Personality and Psychopathology: Genetic Perspectives

Gregory Carey and David L. DiLalla

Genetic factors exert an important influence on adult personality traits, accounting for anywhere between 30% and 60% of the variance. Heredity is also important for most forms of psychopathology and plays a major role in several theories that relate personality to psychopathology. Despite this, there has been surprisingly little multivariate genetic research reported on joint analyses of personality and psychopathology. The small amount of available data suggests that genes may account for over 50% of the observed correlation between neuroticism and state symptoms of anxiety and depression. The mechanisms behind such strong genetic correlations are crucial for understanding the causal relationship between a personality trait and a disorder because genetically influenced biological systems may operate as exogenous "third-party" factors that are responsible for what appear to be phenotypic cause–effect relationships. We illustrate how recent analytical advances in behavior genetics can use multivariate family data to address questions about the causal role of personality in psychopathology.

Behavioral genetic researchers have demonstrated the importance of both genetic and environmental factors for most personality traits and most forms of psychopathology. The question no longer is whether nature or nurture shapes human development but rather how complex genetic and environmental influences act together to form specific behavioral outcomes. To gain perspective on this topic, it is necessary to know something about the genetics of personality and about the genetics of psychopathology. Hence, we present succinct overviews of the behavioral genetic literatures for personality and for psychopathology. In these reviews, we address such questions as the magnitude of genetic and family environmental influence and the genetic and environmental structure behind the phenotypes.

Most existing genetic research has focused either on normal personality or on psychopathology, with comparatively little published research examining the genetic and environmental relationship between the two. This is the result, in part, of the large samples required for multivariate genetic analyses. To those who invest in such research endeavors, the rewards may be great. Behavioral genetic research on relationships between normal personality characteristics and psychiatric disorder holds the promise of elucidating questions that have long been of interest to psychology. Foremost among them is the question of causation. The position that personality and psychopathology are uncorrelated is untenable (see Watson & Clark, 1984; Wat-

son, Clark, & Harkness, 1994 [this issue]). But how are they causally related?

A recent analytical advance in behavioral genetics demonstrates how family and genetic data may complement other quasi-experimental designs in resolving some issues about causality (Duffy & Martin, in press; Heath et al., 1993). Hence, behavioral genetic data may play an important role in answering such important questions as whether a personality trait etiologically influences a disorder or whether the trait is symptomatic of the disorder. In the remainder of this article, we explore how family and genetic data can assist in this endeavor. To this end, we examine existing theories that deal with genetics, personality, and psychopathology to gain perspective on what types of causal models should be tested. We then use behavioral genetics as a conceptual model to decompose an observed phenotypic correlation between a personality trait and a disorder into its constituent components. We follow this topic with an exposition of the types of causal models that can be tested with family data. Finally, we illustrate the approach by analyzing three simple causal models that relate the personality trait of neuroticism with state symptoms of anxiety and depression. Before embarking on these tasks, however, it is necessary to summarize the two principal methods of behavioral genetics, the twin study and the adoption study.

The Twin Method

Twin designs compare the similarity of monozygotic (MZ) or identical twins with that of dizygotic (DZ) or fraternal twins. If the correlation for MZ twins is significantly higher than the correlation for DZ twins, there is evidence for a genetic effect on the characteristic being measured.

The twin study partitions phenotypic (i.e., observed) variance into three sources, usually expressed as a proportion of phenotypic variance. The first is *heritability*. It is defined as the proportion of phenotypic variance attributable to the additive effects of genes. Heritability is usually denoted as h^2 and is estimated as twice the difference between MZ and DZ correlations,

Gregory Carey, Institute for Behavioral Genetics, University of Colorado; David L. DiLalla, Department of Psychology, Southern Illinois University at Carbondale.

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Correspondence concerning this article should be addressed to Gregory Carey, Institute for Behavioral Genetics, University of Colorado, Boulder, Colorado 80309-0447. Electronic mail may be sent via Internet to carey@ibg.colorado.edu.

or $h^2 = 2(r_{MZ} - r_{DZ})$. The second component of phenotypic variance is referred to as *common environmentability*. It is the proportion of observed variance attributable to all those environmental variables that twins share (such as the same parents, the same household, etc.) and that make twins similar on the trait. Common environment is usually denoted by c^2 and is indexed as twice the DZ correlation minus the MZ correlation, or $c^2 = 2r_{DZ} - r_{MZ}$. The final variance component is called *unique environmentability*. It is the proportion of variance attributable to environmental variables that are uniquely experienced by an individual and not shared by members of a twin pair (e.g., different teachers, different perceptions of parental discipline) and that make twins uncorrelated for the trait. It is denoted by u^2 and is indexed as $u^2 = 1 - r_{MZ}$.

A central assumption of the twin method is that MZ twins are not treated more similarly than DZ twins or, if they are, that this difference in treatment is not relevant to the phenotype under study. The appropriateness of the "equal trait-relevant environments" assumption has generally been supported by research on both personality (e.g., Loehlin, 1992; Loehlin & Nichols, 1976) and psychopathology (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993).

The Adoption Method

In a full adoption design, there are three important correlations. The first is that between a birth parent (or other birth relative) and the adoptee. In the absence of assortative mating, it has an expectation of $\frac{1}{2}h^2$ for first-degree relatives. The second is the correlation between two adoptive, biologically unrelated relatives. If the adoptees are siblings, it would have an expectation of c^2 ; if they are not siblings, it would equal some other parameter that would index the extent to which the relatives in question are similar in personality for environmental reasons. Finally, there is the correlation between genetically related individuals who are reared together. This should equal the sum of the first two correlations for the relatives in a class. For example, the correlation for siblings would be $\frac{1}{2}h^2 + c^2$.

The crucial assumption of the adoption method is the absence of selective placement, defined as the nonrandom placement of adoptees with their adoptive parents. Depending on the adoptee sample, selective placement may be strong for traits such as religion and race and moderate for certain physical traits, such as eye color, hair color, and stature. For personality and psychopathology, however, there is no evidence that selective placement seriously biases the expectations of correlations (Plomin & DeFries, 1985).

