simply exemplars of the broad factors they define; they are discrete constructs with their own heritable and thus biological basis."

Table IV. Heritabilities of NEO Personality Inventory Residual Facet Scores[a]

Domain and facet	h^2	c^2	r_{ft}	h^2/r_{ft}
Neuroticism				
Anxiety	.25		.58	.43
Hostility	.21		.53	.40
Depression	.25		.50	.50
Self-Consciousness	.29		.54	.54
Impulsiveness	.27		.59	.46
Vulnerability	.26		.56	.46
Extraversion				
Warmth	.23		.60	.38
Gregariousness	.28		.71	.39
Assertiveness	.29		.72	.40
Activity	.27		.70	.39
Excitement Seeking	.36		.69	.52
Positive Emotions	.30		.63	.48
Openness to Experience				
Fantasy	.25		.60	.42
Aesthetics	.37		.72	.51
Feelings	.26		.57	.46
Actions	.34		.69	.49
Ideas	.33		.69	.48
Values	.35		.71	.49
Agreeableness				
Trust	.31		.62	.50
Straightforwardness	.25		.56	.45
Altruism		.20	.50	
Compliance	.26		.54	.48
Modesty		.26	.64	
Tendermindedness	.28		.64	.44
Conscientiousness				
Competence	.11		.44	.25
Order	.26		.69	.38
Dutifulness	.28		.43	.65
Achievement Striving		.26	.54	
Self-Discipline	.28		.61	.46
Deliberation		.18	.71	

[a] h^2—proportion of the variance attributable to additive genetic influence; c^2—proportion of the variance attributable to shared environmental influence; r_{ft}—retest reliability. Reproduced from Jang *et al.* (1998) with permission.

Sex Differences in Heritabilities

A major debate in contemporary psychology involves the origin and scope of sex differences in personality (Buss, 1995b; Buss *et al.*, 1999; Eagly and Wood, 1999; Feingold, 1995; Mealey, 2000). Behavior genetic methods cannot answer most of the questions underlying this controversy, but they can address the question, Do the genetic causes of family resemblance depend on the sex of the individual? If the adaptations of men and women indeed differ in important respects, one would not be surprised at a "yes" answer. Attempts to answer this question have yielded inconsistent results in the past, partly because of the small sample sizes used in many of the studies. Recent studies have ameliorated the sample size problem, and in addition have incorporated numerous additional kinships besides twins.

Finkel and McGue (1997) applied a sex-limitation model to the MPQ data from the Minnesota Twin Family Registry (220 MZ males, 406 MZ females, 165 DZ males, 352 DZ females, 114 DZ opposite-sex, 210 male sibs, 449 female sibs, 260 father–son, 578 father–daughter, 409 mother–son, and 652 mother–daughter pairs). They found no evidence that different sets of genes influence personality in each sex, but there was evidence that for some traits the same genes contributed differently to the total variance for males and females. Their results are reported in Table V. Alienation, Control, and Absorption (in boldface) show significant sex differences in heritability. The higher-order factors do not.

As noted earlier, the large study by Eaves and his colleagues (1998) of twins and families found that males and females differ in the heritability of Extraversion but not of Neuroticism. They also found that a model with the same genes for both sexes fit their data reasonably well.

Thus the verdict on sex differences in heritability of personality seems to be: sometimes, but certainly not always. The details of how these tie in with evolutionary theory are still to be elucidated.

Adult Age Changes in Heritability of Personality Traits

We center our discussion on changes in the genetic and environmental contributions to Extraversion and Neuroticism during the adult years, a topic on which there is now a large amount of twin-study evidence. By way of background, McCartney *et al.* (1990) reported a meta-analysis of twin studies from 1967 through 1985. The title of their article, "Growing Up and Grow-

Table V. Variance Components and Broad Heritability for the Scales and Higher-Order Factors of the Multidimensional Personality Questionnaire[a]

	a^2	d^2	e^2	h^2b
Well-Being				
Females	.22	.18	.60	.40
Males	.33	.07	.60	.40
Social Potency				
Females	.30	.24	.46	.54
Males	.38	.15	.47	.53
Achievement				
Females	.21	.17	.62	.38
Males	.10	.22	.68	.32
Social Closeness				
Females	.35	.12	.53	.47
Males	.21	.23	.56	.44
Stress Reaction				
Females	.23	.22	.55	.45
Males	.39	.03	.58	.43
Alienation				
Females	.16	.23	.61	.39
Males	.25	.36	.39	.61
Aggression				
Females	.18	.21	.61	.39
Males	.05	.30	.65	.35
Control				
Females	.20	.13	.67	.33
Males	.02	.45	.53	.47
Harm Avoidance				
Females	.22	.23	.55	.45
Males	.27	.19	.54	.46
Traditionalism				
Females	.47	.08	.45	.55
Males	.52	.00	.48	.52
Absorption				
Females	.29	.15	.56	.44
Males	.11	.15	.74	.26
Mean	.25	.19	.57	.44
Higher-order factors				
Positive Emotionality				
Females	.31	.17	.52	.48
Males	.29	.24	.47	.53
Negative Emotionality				
Females	.25	.17	.58	.42
Males	.29	.19	.52	.48
Constraint				
Females	.44	.09	.47	.53
Males	.23	.27	.50	.50
Mean	.30	.19	.51	.49

[a] a^2—additive genetic variance; d^2—dominance variance; h^2b—broad heritability ($a^2 + d^2$); e^2—nonshared environmental variance. Traits in boldface show a significant sex difference in heritability.

ing Apart," suggests their main finding—that twin correlations tended to decrease with age. For personality traits, these decreases were on the whole similar for MZs and DZs, suggesting a decrease in the effects of shared environment but no marked changes in heritability. Although their samples included ages up to 50, most were young—the median age for personality traits across studies was about 16. Thus the relevance of these findings for the adult years remains unclear. In addition, the reviewed studies employed a wide variety of personality traits and measures.