Genetics and Personality

Results from a variety of twin studies of personality have been reassuringly consistent over the past half-century (see Eaves, Eysenck, & Martin, 1989; Loehlin, 1992, or McCartney, Harris, & Bernieri, 1990, for reviews and meta-analyses). With paper-and-pencil personality questionnaires, adult MZ twins correlate somewhere between .40 and .60, depending on the measure, whereas on the same scale, adult DZ twins correlate at

Table 1
Correlations for MZ and DZ Twins on Eight Personality Traits: Results of the Meta-Analysis by McCartney, Harris, and Bernieri (1990)

Trait	Number of studies	r_{MZ}	r_{DZ}
Activity-impulsivity	31	.51	.20
Aggression	8	.49	.29
Anxiety	9	.54	.19
Dominance	7	.51	.21
Emotionality	18	.47	.15
Masculinity-femininity	9	.52	.36
Sociability	33	.51	.19
Task orientation	7	.44	.19

Note. From "Growing Up and Growing Apart: A Developmental Meta-Analysis of Twin Studies" by K. McCartney, M. S. Harris, and F. Bernieri, 1990, *Psychological Bulletin*, 107, p. 229. Copyright 1990 by the American Psychological Association. Adapted by permission. MZ = monozygotic; DZ = dizygotic.

roughly half that level (see Table 1 for summary correlations from McCartney et al.'s meta-analysis for eight personality traits).

This generalization became embodied in Loehlin and Nichols's (1976) hypothesis—If the twin method is valid, then approximately half the observed variance in personality can be traced to genetic variation; the environments that members of a twin pair share (e.g., same parents, same socioeconomic status, same peer group) play only a small role in making twins similar. In terms of the mathematical model outlined above, h^2 is between .40 and .50, u^2 is approximately .50, and c^2 is almost always less than .10, frequently within a statistical sampling error of 0.0.

Five different samples of twins reared apart encourage confidence in the assumptions of the classical twin method (Bouchard & McGue, 1990; Langinvainio, Kaprio, Koskenvuo, & Lonnqvist, 1984; Newman, Freeman, & Holzinger, 1937; Pedersen, Plomin, McClearn, & Friberg, 1988; Shields, 1962; Tellegen et al., 1988). Although such twins are not reared in random environments, they should correlate much less than those reared together if common environment has an important effect. In fact, MZ twins reared apart correlate very closely to MZ twins reared together.

Unfortunately, the wealth of twin data is not matched by adoption studies. Among the major adoption or foster studies of adults (Eaves et al., 1989; Loehlin, Willerman, & Horn, 1987; Loehlin, Horn, & Willerman, 1990; Scarr, Webber, Weinberg, & Wittig, 1981), the heritability estimates as well as estimates of shared and unique environmental influences on personality agree well with those from twin studies in suggesting that the dominant reason for familial resemblance in personality can be traced to genetic factors, with common environment having only a small effect. The same may not be true in childhood, however (Plomin, Coon, Carey, DeFries, & Fulker, 1991).

Subsequent analysis has shown that Loehlin and Nichols's (1976) hypothesis, like most generalizations, is oversimplified. There are statistically detectable effects for such phenomena as nonadditive genetic variance, sex differences, age differences,

and perhaps special twin environments for MZ twins (Carey & Rice, 1983; Eaves et al., 1989; Loehlin, 1992). However, the crux of Loehlin and Nichols's hypothesis remains true. Despite sex differences and nonadditive genetic variance, the majority of higher order personality traits show a substantial genetic effect that is not dramatically different from one trait to another. That is, there have not been consistent replicable results showing that one trait has a heritability of .10 and another trait has a heritability of .80. Instead, the range of genetic effects for personality scales is more restricted, having a lower bound of perhaps .30 and an upper bound of around .60.

The genetic and environmental structure behind personality is also crucial for understanding the relationship between personality and psychopathology. Most psychological theory deals with phenotypic structure (e.g., Watson et al., 1994 [this issue]). However, phenotypic structure is a function of both genetic structure and environmental structure. If genetic and environmental structure differ, then the phenotypic structure may be epiphenomenal. Theorists such as Cloninger (1987a) claim that this is the case.

Methods for the study of the genetic and environmental structure underlying personality parallel those for phenotypic structure in the sense that both use factor models. In an ordinary factor analysis, a variable's phenotypic variance is partitioned into that part predictable from the factors (i.e., the communality) and that part unique to the variable (i.e., its specific variance). A multivariate genetic analysis partitions variance in an analogous manner, but in this case the terms apply specifically to the genetic or environmental variance. What is factored is a matrix of genetic correlations (or environmental correlations, if that is the focus of analysis). A genetic correlation indicates the extent to which the genetic effects on one variable predict the genetic effects on another variable (see Carey, 1988). In a multivariate genetic analysis, the genetic variance of a variable is partitioned into two parts—the first part is that attributable to the genetic factors, the second is that portion specific to the variable. A genetic factor in this case would be defined as a hypothetical, latent variable that predicts genetic covariation among the phenotypes. Analogous definitions would hold for common environmental factors and for unique environmental factors.

There has been surprisingly little research on the genetic structure behind personality, mainly because most of the large twin studies capable of examining this issue deal with the higher order, orthogonal traits of extraversion and neuroticism. Carey (1987b) reported on a multivariate genetic analysis of extraversion subscales on the California Psychological Inventory and found considerable genetic variance unique to each subscale. A similar patterning of results was reported by Eaves et al. (1989) for two different traits—impulsivity and neuroticism. Table 2 summarizes Eaves et al.'s results by giving the proportion of the genetic variance attributable to the genetic factor (only a single factor was extracted in each analysis) and the proportion of genetic variance specific to the subscales. For example, for impulsivity, the general genetic factor accounted for 42% of the genetic variance in narrow-sense impulsivity in women and 39% of the genetic variance in nonplanning in women; 61% of the genetic variance in liveliness in women was specific to the Liveliness subscale.