Age trends in heritability for measures of the Eysenckian traits Extraversion and Neuroticism are now available for large twin samples in three countries: 4766 Finnish twin pairs (Viken *et al.*, 1994); 12,898 Swedish twin pairs (Floderus-Myrhed *et al.*, 1980); around 550 older Swedish pairs, about half reared apart (Pedersen and Reynolds, 1998); and 3739 Australian pairs (Loehlin and Martin, 2001). The Finnish study covered an age range of 18 to 53 years at first testing. It was divided into six 6-year age groups, each tested twice, 6 years apart. The first Swedish study reported correlations separately for three age groups, 16–28, 29–38, and 39–48. The older twins in the second Swedish study participated in up to four testings at 3-year intervals; their average age at first testing was 60 years. The Australian study involved three separate twin samples tested at mean ages of 23, 37, and 61 years. The Australian study included Psychoticism and Lie scales, and the Swedish study Openness, but we focus on the traits measured in all four studies, Extraversion and Neuroticism.

The results can be readily summarized. First, the contribution of shared environment appeared to be negligible at all adult ages for both traits. Second, models with equality of genetic parameters across ages provided acceptable fits for the older Swedish twins and the Australian twins; however, such models led to a poor fit in the Finnish and the Floderus–Myrhed data (reanalyzed by Eaves *et al.*, 1989). Most of the discrepancy involved the youngest age groups, 18–23 and 16–28, respectively, for whom heritabilities were higher. A smaller longitudinal twin study in Minnesota (127 twins retested after 10 years) also found declines in heritability in early adulthood for Positive and Negative Emotionality measures resembling Extraversion and Neuroticism (McGue *et al.*, 1993).

One the whole, then, heritabilities for Extraversion and Neuroticism appear to be stable over much of the adult age span, although they may be higher for late adolescents/young adults. Other personality traits have been less studied. Psychoticism in the Australian study had a somewhat lower heritability (and some psychometric problems), but its heritability did not vary significantly across the three age groups, nor did Openness across the four testings in the Swedish study, nor did Constraint at the two testings in the Minnesota study.

"UNSHARED ENVIRONMENT": A CLOSER LOOK

The Importance of Multiple Viewpoints

There are a number of more specialized topics in the behavior genetics of personality yet to be discussed, such as gene–environment interactions, prenatal biases, birth order, and the role of developmental "noise" in contributing to trait variation. However, before we address them we need to consider three important recent studies that may drastically alter our perspective on some of these matters. These three studies assess the heritabilities of traits measured from multiple viewpoints. One is the study of MZ and DZ twins in Germany, from which data have already been reported. In it, the Big Five traits were measured by a self-report questionnaire—a German version of Costa and McCrae's NEO-FFI—and by having two peers of each twin fill out a third-person version of the same questionnaire (Riemann *et al.*, 1997). A joint model fitting based on the three data sources gave the familiar result of a substantial genetic component, sometimes nonadditive; no contribution of shared environment; and a residual nonshared environmental term. However, there was an important difference. The genetic component, rather than accounting for about half the total variance, accounted for 66 to 79% of it, and the "nonshared" component fell to 21 to 34%. One can speak of this as "increased reliability," and it is, in a sense. But individual self-report personality scales are often quite reliable, in conventional terms. It is perhaps more useful to think of the process of consolidating self- and peer ratings as getting rid of specificities of viewpoint. If we define a trait as a consensus of viewpoints about a person (a consensus trait measure), that trait is *much* more heritable than that same trait measured just by a self-report questionnaire. (The latter estimates were, of course, also available in this study; they are given in Table III. It will be recalled that they fell in the familiar .42 to .56 range.)

Similar, although less striking results come from a study using brief Eysenck scales for Extraversion and Neuroticism and ratings by self and twin (Heath *et al.*, 1992). These ratings were obtained for 460 MZ and 366 DZ female twin pairs from Virginia. The authors

fit rather complex path models to the data, but one can calculate simple correlations for the self-ratings, twin ratings, and a combined rating, from the covariance matrices they present. Since the DZ correlations are less than half the MZ correlations, the latter provide an approximate estimate of broad heritability. For Extraversion, these are .39, .27, and .44 for self-report, twin's report, and a composite of the two; for Neuroticism they are .35, .30 and .60. The single-viewpoint heritabilities are perhaps a trifle on the low side for a twin study, but the scales were rather short (8 and 12 items, respectively). For Neuroticism, at least, the heritability of the composite was markedly higher. Note that ratings by self and twin may not constitute viewpoints as distinctive as the self- and two peer ratings in the German study.

The third study yielded even more dramatic results. A large research project, sometimes referred to as the NEAD (Nonshared Environment in Adolescent Development) study, is described in the book *The Relationship Code* (Reiss *et al.,* 2000). It is an impressive study of over 700 same-sex adolescent sibling pairs and their parents. The study is pioneering in several respects. Six genetically informative groups were used: MZ twins, DZ twins, siblings from ordinary families, and full sibs, half-sibs, and genetically unrelated sibs from step-families. Seven broad dimensions of adjustment were assessed for each adolescent: antisocial behavior, depression, cognitive agency (school performance and the like), sociability, autonomy, social responsibility, and sense of self-worth. For all but the last, multiple sources of data were used: self-report, mother's and father's ratings, and, in some cases, ratings from videotaped interactions. The study was based on a national, not a local sample, although one somewhat upwardly biased in socioeconomic status by the requirements of sample definition and cooperation. Many of the adolescents were measured twice, 3 years apart.

The behavior genetic results, summarized in Table VI, were noteworthy in several respects. One was that the seven dimensions were not all alike, although the genes contributed to all. Four dimensions—Antisocial Behavior, Depression, Cognitive Agency, and Social Responsibility—showed the usual minor effect of shared environment—0 to 10%—but the effect of the genes was decidedly higher—71 to 89% at the initial testing—than the heritabilities typically reported for personality traits in twin and adoption studies. The role of the nonshared environment correspondingly declined, to 11 to 23%. It is ironic that a study designed to elucidate nonshared environmental effects wound up making them vanish, or, more accurately, reducing them almost entirely to specificity and error. (The cited internal consistency reliabilities of the trait composites fell in the .73 to .85 range, yielding error estimates of 15 to 27%, easily as large as the nonshared environment estimates of 10 to 22%.) Apparently, what is common to multiple perspectives on a given trait is, for these four dimensions in this population, almost entirely genetic. What is not genetic is reducible to specificity of viewpoint and unreliability of measurement.

Table VI. Components of Variance of Adjustment of Adolescents in the NEAD Study[a]

Composite	h^2	c^2	e^2	Error
Antisocial behavior	.70	.10	.18	.15
Depression	.77	.00	.23	.15
Cognitive agency	.75	.02	.23	.15
Sociability	.57	.31	.12	.24
Autonomy	.40	.46	.14	.20
Social responsibility	.89	.00	.11	.27
Global self-worth	.17	.03	.80	

[a] Estimated from the graph on p. 387 of Reiss *et al.* (2000). First occasion of measurement. Errors are $1 - \alpha$ from pp. 114–117.

What of the other three dimensions? One was self-worth, the only one of the seven assessed from only one viewpoint—the person's own. Its variance was mostly split between the genes and the nonshared environment, but the genes contributed only 17%. Finally, the remaining two traits, sociability and autonomy, in addition to genetic effects of 57 and 40% and minor nonshared components of 12 and 14%, showed quite substantial *shared* environmental effects as well: 31 and 47%. These two traits referred, respectively, to the extent of involvement with friends, peers, and social organizations and to the adolescent's degree of independence for his age. Apparently, one way in which families *do* have an effect is in how soon the children are encouraged or permitted to get away from them. Neighborhoods and available peer groups might play a role here—they are part of shared environment in the usual behavior genetic study.

Results like these, if supported in future studies, present quite a challenge to workers in areas such as socialization, parenting, family systems, and the like. Consider only the dimension of social responsibility, defined in this study by such attributes as helping and sharing behaviors, adherence to social norms, and internalization of moral principles. At initial testing, individual differences on this trait were estimated as 89% genetic, with the remainder presumably all due to speci-

ficity of viewpoint and measurement error. Shared environment accounted for 0%. This means either that parenting did not vary systematically from family to family, or that it was correlated perfectly with each individual child's genes, or that it made no difference (Loehlin, 2001). Harris (1998; J. R. Harris, 2000) argues that socialization proceeds via peer groups rather than parents, but insofar as peer groups are related to neighborhoods and schools, this should show up as part of the shared environment component in a behavior genetic analysis, and, in any case, the size of the genetic contribution suggests that peer relationships, like parent–child relationships, must largely reflect the active and reactive effects of the adolescent's genes.

The implications for the behavior genetics of personality are equally profound. If the "nonshared environment" variance term (beyond measurement error and specificity) is vanishingly small, then such potential contributors to it as gene × environment interactions and random developmental noise must also be relatively small sources of variance. For consensus trait measures these findings lighten Turkheimer's (2000) "gloomy prospect" of a large intractable nonshared environmental component in behavior. For such measures the answer to Plomin and Daniels' (1987) famous question, "Why are children in the same family so different?" becomes simply, "Because their genes are different."

Developmental Noise

A source of variance that may make a contribution to variance in behavioral traits is developmental noise (often called chance). Williams (1956) long ago demonstrated the dramatic biochemical individuality of monozygotic armadillos. Stent (1981) has discussed the role of noise in the development of the nervous system, and Koshland (1984) has found that chance plays an important role in the behavior of bacteria and speculated on the role of chance in influencing traits in higher species including humans. Gärtner (1990) has reported on numerous studies designed to explain the limited success of a 3-year-long effort to standardize laboratory animals. These studies included an experimental manipulation of the environment of different strains of animals and cross-fostering of twin mates (transferring divided eight-cell-stage embryos of cattle and inbred mice to the same or different foster mothers). Baunack and Gärtner (1984) compared body weights and other developmental measures of such artificially created mouse "MZ twins" with those of similarly treated mouse "DZ twins"—which were genetically identical also, since these were highly inbred strains. They conclude that there is a third contribution to variance that is effective at or before the third cell division and is relatively unaffected by the prenatal and postnatal physical environment. This source of variance is nongenetic, probably cytoplasmic, and shared by monozygotic twins, thus making them more similar than one would expect from their genetic identity alone. In fraternal twins this source is nonshared and confounded with nonshared environmental variance. Molenaar *et al.* (1993), elaborating on the work of Gärtner and others, have argued that stochastic processes are perhaps best regarded as a third source of trait variation, over and above heredity and environment. A recent comprehensive treatment of this topic is given by Finch and Kirkwood (2000). As we noted previously, however, studies based on data from multiple sources suggest that this source of variance may play only a small role for personality.