Table 2 shows that there was important genetic variance

Table 2

Percentage of Genetic Variance Attributable to a Genetic Factor and to Genetic Effects Specific for Each Subtrait of Impulsivity and Neuroticism

Trait and subtrait	Women		Men	
	Genetic factor	Trait-specific effect	Genetic factor	Trait-specific effect
Impulsivity				
Impulsiveness	42	58	39	61
Nonplanning	39	61	100	0
Risk Taking	39	61	39	61
Liveliness	39	61	39	61
Neuroticism				
Depression	64	36	67	33
Worry	42	58	38	62
Insomnia	52	48	45	55
Paranoia	65	35	46	54
Inattention	57	43	65	35
Shyness	20	80	10	90
Psychosomatic illness	53	47	49	51

Note. Based on data from Eaves, Eysenck, and Martin (1989).

unique to each subtrait of impulsivity (with the exception of nonplanning in men); there was generally more genetic variance specific to a subtrait than there was to the factor. A similar pattern held for the subtraits of neuroticism, but here, with the exception of shyness, there was somewhat more genetic variance attributable to the factor. Indeed, Heath, Eaves, and Martin (1989) presented evidence that there is important genetic variance specific to items.

These results have great importance for the understanding of the relationship between genes and personality. Genes do not appear to operate exclusively by determining broad dispositional patterns of temperament, which suggests that there is more to the heritability of personality than simply a genetic Big Five. It may be that there are microgenetic effects for the behaviors, specific to the lower order traits of personality. Moreover, these microgenetic effects may be almost as large as the genetic effects on temperament.

To our knowledge, there are no multivariate genetic studies focused on the larger issue of determining the genetic and environmental structure behind a substantive number of lower order traits. To give some indication as to what such a structure might look like, we performed an analysis on the empirically keyed scales of the CPI (Megargee, 1972) along with two ad hoc scales of the CPI developed by Eysenck to measure extraversion and neuroticism. The sample was the National Merit Twin sample described by Loehlin and Nichols (1976). To ease data presentation, we first standardized all scores by sex and pooled men with women. We then computed the covariance matrices for MZ and DZ twins and calculated the genetic correlational matrix, the common environmental correlational matrix, and the unique environmental correlational matrix subject to the constraint that each matrix be positive definite. We then extracted the principal components of these matrices.

Both the genetic correlation matrix and the unique environmental correlation matrix had five eigenvalues greater than 1.0.

Table 3

Factor Loadings (Standardized Regression Coefficients From a Promax-Rotated Factor Pattern Matrix) for the Unique Environment (U), Common Environment (C), and Additive Genotype (A) Components for the Three Major Factors of the California Psychological Inventory

Scale	Component-factor combination								
	U-1	C-1	A-2	U-2	C-3	A-1	U-3	C-2	A-3
Extraversion				0.82	0.81	0.98		-0.44	
Neuroticism	-0.77	-0.73	-0.93						
Dominance				0.77	0.98	0.86			
Capacity for Status				0.46	0.52	0.75		0.57	
Sociability		0.50		0.80	0.80	0.95			
Social Presence				0.47	0.59	0.83		0.71	
Self-Acceptance				0.85	0.90	0.97			
Well-Being	0.73	0.81	0.80						
Responsibility	0.55	0.68							0.92
Socialization	0.51	0.94				-0.40			
Self-Control	0.94	0.93	0.92			-0.53			
Tolerance	0.62	0.61						0.55	0.52
Good Impression	0.88	0.96	1.01						
Communality									
Achievement-Conformity	0.73	0.98	0.60						
Achievement via Independence							0.77	0.87	0.77
Intellectual Efficiency	0.50	0.43				0.50		0.70	0.47
Psychological Mindedness			0.56			0.42		0.72	
Flexibility		0.44					0.81	1.06	0.51
Femininity									

Note. Only loadings greater than 0.40 are shown.

The common environmental matrix had four eigenvalues exceeding 1.0, but the fifth eigenvalue was 0.99. Hence, five principal components were extracted from each matrix. The promax-rotated factor pattern loadings for the first three components are given in Table 3, rearranged so that factors in one analysis that corresponded to those in the others are juxtaposed.

Given the large standard errors of genetic and common environmental correlations, the loadings from the three matrices are remarkably similar and, moreover, parallel the first three phenotypic factors of the CPI (Gough, 1987; Megargee, 1972). The first factor is bipolar, with neuroticism at one pole and CPI good-adjustment scales on the other pole. The second factor is clearly an extraversion factor with the exception of a few low loadings on other scales in the genetic analysis. The third factor is less replicable than the first two, but it has high loadings on flexibility and achievement via independence in all matrices—the two variables that largely define the third CPI phenotypic factor. It appears to have congruence with the third of the big five factors, termed *conscientiousness* (Digman, 1990). The fourth and fifth factors are not shown because they each had only a single loading. The fourth was a femininity factor, and the fifth was marked by the communality scale.

In summary, this analysis suggests that there may be correspondence between the genetic and environmental structures behind personality, thus justifying studies of phenotypic structure. These types of analyses definitely must be replicated on other samples and with scales with better discriminant validity than the CPI's empirically keyed scales.

Genetics and Psychopathology

As with personality, the literature on the genetics of psychopathology is too voluminous to review in detail here (see Van-

denberg, Singer, & Pauls, 1986). Instead, we concentrate on the major aspects of the research by offering the following generalizations.

1. Almost all twin and adoption studies conducted to date have shown evidence for a genetic effect. The results are strong for schizophrenia, bipolar illness, alcohol dependence and abuse, the anxiety disorders, and severe depression. The heritability of personality disorders has been relatively ignored, although there is some evidence for a genetic liability toward schizotypal and borderline personality disorder (Kendler et al., 1991; Torgersen, 1984), and the data on crime are consistent with a genetic diathesis for antisocial personality disorder (Carey, in press).

2. For all disorders studied so far, risk for identical cotwins is always less than 1.0, and for schizophrenia, often taken as a strongly genetic disorder, the risk is closer to .50 (Gottesman, 1991). No behavioral disorder is a pure genetic disease. The environment is critically important. Hence, models that relate the genetics of personality to psychopathology must consider the real possibility of environmental covariation.