Prenatal Biases in Twin Studies

Chorion type, particularly in MZ twins, has been frequently mentioned as a possible confound in twin studies. MZ twins who share a chorionic membrane (monochorionic twins) share blood circulation, while twins who do not share a chorionic membrane (dichorionic twins) do not share blood circulation. As far as is known, DZ twins are never monochorionic. The standard view has been that expressed by Price (1950, 1978), that prenatal biases, including those possibly introduced by chorion type, are largely difference producing (Rose, 1995). This view has been questioned by, among others, Phillips (1993) and Phelps (1997), particularly as it relates to disease states. Prescott *et al.* (1999) have developed a linear model of the possible bias introduced by such an effect. As these authors admit, the findings in this area are at best mixed. Recent studies continue to fail to find effects and fail to replicate previously reported effects (Gutknecht *et al.*, 1999; Riese, 1999). See also Macdonald (1993) and Christensen *et al.* (1995), both of whom conclude that the twin method is highly robust against these biases.

Martin *et al.* (1997) present an impressive list of possible prenatal sources of differences between MZ twins. They also point out, however, the possibility that reconvergence of the twins may occur during postnatal development, due to the influence of the twins' shared genotypes on perceiving, selecting, and internalizing similar environments. To the extent that such

convergence fails to take place, such prenatal effects would be confounded with nonshared environment in a typical behavior genetic analysis. They could be considered a component of the developmental "noise" discussed by Molenaar and others. Now that large prospective twin studies are being carried out (Derom *et al.*, 1996) we can expect clearer answers to these questions in the future.

Birth Order

One potential systematic source of within-family environmental variation is birth order. Interest in this topic was revived by the recent publication of the book *Born to Rebel* (Sulloway, 1996), which in part takes an evolutionary psychology perspective. A recent attempt to account for the frequent empirical failures to find birth order effects suggests that they may be context specific and, thus, can explain behavior within the family but not outside it (J. Harris, 2000). An ingenious approach by Beer and Horn (2000) used adoption study data. They looked at biological first-borns reared in various positions within adoptive families to unconfound the effects of birth and rearing order on personality. Contrary to expectations derived from Sulloway's theory, they found the effects of position in the family to be slight, at best. To the extent that the within-family component of variation dwindles in size when traits are multiply measured, these matters become less critical, although they remain of some interest.

Birth order research is extraordinarily complex as revealed by the three main articles and four commentaries in the June/July 2001 issue of the *American Psychologist.* While now somewhat dated with regard to its empirical databases and new analytic procedures (namely, meta-analysis), the classic book by Ernst and Angst is still worth reading for its methodological acumen (1983).

Genetic × Environmental Interactions

A great deal has been made of the possible importance of genetic × environmental interactions as a major source of variance in psychological traits (cf. Cadoret *et al.*, (1995)). This is an extension of the general fascination that psychologists have with interactions. There is no doubt that in principle this is an important issue, although it may or not be an important factor in practice. As we have noted, when phenotypes are properly, i.e., multiply, measured, there may be little scope for interactions in accounting for individual differences (they could still play roles in the proximal processes of development).

In any case, most direct research with humans in this area involves phenotype × environment interaction, not genotype × environment interaction, since environments and phenotypes are measured, and genes are not. While genes carry a precise message, developed organisms do not translate that message unequivocally. The great range of phenotypic differences between monozygotic twins, whether they be armadillos or humans, illustrates the point. Much of the emphasis on the importance of interactions comes from demonstrations of such effects using inbred strains. It is important to recognize that highly homozygous organisms (i.e., inbred strains) are unrepresentative of their species. Long ago Falconer (1960) pointed out that inbred strains are unusually sensitive to environmental variation and argued that "this phenomenon interferes with the experimental study of the changes of variance, and until it is better understood we cannot put much reliance on the theoretical expectations concerning variance being manifest in the observable phenotypic variance" (p. 265). Crabbe *et al.* (1999) recently made a similar point. Comparing inbred mice tested on the same behavioral tasks in different laboratories they found that "for some tests, the magnitude of genetic differences depended on the specific testing lab" (p. 1670). The problem discussed by Falconer remains to be solved and may be more pervasive than has been generally realized. In any event it is not obvious how one generalizes from demonstrable interactions based on homozygous organisms to the behavior of more robust heterozygous organisms, including human beings.

As a result of their simulation of genotype × environment interaction in the context of longitudinal genetic models, Molenaar *et al.* (1999) have made the mathematical point that "the interactive nature of a developmental process becomes invisible after a sufficient amount of time, even under the most favorable circumstances. . . . even if the causal interaction is enduring, stable, and effective during the entire life span" (p. 68). In their simulation, genotype × shared environment interactions looked like genetic effects, and genotype × unshared environment interactions looked like nonshared environmental ones. If these results hold generally, they may partly explain the lack of interaction effects in most adult data sets, and they imply that "genetic" effects need not be entirely genetic. This point has already been made above in the context of experience-producing drives; also, recall Dawkins'

statement quoted earlier that "all genetic effects are 'by products' except protein molecules."

Replicable interactions, whether or not involving genotypes, are very difficult to find. The large NICHD Early Child Care Study (1041 children) recently reported "that virtually none of the anticipated interactions among child-care factors or between them and family or child measures proved significant" (NICHD, 1998). Bergeman *et al.* (1988) also searched for personality interactions using MZA twins and found very little solid evidence for them. Interactions should be subjected to the requirement of replication as urgently as main effects—perhaps more so, since they are potentially so much more numerous and thus likely to show up more often by chance. See Bouchard (1993) for a more extensive discussion and relevant references.

A recent example of phenotype by environment interaction illustrates why they retain their fascination. In a large Dutch study of adolescent and young twins and their families, Boomsma, *et al.* (1999) have shown that religious upbringing significantly affects the expression of the Disinhibition scale of the Sensation Seeking Questionnaire in males. The Disinhibition scale consists of items that represent the desire for social and sexual disinhibition as expressed in social drinking, partying, and a variety of sexual partners. Its relevance to religious upbringing is obvious. About half the twin pairs in the study were reared by religious families (MZM = 149, DZM = 124, MZF = 227, DZF = 169, DZOS = 259) and the other half by nonreligious families (MZM = 143, DZM = 123, MZF = 188, DZF = 151, DZOS = 214). The Disinhibition scale showed typical sex and generation differences, with males having higher scores than females and the older generation having lower scores than the younger generation. The results of model fitting for the four groups are shown in Fig. 2.

The only group that does not show genetic influence is the male religious group. This effect is mediated entirely by the 124 DZM twins, who generate the same correlation as the MZM twins (.62). The average age of the twins in this sample is 17.8 years. It will be interesting to see if this effect, and the common family environmental influence (already gone in the nonreligious female group), survives the transition to adulthood. There was some effect of religious upbringing on the neuroticism, anxiety and depression scales used in this study, but the effect was most pronounced on Disinhibition. Two other findings in this study deserve attention. First, model fitting showed that Disinhibition is influenced by the same genetic and shared environmental factors in both sexes. Second, all the variance

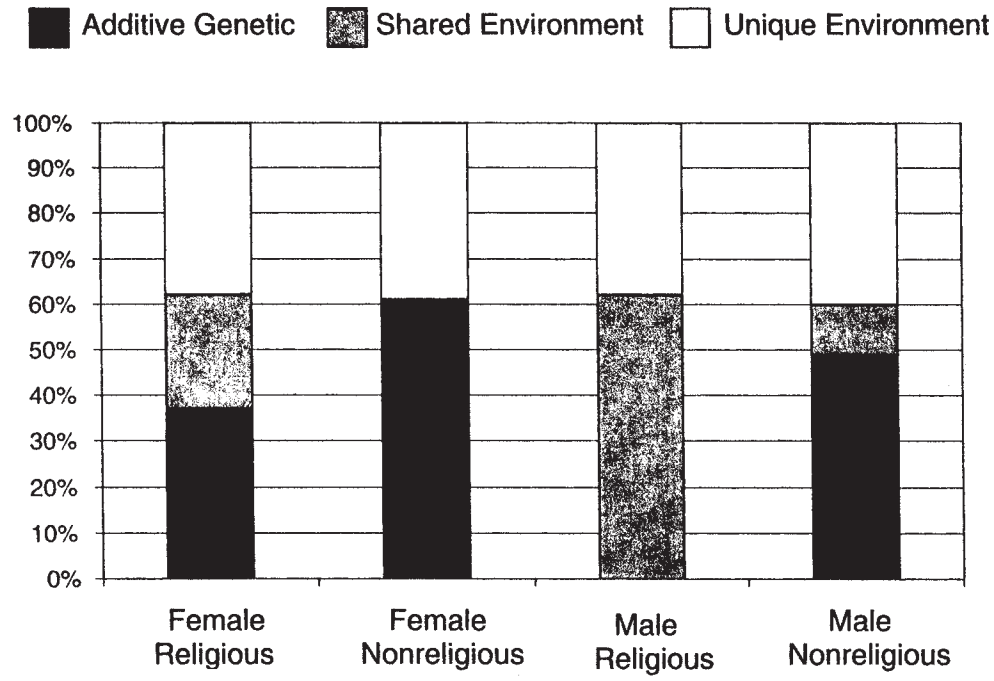

Fig. 2. Percentage of variance in disinhibition due to additive genetic, shared environment, and unique environment by sex and religious upbringing.

in the religiousness measure (a single item asking about participation in religious activities) was environmental for both males and females.

PERSONALITY AND ITS NEIGHBORS

Attitudes and Values

Personality theories have often been presented as general theories of behavior, and some theorists have argued that intelligence should be included in the FFM on these grounds (Brand, 1994). If intelligence, why not interests (Holland, 1997), and why not attitudes and values (Robinson *et al.,* 1991)?

We raise the question here partly because in scientific research the choice of both an appropriate contrast variable and a control group can be very enlightening. Consider the adoption study of mental ability by Scarr and Weinberg (1981). They included a measure of Authoritarianism as a "non genetic control." To their surprise they found that it was highly heritable. Their explanation of the findings was that Authoritarianism was so highly correlated with verbal ability that the genetic transmission of verbal intelligence explained the transmission of Authoritarianism. Their measure of Authoritarianism [the F scale (Adorno, *et al.,* 1950)] was, however, less than ideal. The Right Wing Authoritarianism scale (RWA) appears also to be heritable, and the findings are not explicable by the transmission of verbal ability (McCourt *et al.,* 1999). In addition, there is a high level of assortative mating for RWA (.62), and it is clearly associated with number of offspring [.24 (unpublished data, Minnesota Twin Family Study)].

The Traditionalism scale of the MPQ is another relevant scale. It correlates .71 with the RWA scale. That it shows a substantial heritability (see Table V) should be of interest to social psychologists, who tend to consider such traits as being shaped largely by social learning (Altemeyer, 1988)—although recent work by Tesser and his colleagues (Crelia and Tesser, 1996; Tesser, 1993) reflects something of a change in attitude. Waller and Shaver (1994) administered a brief, 33-item version of the MPQ to an adult sample of 345 MZTs, 100 DZTs, and 181 spouse pairs. For most scales their results were like those of Finkel and McGue reported in Table V; however, Traditionalism showed only a modest heritability (.17) and a sizable common environmental component (.29). The explanation for this difference in results, if real, is not yet obvious. An interesting feature of the Waller and Shaver study was the inclusion of a Love Attitude Scale (Hendrick and Hendrick, 1986) which measured six "love styles." The scale yielded almost no genetic variance (mean = .07) and modest shared environmental variance (mean = .20), thus revealing a very rare class of phenotype, one with little or no genetic variance. The inclusion, within a single study, of measures that are likely to demonstrate different forms of transmission is a highly desirable methodological feature.