3. Familial risk rates are inconsistent with simple, fully penetrant, Mendelian dominant or recessive transmission. It may turn out that there are some genes of large effect (i.e., major genes) for some forms of psychopathology, but they may operate against a background of other genes along with important contributions from the environment.

4. Genetic model fitting has yet to resolve unequivocally the mode of transmission for any form of psychopathology. Hence, we do not know whether a disorder like schizophrenia is largely genetically heterogeneous (a collection of a large number of rare Mendelian disorders with some environmental cases), the tail

end of a polygenic distribution of liability, or some combination of the two (as is mental retardation). This is not to say that genetic modeling is suspect (see Carey, 1987a, for a discussion of this point with respect to studies of affective disorder). On the contrary—it is necessary to pursue the development of better analytical methods for resolving competing hypotheses.

5. To date, linkage and candidate gene strategies have yet to uncover a replicable gene of large effect, a possible exception being the aldehyde dehydrogenase II allele in Asians, which may reduce susceptibility to alcoholism. However, molecular genetic studies are in their childhood, so it is premature to conclude that there are no major genes.

6. Estimates of heritability, common environment, and unique environment from behavioral genetic studies of psychopathology are much less precise than those for personality. This, of course, is due to the rarity of twins and adoptees with the disorders of interest. An exception is schizophrenia, for which, in a polygenic model, heritability may be as high as .70, with unique environment accounting for most of the remaining variance (McGue, Gottesman, & Rao, 1983).

7. Little is known of the multivariate genetic structure of psychopathology. Almost all disorders have significant comorbidity with other disorders (Robins & Regier, 1991). Thus, there is probably a positive manifold to the phenotypic correlational matrix. The extent to which genes contribute to this structure is not known, but the limited available data suggest they may be important. In a sample of twins, Kendler, Neale, Kessler, Heath, and Eaves (1992) estimated the genetic correlation between major depression (MD) and generalized anxiety disorder (GAD) as between 0.83 and 1.0, depending on how GAD was diagnosed. This suggests that the genetic influences on MD are essentially the same as those on GAD. Kendler et al. (1992) hypothesized that for these two disorders, at least, genes may confer a general liability to succumb to stress, with environmental factors determining whether the disorder takes the form of anxiety or depression.

Genetics, Personality, and Psychopathology: Empirical Data

Given the strong theoretical links among genes, personality, and psychopathology—to be reviewed in the next section—it is surprising how few data have been published on the topic. That is, there are considerable genetic data on personality per se and on psychopathology per se but precious few on both phenotypes simultaneously.

Gottesman and Shields (1972) noted more abnormal Minnesota Multiphasic Personality Inventory (MMPI) profiles among the MZ cotwins of schizophrenics than among the DZ cotwins. This suggests important genetic correlations between personality and schizophrenia, but the extent to which the MMPI taps personality versus psychopathology in such a sample is problematic.

Nicholas Martin collected postal questionnaire data on almost 4,000 pairs of Australian twins. The questionnaire included Eysenck Personality Questionnaire and the Delusional-Symptoms-States Inventory: Anxiety and Depression Scales (Bedford, Foulds, & Sheffield, 1976). The latter instrument measures seven symptoms of anxiety and seven symptoms of

depression, highlighting respondents' current state on each of the 14 symptoms. Reporting on the Australian data, Jardine, Martin, and Henderson (1984) and Eaves et al. (1989) undertook multivariate analyses of neuroticism and the anxiety and depression scales that fitted a single genetic factor, a single unique environmental factor, and specific genetic and specific unique environmental variances for the three measures. There was statistically significant genetic variance specific to each of the three measures, but it was small in magnitude for all three variables. For both male and female twins, the largest source of covariance for the three measures was the single genetic factor. For example, the phenotypic correlation between anxiety and neuroticism was .61 in women. The genetic contribution to this correlation was .37, or about 60% of the correlation. These results suggest that genetics cannot be overlooked in trying to account for the correlation between personality and psychopathology.

Genetics, Personality, and Psychopathology: Theory

In this section, we summarize three theories that relate personality to psychopathology and also invoke genetics as an important source of individual differences. Our purpose here is not to evaluate the theories—that would require three different articles. Instead, we wish to provide a framework for the way in which theorists perceive genetics to be related to personality and psychopathology so that we may develop methods of analytically testing hypotheses.

Eysenck's Three-Factor System

Eysenck has long championed the view that personality traits are heritable and represent important risk factors for psychopathology. Eysenck (1952, 1956; Eysenck & Eysenck, 1976, 1985) posited three orthogonal personality traits: extraversion (marked by outgoingness, surgency, sociability, and liveliness), neuroticism (denoted by worries, anxiety, anger, lability of mood, and a general tendency to become upset easily), and psychoticism (indexed by aggressiveness, unconventional—often impulsive and antisocial—tendencies, toughmindedness, and cold and impersonal feelings). Eysenck suggested that neuroticism influenced the probability that a disorder would occur, with individuals scoring high on neuroticism being more likely than low scoring individuals to experience psychiatric disorder. Extraversion and psychoticism, in contrast, were posited to influence the form of the disorder. Introverted individuals high on neuroticism are viewed as more likely to experience anxiety, depression, phobias, and obsessions, whereas extraverts high on neuroticism are seen as at high risk for externalizing disorders, such as hysteria and sociopathy. Individuals high on psychoticism are hypothesized to be at risk for a range of psychopathology, in the extreme being predisposed to schizophrenic-like psychoses. Eysenck (1987) also suggested that the broad clusters of personality disorders outlined in the revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R*; American Psychiatric Association, 1987) mirror the dimensions of his theoretical system.

More genetic data have been obtained with Eysenck's measures of personality than with all other instruments combined.

Excellent overviews may be found in Eaves et al. (1989) and Loehlin (1992). Although simple models may be statistically rejected for extraversion and neuroticism, the data suggest that 40% or more of the variance is genetic in origin, with the remaining variance being largely due to unique environment. There are fewer data on psychoticism, but genetic effects remain important, with the possibility of some contribution from common environment.