Eaves and his colleagues (1999) have reported on a study which administered an abbreviated version of the Eysenck Personality Questionnaire and a brief attitude scale (Conservatism) to the Virginia 30,000 (14,761 adults twins, their 2360 parents, 4391 spouses, 3184 siblings, and 4800 adult children). One of their findings was a large genetic influence on Conservatism, .64 in males and .45 in females. These findings confirm previous results based on twins only (Martin *et al.,* 1986) and are consistent with other studies that demonstrate a genetic influence on attitudes (McCourt *et al.,* 1999; Olson *et al.,* 2001).

Another attitudinal trait around which many individuals organize their lives is religiousness. Religiousness is a highly stable trait significantly correlated with important life outcomes (health, drug abuse, etc.), unrelated to most major personality traits measured by standard instruments, and influenced by genetic factors (Bouchard *et al.,* 1999). Religiousness is also related to fertility (Boomsma *et al.,* 1999). When both religious affiliation and religiousness are studied simultaneously, the former is primarily culturally transmitted (D'Onofrio *et al.,* 1999). A recent issue of *Twin Research* is devoted entirely to genetics, religion, and health (Kirk and Martin, 1999). It and an issue of the *Journal of Personality* (Emmons and McCullough, 1999) devoted to religion and personality both make for fascinating reading. We note that Ramachandran and Blakeslee (1998, p. 175) discuss a recently discovered brain circuit in the limbic system that is clearly involved with religious experience. Research with drugs in the 1960s and 1970s repeatedly reported on the elicitation of religious experiences. The topic has also been addressed by evolutionary psychologists (Goodenough, 1993; Kirkpatrick, 1999; Maser and Gallup, 1990).

Interests

Personality variables overlap only modestly with interests (Waller *et al.,* 1995). Given the fact that in industrialized countries most people spend half their waking hours in the world of work, it is odd that personality researchers, behavior geneticists, and evolutionary psychologists have largely ignored psychological in-

terests. It is still widely believed by psychologists that interests are not influenced by genetic factors (Gati, 1991), but this is simply not true (Betsworth *et al.*, 1994; Loehlin and Nichols, 1976; Lykken *et al.*, 1993).

Psychopathology

A review of the behavior genetics of psychopathology would require another article as long as this one. We merely note here the potential relevance of behavior genetics to the resolution of a basic theoretical question (e.g., Carey and DiLalla, 1994): Are conditions commonly considered psychopathological merely the maladaptive extremes of ordinary personality dimensions, or is psychopathology a whole different ballgame involving a series of distinct gene-based diseases? The dimension running from the trait of aggression within the normal range to extreme criminal violence is an example. Is extreme violence just what ordinary people do in extreme circumstances, or are there genetically distinct individuals called psychopaths who are basically different from the rest of us (Mealey, 1995)? Evolutionary psychologists have been interested in aggression because it shows average differences between the sexes—although of course here, as elsewhere, there is marked overlap: males are on average more aggressive than females, but many individual females are more aggressive than many individual males. Twenty-four studies on the behavior genetics of aggression were subjected to a meta-analysis by Miles and Carey (1997), who found a trend toward increasing heritability and decreasing shared environmental influences with age. Another direction that behavior genetic research in this area can take is illustrated by the recent twin study by Vernon *et al.* (1999), which incorporated 18 measures of aggression. A broad construct of externalizing behavior has been popular in studies with children and adolescents. It is exemplified, for example, in the antisocial behavior dimension in the NEAD study (Reiss *et al.*, 2000) and in the Dutch adoption study by van der Valk *et al.* (1998). In both cases, this trait showed substantial heritability.

The area of personality disorders represents a region intermediate between the major psychoses and normal personality; it is perhaps especially likely that the dimensional approaches of normal personality can be usefully extended here (Costa and Widiger, 1994; Jang *et al.*, 1996b; Livesley *et al.*, 1998; Nigg and Goldsmith, 1998; Watson and Clark, 1994). The multivariate behavior genetic methods discussed previously are especially applicable in this domain.

Many gene-mapping studies (see below) have been focused on psychopathological conditions such as schizophrenia and bipolar disorder and may, in the long run, provide decisive answers to the questions raised here.

WHERE DO WE GO FROM HERE?

Is Behavior Too Complex for Behavior Genetics?

Charlesworth (1992) has pointed out that evolutionary thinking has had at best a weak impact on developmental psychology. Among the numerous reasons he gives for this is what we will call the "complexity argument," namely,

> the extreme epigenetic argument that development is so complex, so nonpredictable, and so conditioned by environmental factors that genetic influences (as the most recent deposits of evolutionary history) lose any meaningful force in shaping it. (p. 13)

Orr (1999), in a recent book review in *Science*, gives another example of how this argument is often used. He calls it the unintelligibility argument.

> Now and then the clash of ideas gets so bad that the text slips into unintelligibility, as when we are told, "the innate complexity of genetic systems necessarily leads to emergenic properties arising from epigenetic processes that integrate the output components of myriad local genetic programs into a functional global phenotype." Such talk seems an unlikely first step to clearer understanding. (p. 344)

It does not seem to be recognized by many practicing scientists that the "It's too complex" argument is an old one (Richards, 1987, Chap. 5). Alfred Wallace, the codiscoverer of the theory of natural selection, broke with Darwin on this very point. While agreeing that morphological traits could be accounted for by natural selection, he argued that man's "intellectual and moral faculties . . . must have had another origin . . . in the unseen universe of the spirit" (Wallace, 1889, p. 31). A Darwinian approach to the question, however, makes the assumption of continuity. That is, humans are simply assumed to be another unique species (Foley, 1987). From this point of view the first question to ask is, Are behavioral traits different from other types of traits? What is the evidence? Figure 3, in part, answers this question. The data are from Mousseau and Roff (1987) and do not include *Drosophila* studies, which have a similar pattern (Roff and Mousseau, 1987).