Cloninger's Biosocial Theory of Personality

Eysenck identified personality dimensions through phenotypic factor analysis of items and worked inward, postulating attributes of the central nervous system that underlie personality. Theorists such as Gray (1981) and Cloninger (1986, 1987a, 1987b), on the other hand, began by postulating brain systems and then worked outward to their behavioral manifestations.

Cloninger (1986, 1987a, 1987b) postulated three brain systems, each associated with a major monoamine neurotransmitter, that underlie three dimensions of personality. The first is the *behavioral activation system*, associated with the neurotransmitter dopamine. This modulates tendencies toward excitement and exhilaration, including active pursuit of reward and active avoidance of punishment and boredom, and it underlies the personality temperament of *novelty seeking* (NS). The second is the *behavioral inhibition system*, using the neurotransmitter serotonin. It is responsible for passive learning processes that inhibit behavior associated with aversive consequences, novelty, or nonreward, and it is phenotypically manifested in the personality domain by *harm avoidance* (HA). The final system is the *behavioral maintenance system*, with norepinephrine as the primary neurotransmitter. It is the foundation for responses to cues related to reward, yielding individual differences in resistance (or susceptibility) to extinction of behavior that has been rewarded in the past. It is the physiological basis for the trait of *reward dependence* (RD).

The three brain systems and their associated personality traits are assumed to be genetically orthogonal. The interaction (Cloninger's term) between these systems and the environment, however, may induce phenotypic correlations. Hence, genetic and environmental structures differ, and Cloninger particularly eschewed phenotypic factor analysis as a method for assessing underlying biological dimensions of personality (see Cloninger, Svrakic, & Przybeck, in press).

Cloninger has applied his personality theory to alcohol dependence and abuse (Cloninger, 1987a), personality disorders (Cloninger, 1987b), and anxiety states (Cloninger, 1986). In some cases, the relationship between personality and psychopathology is linear (e.g., RD is linearly associated with anxiety). For alcohol problems, the relationship is U-shaped, with individuals low and high on NS, HA, and RD being susceptible to alcohol abuse and dependence. Like Eysenck, Cloninger claims that his system correctly captures the dimensions that, in their extremes, give rise to *DSM-III-R* personality disorders.

There is only one genetic study of Cloninger's system. Heath, Cloninger, and Martin (in press) collected data from 2,680 twin pairs, using abbreviated forms of both Cloninger's and Eysenck's questionnaires. As might be expected, NS, HA, and RD showed moderate heritability, strong unique environ-

mentability, and little common environmental variance. Multivariate genetic analysis of Cloninger's and Eysenck's constructs gave convincing evidence that one system was not simply a "genetic factor rotation" of the other system. Hence, there is more to the genetics of personality than either Cloninger's or Eysenck's systems theorize.

Cloninger recently amended his theory because it failed to distinguish "individuals with personality disorders or poor social adjustment from other well-adapted individuals with extreme personality profiles" (Cloninger et al., in press). Now there are seven personality dimensions—four dimensions of temperament and three of character. There are no genetic data evaluating the amended theory.

Meehl's Theory of Schizophrenia

Meehl's (1962, 1989, 1990) principal genetic assumption is that a single major dominant gene underlies a central nervous system attentional anomaly termed *schizotaxia*. Schizotaxia operates as a *sine qua non* for the subsequent development of schizophrenia. The interplay between the primary schizotaxic defect, various other genetic dimensions of liability (which Meehl terms *polygenic potentiators*), and the social learning environment produce a personality organization called *schizotypy*. Not all schizotaxics develop schizotypy, but it is a highly probable outcome. Schizotypy as Meehl defines it and schizotypal personality disorder as defined by the *DSM-III-R*, though correlated, are not synonymous (Lenzenweger, 1992; Meehl, 1990).

Schizotypy has its own group of signs and symptoms, such as cognitive slippage, interpersonal aversiveness and social fear, hypohedonia, and ambivalence. These behavioral phenomena can lead to their own consequences in terms of inappropriate social behavior, bizarre thoughts, and attitudes, and so forth. The important point for our purpose here is that through a series of reciprocal causal pathways (e.g., aberrant social behavior leads to social isolation which leads to even more aberrant sociality), the schizotypal individual may decompensate, and clinical schizophrenia will develop. Decompensation is not inevitable, however, and Meehl postulated that most schizotypes will not develop an overt psychosis.

Testable Models Implied by the Three Theories

Obviously, the specific aspects of the testable predictions will vary from theory to theory. However, a central thread runs through all three accounts, namely, that the relationship between the genetics of personality and the phenotype of a psychopathologic disorder is phenotypically driven. Eysenck speaks of the personality traits (i.e., the phenotypes) as conferring liability or being dimensions on which some forms of psychopathology, like personality disorders, map. Meehl postulates a causal sequence that starts with a major gene, but it is the schizotype—a personality phenotype—that impinges on the schizophrenic phenotype. Cloninger is less specific but also appears to favor the personality phenotype as a direct causal factor.

In a phenotypically driven system, the risk for a disorder will be the same for all individuals who have the same phenotypic

value, regardless of their genotypes. For example, assume a single genetic locus with two alleles, a and A , that contributes to a personality trait, say, neuroticism. There will be three genotypes— aa , Aa , and AA . If we take individuals 2 standard deviations above the mean for neuroticism, then a phenotypically driven system implies that the selected individuals will all have the same risk for a disorder, regardless of whether they have genotype aa , Aa , or AA .

This relationship between the hypothetical neuroticism gene and the disorder is not necessary. For example, suppose that the locus coded for a noradrenergic receptor had a direct effect on neuroticism and also a direct effect on panic disorder, with the risk for panic increasing with the number of A alleles. Also assume—for simplicity's sake only—that the environment and all other genetic loci for neuroticism are uncorrelated with the other genes and environmental effects for panic. Now, if we select neuroticism phenotypes 2 standard deviations above the mean, risk for the disorder will vary as a function of genotype, with AA individuals at highest risk and aa individuals at lowest risk. This system might be described as genotypically driven.