As Fig. 3 shows that the cumulative frequency distribution of heritability estimates of behavior traits and physiological traits derived from wild animal popula-

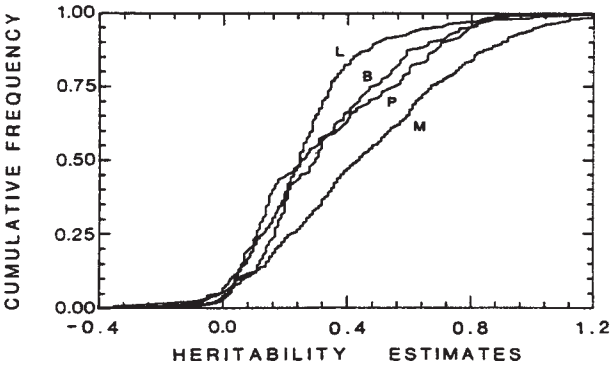

Fig. 3. Cumulative frequency distributions for heritability estimates derived from numerous wild animal populations. L, life history; B, behavior; P, physiology; M, morphology. These data do not include *Drosophila* studies, which yield a similar pattern. [From Mousseau and Roff (1987) with the permission of the publisher.]

tions are very similar, and intermediate to life history (generally fitness traits) and morphological traits. Life history traits are somewhat less heritable on average, and morphological traits are considerably more heritable on average. Two major points are summarized by Fig. 3: (a) genetic variance is ubiquitous, and (b) the heritability of life history (fitness) traits is much higher than expected from theory and the *reasons are not well understood* (cf., Stearns, 1992, Chap. 3). Regarding the ubiquity of genetic variance, Lynch and Walsh (1998, p. 174), point out that "if one's sole interest in performing a quantitative-genetic analysis is to demonstrate that the character of interest is heritable, there is probably little point in expending the effort. The outcome is virtually certain. Almost every character in almost every species that has been studied intensively exhibits nonzero heritability."

It seems to us that the evidence and theoretical arguments that we have drawn on demonstrate that human behavior genetic research in personality is indeed complex, but no more or less so than other organismic traits. It is also the case that considerable progress has been made and continues to be made. We know a great deal more about important sources of variance than we knew only a few years ago. After more than 50 years of relative neglect, developmental psychologists now admit the importance of incorporating behavior genetic designs (Collins *et al.*, 2000) into their research program. Part of the resistance of developmental psychologists to behavior genetic designs is due to the complexity they add to research projects. The simple biological family design with one child is easy to implement. We hope that our review, however, makes the same point regarding behavior genetic designs themselves. The simple twin design, while somewhat more informative than a simple biological family design, continues to be used, largely because it is easy to implement. Progress will depend on the use of better phenotypic measures of personality (preferably multiple measures) and the use of large representative samples of many informative kinships. Almost all investigators have argued that these two criteria are in conflict with each other. This is true only if one expects a single team of investigators in one location to do all the work. The NEAD project is evidence that large multiple-investigator projects with multiple measurement of traits, which access representative samples of genetically informative kinships, are possible and extremely informative.

Of course, the relation of behavior to its genetic underpinnings *is* often complex. Here is E. O. Wilson (1978, p. 198):

> If variation in mental and athletic ability is influenced to a moderate degree by heredity, as the evidence suggests, we should expect individuals of truly extraordinary capacity to emerge unexpectedly in otherwise undistinguished families, and then fail to transmit their qualities to their children. The biologist George C. Williams (1975) has written of such productions in plants and animals as Sisyphean genotypes; his reasoning is based on the following argument from elementary genetics. Almost all capacities are prescribed by combinations of genes at many sites on the chromosomes. Truly exceptional individuals, weak or strong are, by definition, to be found at the extremes of statistical curves, and the hereditary substrate of their traits come together in rare combinations that arise from random processes in the formation of new sex cells and the fusion of sex cells to form new organisms. Since each individual produced by the sexual process contains a unique set of genes, very exceptional combinations of genes are unlikely to appear twice even within the same family. So if genius is to any extent hereditary, it winks on and off through the gene pool in a way that would be difficult to measure or predict. Like Sisyphus rolling his boulder up and over the top of the hill only to have it tumble down again, the human gene pool creates hereditary genius in many ways in many places only to have it come apart in the next generation.

This is an articulate description of genetic nonadditivity of the epistatic variety, and the citation of the plant breeding literature points to the hard evidence of such effects (cf. Lynch and Walsh, 1998, Chap. 15). The demonstration of nonadditivity for human traits depends almost entirely on the inclusion of MZ twins in our models. Such dependence on a single type of kinship for the demonstration of an important effect should

be recognized for the severe limitation that it is. Nevertheless, nonadditive genetic effects are real, pervasive, and relevant to many evolutionary arguments (Wolf *et al.,* 2000) and can be modeled (Li, 1987). Their use in attempting to understand phenomena such as genius and superior performance in a wide array of domains, as suggested by Wilson in the above quote, by Simonton (1999, 2001) is especially interesting, as it helps to integrate a vast literature.