The key point is that none of the theorists appear to postulate this type of effect. Cloninger's theory applied to a locus with a major effect on one of the major monoamines is a possible exception, but he is not specific on the point. Even Meehl's complex path diagrams (see Meehl, 1972) do not have pathways emanating from a genotype into two phenotypes; all pathways, with the exception of that between a major gene and schizotaxia, connect phenotype to phenotype. Indeed, we surmise that if one were to examine causal models in psychology as a whole, the vast majority would be phenotypically driven. There is one major advantage to such models of personality and psychopathology: They are testable.

Direction of Causation

General Considerations

We now explore how behavioral genetic research can assist in resolving issues of causality between personality and psychopathology. First, however, we examine the different formal mechanisms that can induce a correlation between a personality trait and a disorder.

Figure 1 presents a path diagram that is meant for use as a foil to illustrate the different sources of covariation. Depicted are the phenotypes for a personality trait (P) and for a disorder (D) and two genetic loci or genes (L_1 and L_2). To keep matters simple, other genetic loci and environmental variables are omitted but, if added, would take a form similar to that of the two loci. The straight arrows connecting the loci to P and D denote causal pathways, with the path coefficients (a , b , c , and d) quantifying the magnitude of the causal effect. The personality trait influences the disorder (path e) and the disorder may at the same time influence the personality trait (path f). Mechanisms such as assortative mating, linkage disequilibrium, and the mixture of genetically heterogeneous populations may produce correlations among the loci. We permit this with the double-headed arrow connecting L_1 to L_2 , the coefficient g being the correlation coefficient.

The meaning of the genetic effects is most easily illustrated by

Figure 1. A hypothetical causal model of two genetic loci (L_1 and L_2), a personality trait (P) and a disorder (D).

fixing paths e and f to 0. If both paths emanating from a locus are non-zero, then that locus is *pleiotropic*, a term used in genetics to reflect the fact that a single gene may influence more than one phenotype. If one of the paths emanating from a locus is zero, then that locus may be termed *monotropic* in terms of the phenotypes. The difference between the two types of loci may be illustrated by a thought experiment. Suppose that we could genetically engineer a change in an individual's genotype from, say, aa to Aa , keeping everything else constant. If this produces a change in both the personality trait and the disorder, then the locus is causally pleiotropic. If it produces a change in only the personality trait (or only the disorder), then the locus is monotropic.

As a hypothetical example, suppose that the neurotransmitter dopamine in the striatal area of the brain contributed to individual differences in a personality trait but not to a given disorder and that levels of dopamine in the basal ganglia influenced the disorder but not the personality trait. A locus that influenced dopamine levels in the whole brain would be pleiotropic. A locus that influenced the anatomy or physiology of, say, dopamine storage in the striatum but not the basal ganglia would be monotropic for personality. Finally, a locus that influenced regulation of dopamine receptors in the basal ganglia but not the striatum would be monotropic for the disorder.

The phenotypically and genotypically driven systems discussed in the previous section are specific cases of this model. In the phenotypically driven system, paths e or f or both would be retained, but there would be only one pathway emanating from each locus. That is, each locus would have a direct effect on only one phenotype, either P or D , and no locus would have a direct effect on both. If we depicted different environments, the same principle would hold—only one path from an environment to a phenotype. In the genotypically driven system, paths e and f would be eliminated and loci would be permitted to influence both P and D . One could also construct an environmentally driven system in an analogous fashion. The model we propose is general in that it permits elements of all three systems—genetic, environmental, and phenotypic—to operate simultaneously.

The algebraic correlation between P and D is very complicated and is not presented here. But we have derived it, and from the terms we have identified five conceptually different sources for the observed correlation. The five, along with their pathways, are (a) a phenotypic causal pathway in which personality causes the disorder (e); (b) a phenotypic consequential pathway in which the disorder causes personality (f); (c) causal-factor pathways in which personality and the disorder are jointly caused by the same variable (e.g., ab); (d) correlated-causes pathways in which the causes for personality are correlated with those for the disorder (e.g., adg); and (e) compound pathways that are functions of common causal or correlated causal pathways and the reciprocal causality between P and D (e.g., $abfe$). If other loci or environmental variables analogous to the loci (or both) were added and the same process gone through, the same five conceptual sources for the observed correlation would be arrived at. Hence, these five sources are general.

The distinction between causal-factor (ab and cd in Figure 1) and correlated-causes (adg and bcg) pathways deserves comment. In a causal-factor pathway, the manipulation of the factor necessitates a change in both personality and the disorder. In the correlated-causes pathway, changing the genotype at one of the loci may not change both the personality and the disorder. The genes for skin pigmentation and sickle cell anemia illustrate loci that are correlated but not causally related. In America, alleles for African pigmentation are associated with the disease. However, this relationship is correlational and not causal. If one could take a group of White Americans and change a pigmentation locus from aa to Aa in the African direction, keeping all other factors constant, one would observe a change in pigmentation but not a change in liability to sickle cell anemia.

To return to the five sources of covariance, the sources are obviously not mutually exclusive. For example, it is possible that personality causes the disorder and, at the same time, that a common causal factor contributes to covariation. If we try to account for all the possibilities in fitting models to any practical data set, we are likely to end up with an unidentified model (i.e., there are more unknowns than information in the data). The result is that even some cherished designs, often touted to untangle cause and effect, may be subject to equivocal interpretation. We illustrate the principle with two examples.

First, consider a longitudinal study of phenotypes in which changes in personality always precede changes in the disorder. This evidence is consistent with the personality-causes-disorder hypothesis but is insufficient to prove that hypothesis. A common causal factor may be responsible for the covariation between the two. For example, return for a moment to the example of dopamine levels in the striatum and the basal ganglia. Suppose there is a regulatory gene that changes overall brain dopamine at some developmental stage, a change first felt in the striatum and later in the basal ganglia. This could give the same correlational pattern. Hence, genes may function as an exogenous third variable that confounds the causal interpretation of panel data (Kenny, 1975; Rogosa, 1980).

As a second example, consider introversion and schizophrenia. Suppose that schizophrenics are more introverted than average but that the discordant MZ and DZ cotwins of schizophrenics are no more introverted than the nonschizophrenic population. This might imply that schizophrenia causes intro-

Figure 2. A direction-of-causation model for the strong hypothesis that a personality trait causes a disorder.

version, but once again, the evidence cannot rule out a unique environmental common factor, such as a virus, that jointly influences schizophrenia and introversion.

These examples should instill a humble appreciation for what can and cannot be done with correlational data in terms of inferring causal pathways between personality and psychopathology, but they should not lead to nihilism. Although it may not always be possible to prove a certain model with correlational data, it may be possible to reject certain simple models with the same data. For example, the hypothetical example about schizophrenia and introversion given in the previous paragraph can be used to reject the hypothesis that the only reason that introversion and schizophrenia are correlated is because introversion is a causal factor for schizophrenia. It has recently been shown that genetic data—indeed, family data in general—are especially useful because they may reject simple causal models that cannot always be rejected with cross-sectional data on individuals. Along these lines, we now explore the testable direction of causation models.

Testable Models

Here we offer a nontechnical introduction to the topic of direction of causation. Heath et al. (1993) and Duffy and Martin (in press) have provided comprehensive overviews of the subject and their articles should be consulted for more detail, particularly if one wants to apply these models to data. The direction-of-causation approach is general in that it can be used for any hypothesized causal risk factor for a disorder (or any two phenotypes for that matter). It is not restricted to personality.

The phenotypically driven model implied by Eysenck, Meehl, and Cloninger is illustrated in Figure 2 for a pair of relatives. Depicted are the personality phenotypes of two relatives (P_1 and P_2 for Relative 1 and Relative 2, respectively) and the

Figure 3. A direction-of-causation model for the strong hypothesis that a disorder causes a personality trait.

phenotypes for a disorder for the two (D_1 and D_2). It is assumed that all variables are standardized. An uppercase U denotes a residual variable. Here, there are two residuals, one for the disorder in Relative 1, the other for the disorder in Relative 2 (U_{D_1} and U_{D_2}).

Path b is the causal pathway between the P and D of an individual and also gives the predicted correlation between the personality trait and the disorder. The personality traits of the two relatives are correlated via path a . The disorders in the two relatives are correlated through two pathways—first because personality causes the disorder and because personality is correlated in relatives (pathway ab^2), and second because there is a correlation between the residuals for the disorder (path d^2).

The correlation between the personality trait in one relative and the disorder in the second relative is the data point that can refute the strong causal hypothesis. This predicted correlation is ab , or the product of the correlation between the personality trait and disorder within an individual and the correlation between relatives for the personality trait. For example, suppose the personality trait were impulsivity and the disorder were conduct disorder (CD). If the correlation between impulsivity and CD were .60 within an individual and the sibling correlation for impulsivity were .25, then the predicted correlation between impulsivity in one sibling and CD in the other sibling should be $.25 \times .60$, which equals .15. If the observed correlation differs significantly from this value, then the strong personality-causes-disorder hypothesis can be rejected.

The utility of the direction-of-causation model can be seen if we compare the above hypothesis to another strong hypothesis, namely, that impulsivity is correlated with CD only because it is a symptom of the disorder. Here, the causal pathway should originate in the disorder and point to the personality trait. Figure 3 depicts this model. Again, Path b gives the predicted correlation between impulsivity and CD. The correlation for CD in siblings will be e , say, .40 in this example. As in the impulsivity-

causes-CD model, the critical correlation remains the one between impulsivity in one sibling and CD in the other sibling. But in this causal model, the predicted correlation will be the product of the correlation between impulsivity and CD within an individual and the sibling correlation for conduct disorder, or $.40 \times .60$, which equals .24. If the observed correlation differs from this value, then a strong disorder-causes-personality model can be rejected. Hence, with large sample sizes, either the impulsivity-causes-CD model or the CD-causes-impulsivity model will be rejected because the models make different predictions about the correlation for impulsivity in one relative and CD in the other relative (provided that the sibling correlation for impulsivity differs from that for CD).

Genetic data may be used to reject more complicated models. For example, if the genetic architecture behind impulsivity is sufficiently different from that of CD, twin data could be used to reject the hypothesis that there is reciprocal causation between impulsivity and CD—that is, that impulsivity causes CD and at the same time is a symptom of CD. This topic is beyond the scope of the current discussion; interested readers should consult Heath et al. (1993).

From the numerical values used in these examples, it is clear that large sample sizes would be required if data were gathered on ordinary nuclear families, in which the personality correlations are around .20. This is where identical twins become valuable. MZ correlations for personality and for psychopathology are often twice that of first-degree relatives, substantially increasing statistical power.

Finally, we note that direction-of-causality models are not restricted to relatives. For example, dyads of best friends or even whole peer groups could be used in place of relatives.

An Illustration

To illustrate direction-of-causation models, we reanalyze data from the Australian twin registry on Eysenck's neuroticism and state symptoms of anxiety and depression (described earlier). The actual data were the intraclass covariance matrices calculated from the mean squares and cross-products matrices presented by Eaves et al. (1989, p. 303).

Three simple causal models were fit to the data. The first model (depicted in Figure 4) tested the strong hypothesis that neuroticism causes anxiety and depression. Here, subscripts 1 and 2 refer to Twins 1 and 2 respectively, N denotes the neuroticism phenotype, and A and D denote the phenotypes for anxiety and depression, respectively. Variables with an uppercase U denote residuals, with subscripts associating the residual with its observed variable. Path c permits a correlation between the residuals for anxiety and depression within an individual. The remaining correlations, denoted by the subscripted r s, give the cross-twin correlations. For example, r_N is the correlation between neuroticism in Twin 1 and Twin 2 and r_{AD} is the correlation between the residuals for anxiety in Twin 1 and the residuals for depression in Twin 2.