Gene Mapping and Personality

The terms "complex trait" and "complex phenotype" have come into common usage in genetics. These terms imply that the trait is influenced by many factors, both genetic and environmental. Normal personality traits quality as "complex traits," as do most psychiatric traits. Normal personality traits are generally conceived of as being approximately normally distributed, or at least continuously distributed, whereas psychiatric traits are often conceived of as types (Waller and Meehl, 1998). The typological nature of most psychiatric traits is, however, a hypothesis, and the liability for the trait is often treated as a near-normal distribution reflecting the influence of multiple factors. Thus heritability estimates of 80% for schizophrenia and bipolar disorder (Cardno *et al.,* 1999), based on twin studies, apply to variance accounted for in the liability distribution.

With the rapid development of molecular genetics, the factors underlying the genetic component of such traits, now called quantitative trait loci (QTLs), were expected to be quickly discovered. In fact, progress has been exceedingly slow. The first QTLs were demonstrated in tomato plants in 1988 and have only recently been characterized and cloned (Doebley, 2000; Frary *et al.,* 2000). In psychiatry, where QTLs of larger effect were expected to be found, it now seems likely that such genes are rare, and genes of small effect as well as epistatic effects are more likely (Owen *et al.,* 2000). In the normal personality domain the purported "discovery" that a polymorphism of the D4 receptor gene could account for about 10% of the heritable variance in Novelty Seeking (Cloninger *et al.,* 1996; Ebstein *et al.,* 1996) led to a flurry of attempts to replicate. There were many failures to replicate, and the picture is still unclear as to whether this is a real effect or not (Plomin and Caspi, 1998, 1999). Replications, however, continue to appear (Strobel *et al.,* 1999), as do studies of interactions between suspected candidate genes. McGue (2001) reports a meta-analysis of 17 studies with a mean effect size of .21, with a great deal of heterogeneity among the studies. Cloninger *et al.* (1998) tested for QTLs on the TPQ Harm Avoidance scale using a genomewide scan. The sample consisted of 758 sibling pairs from 177 nuclear families of alcoholics. They found a locus that explained 38% of the trait variance and significant evidence of epistatic interactions. When taken together they explain most of the genetic variance in Harm Avoidance (54–66%). If replicable, these results will change the face of personality psychology, as they will provide the ability of researchers to test for real genotype × environment interactions and genotype × environment correlations. This type of work will also allow us to explore the pleiotropic effect (multiple effects on different traits due to the same gene) of genes and the genetic correlations between characters. Martin *et al.* (1997) provide a particularly informative example of how QTLs can be integrated into multivariate structural equation modeling to answer these types of questions using twin and sibling data.

There are, however, reasons to await replication before becoming too excited about any particular findings. Cloninger *et al.* (1998) cite the work of Lesch *et al.* (1996) on the serotonin transporter (5-HTTLPR) polymorphism association with neuroticism (TPQ Harm Avoidance) as one basis for their work. There have now been five failures to replicate this finding constructively (see Flory *et al.,* 1999). Benjamin *et al.* (2000) also incorporated (5-HTTLPR) in a study of epistatic effects (gene × gene interactions) and found no effects for Harm Avoidance. They did find some suggestive interaction effects for Novelty Seeking. These initial results are discouraging to some observers, but the findings of animal studies are sufficiently convincing that the likelihood of eventual payoff from this work is high. The optimistically inclined may wish to keep an eye on the relationship of the dopamine transporter gene (DAT1) to internalizing behaviors (Rowe *et al.,* 1998), and the relation between number of CAG repeats on the androgen receptor gene and one aspect of psychological femininity (Loehlin *et al.,* 1999).

The history of scientific research tells us that exploring any new domain entails an enormous amount of effort, much of it trial and error. If QTLs in the domain of personality turn out to be in the 3–5% range, as currently projected, the issues of sample size, adequate sampling, and quality of measurement will have to be addressed seriously. The statistical tools are in place and the molecular genetic tools (gene chips) and maps (the full human genome) are developing so rapidly that this discussion will be outdated by the time it appears in

print. As we write, preprints refer to a possible 50,000 to 140,000 genes, whereas the genome projects now suggest that it is much lower (35,000) (Szathmáry *et al.*, 2001). The journal *Molecular Psychiatry* was established for rapid dissemination of research in this area. As with the Human Genome Project, it is likely that in the near-future results will appear on the World Wide Web first, with print publications following.

SUMMARY

- Viewing personality as a set of evolved causal traits, many of which are shared with lower animals, we contrasted some of the major personality models and argued that they all reflect a common underlying hierarchical structure, although all the details of that structure have not been worked out.
- Borrowing from Eaves (1982), we outlined the major epidemiological questions regarding sources of population variance in any psychological trait.
- We briefly addressed the argument that "there are no genes for personality," showing that it is misdirected.
- We summarized previous reviews of genetic findings and brought them up to date by summarizing more recent studies, most of which, in contrast to previous work, make use of large samples.
- We highlighted recent findings suggesting that measures of personality that incorporate multiple viewpoints yielded much higher estimates of genetic influence than those based on a single viewpoint. If these findings hold generally, they significantly change the research landscape, reducing in importance questions long thought critical to understanding personality development (i.e., G × E interactions, nonshared environmental influence, developmental noise).
- We specifically recommended that more research be focused on lower-order personality traits as well as the interface among personality, attitudes, and interests. The latter traits are perhaps more specifically human and more likely to be partly shaped by cultural and environmental mechanisms, thereby providing a contrast for findings in the domain of personality.
- We argued that, contrary to some contemporary views, behavior is not too complex for behavior genetics and that, in fact, behavior cannot be properly studied without behavior genetics.
- We briefly reviewed and commented on the apparent slow progress in the field of gene mapping but argued that the exploration of new domains is always characterized by an enormous expenditure of effort and much trial and error and that cautious optimism may still be an appropriate stance.

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