The second model tests the strong hypothesis that the two disorders cause the personality trait. It is depicted in Figure 5. For convenience, the same symbols are used to denote path coefficients and correlations in Figures 4 and 5, but of course, they take on different numerical values in the data analysis.

Figure 4. A direction-of-causation model for twin data in which neuroticism (N) causes symptoms of anxiety (A) and depression (D).

Finally, we tested the strong hypothesis that the three phenotypes—neuroticism, anxiety, and depression—are correlated through a common phenotypic factor that runs in families. This model is depicted in Figure 6. Here, *F* denotes the latent phenotypic factor, with *r_F* being the correlation between the latent factor of Twin 1 and that of Twin 2. The residuals for a trait are permitted to be correlated across twins (e.g., *r_N*). We do not permit cross-trait correlations for the residuals in relatives (e.g., between *U_{N1}* and *U_{A2}*). If such correlations occurred in relatives, they would probably occur within individuals, thus violating the assumptions of a single-factor model.

We fitted these models to the data separately for male and female same-sexed twins. For each sex, we fitted six standard deviations, one each for neuroticism, anxiety, and depression in the MZ and in the DZ twins. We assumed that the path coefficients

Figure 5. A direction-of-causation model for twin data in which anxiety (A) and depression (D) cause neuroticism (N).

Figure 6. A direction-of-causation model for twin data in which a phenotypic factor (*F*) causes neuroticism (*N*), anxiety (*A*), and depression (*D*).

and correlations within individuals were the same in MZ and DZ twins (e.g., in Figure 4, we constrained *a*, *d*, and *c* to be equal across zygosity), but we permitted the cross-twin correlations to differ for MZ and DZ twins (e.g., in Figure 4, we fit two *r_N*s, one for MZ and one for DZ twins). An important, but untested, assumption in the analysis is that the short-term reliabilities of the three variables are approximately equal.

Table 4 presents the chi-square goodness-of-fit results for the three models for the male and female twins separately and together (which is the sum of the chi-squares for the sexes separately). Large and significant chi-square values suggest that models fit poorly and should be rejected. Clearly, Model 2 (the disorders-cause-neuroticism model) can be rejected with considerable confidence. Neither of the other models, however, gave particularly satisfactory fits. The personality-causes-disorder model can be rejected in men and in the sexes together but gave a nonsignificant, albeit poor, fit in women ($.10 < p < .15$). The phenotypic factor model can be rejected in women and in the sexes together, with a trend toward rejection in men.

If these results are not due to differential reliability of the measures, then the causal relationship between personality and psychopathology may not be a simple one (see Clark, Watson, & Mineka, 1994 [this issue], for a related discussion of anxiety,

Table 4
Goodness-of-Fit Chi-Square Values for Three Causal Models of Neuroticism (*N*) and Symptoms of Anxiety (*A*) and Depression (*D*)

Model	Men	Women	Men and women
N causes A and D	15.65**	11.75	27.40**
A and D cause N	32.18****	73.98****	106.16****
Common phenotypic factor	12.42*	17.80**	30.22***

Note. There are 7 degrees of freedom for the sexes separately and 14 for the sexes together.

* $p < .10$. ** $p < .05$. *** $p < .01$. **** $p < .001$.

depression, and personality). Perhaps several of the five different conceptual sources of covariation are operating at the same time. For example, there may be both a causal factor and a personality-causes-disorder pathway. Or it is possible that factor models are appropriate but that there are different genetic and environmental factor patterns, giving a poor fit to the phenotypic factor model.

Discussion

Genetic factors have a large effect on personality traits. They have a major impact on the variance of an individual trait, on the covariance across traits, and on the general structure behind traits. Heredity is also important in most forms of psychopathology. Given the strong theoretical link between personality and psychopathology and the importance of genetics for both phenotypes, it comes as a surprise that there is little multivariate genetic data relating the two. Such research, however, is in progress (e.g., DiLalla, Gottesman, & Carey, 1993), and greater insight into the relationship can be expected within the next few years. The few available data, however, suggest that heredity is an important component in the correlation between personality and some types of psychopathology. The Australian twin data suggest that genes may be the major contributors to the correlations among neuroticism, anxiety, and depression.

The manner in which genes contribute to this correlation, however, is not understood. The analytical model we explicated earlier suggests three formal mechanisms behind the genetic correlation. First, the correlation may be induced through phenotypic causation. For example, if personality causes a disorder, all the genetic polymorphisms behind the personality trait will also contribute to the heritability of the disorder. However, genes do not induce correlations between personality and disorder. Just the opposite—the causal link between personality and disorder induces the genetic correlation.

The second mechanism is causal pleiotropism, in which some loci contribute to a biological system that has a direct causal influence on personality and also a direct causal influence on disorder. Pleiotropism cannot be lightly dismissed. For physical traits in which the biological mechanisms of gene action are well understood, pleiotropism has been so often encountered that the eminent geneticist Sewall Wright concluded that “the available evidence indicates that pleiotropy is virtually universal” (Wright, 1968, p. 61). Mendelian disorders give abundant evidence for pleiotropism for human behavior. The gene for Huntington’s disease, for example, produces both motoric aberrations and dementia. The extent to which pleiotropism contributes to variation for more common behavioral phenotypes is unknown.

The third formal mechanism is that of correlated genetic values, but it is probably unimportant for personality and psychopathology. The forces that induce such a correlation of genetic values (linkage disequilibrium, assortative mating, and the mixture of genetically heterogeneous populations) may influence some forms of psychopathology, but their influence is probably very small for personality.

Until individual genes can be detected and their mechanisms of action related to behavior, the role for family and genetic data in distinguishing among these mechanisms is modest—the re-

jection of simple causal models. However, in view of the number of causal mechanisms behind an observed correlation, it is unclear whether other correlational designs in psychology can accomplish much more. We suspect that it will take a synthesis from a number of different research designs—quasi-experimental studies, prospective longitudinal studies, and so forth—to arrive at a reasonable model for the causal relationship between personality and psychopathology. Genetic designs will be an important part of this synthesis.

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