CHAPTER 1

Genetics of Personality

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INTRODUCTION

In this current chapter, we review findings from an important approach to understanding the etiology of personality—behavior genetics. Behavior genetics encompasses a series of methods for disentangling the relative influence of genes and environment on the variation in a phenotype (observed variable). Personality, or the characteristic ways that people think, feel, and behave, varies considerably within and between different populations. There are people who are more or less aggressive, more or less modest, more or less sociable, more or less humorous, and so on. It is the variation in personality that defines us and makes us distinct from every other individual on the planet. There are certainly those who have questioned the existence of consistent personality dispositions (Mischel, 1968), or theorized as to why any variation in personality exists (Penke, Denissen, & Miller, 2007). Behavior genetics is vitally important in showing that personality does exist, that it does vary in meaningful ways between people, and that this variation is due, in part, to genetic influences.

Indeed, the field of behavior genetics, particularly the use of genetically informative family data, was a driving force in establishing the importance of both nature and nurture in the development of personality. Quantitative behavior genetic methods, which parse out the relative influence of genes, family environment, and unique environmental experiences, have consistently demonstrated the importance of genetic influences. Biometric modeling with twin data reveal one of the most reliable findings in behavior genetics and in the personality literature—heritability, or proportion of variance due to genetic influences, for most major personality traits is approximately 40 to 50%. A robustly replicated finding, this has caused consternation in the molecular genetic field of personality research, where scientists have been largely stymied in their attempts to find measured genes that explain a significant portion of the variance in personality traits. Why, they have asked, is it so difficult to replicate measured gene-measured personality findings, if we know that genetic influences explain 50% of personality variation? In the current chapter, we review what has been found, and how improving technology may partly solve the problem. We also note that part of the difficulty for molecular genetics is the complex interactions that might be occurring, not only between different genes but also between genes and the environment. Gene-environment interaction, or the impact the environment has on the expression of genetic influences, may be important in our search for the etiology of personality. It might be a return to quantitative genetic methods, which can provide estimates of gene-environment interplay, which best informs our understanding of personality development. Thus, the field of behavior genetics comes full circle, from an early reliance on quantitative methods that set the stage for molecular work, which again must now rely on statistical modeling of family data to move forward. In the current chapter, our goal is to present a general conceptual overview of behavior genetics methodology, including both biometric models and molecular genetics techniques, as well as a snapshot of seminal work from the past, current issues and controversies, and recommendations for future research.

BEHAVIOR GENETICS OF PERSONALITY

We begin by briefly reviewing the major biometric modeling approaches and the assumptions underlying them, before turning to a broad overview of findings from univariate and multivariate twin studies and adoption studies.

Biometric Modeling Approaches

Much of the classic work in the behavior genetics of personality has been conducted with biometric modeling of twin data. These statistical models are *biological* in that they utilize known genetic relationships among individuals, and *metric* because they attempt to provide estimates of genetic and environmental influences based on careful observed measurement of a phenotype. Biometric modeling of personality data is based on certain assumptions about the etiology of personality. As with other types of individual difference phenotypes (e.g., psychopathology), the etiology of personality is probably best explained by a multifactoral polygenic model of inheritance. According to this model, there is a continuum of liability throughout the general population resulting from genetic and environmental influences that act in an additive manner (Falconer, 1965; Gottesman & Shields, 1967). Given that behavior genetic models generally assume there are multiple gene systems, or quantitative trait loci (QTLs), underlying complex behavioral phenotypes like personality, quantitative genetic research (i.e., biometric modeling) is therefore appropriate for examining the etiology of personality. The method most commonly used for examining genetic and environmental influences on the variation in personality is the twin study, therefore much of our review will spotlight this method and the findings from twin studies; adoption methods have also been utilized and we discuss findings from this work later in the chapter.

Twin pairs are a fascinating form of natural experiment, allowing researchers to disentangle the relative influence of genetics and environment on a phenotype, like personality. Identical (monozygotic, MZ) twins are the result of one fertilized egg splitting in two while in utero. Fraternal (dizygotic, DZ) twins are the result of two separate eggs being fertilized at the same time, and are no more alike (genetically) than two nontwin siblings. MZ twins thus share 100% of their genes, while DZ twins share an average of 50% of their segregating genes. When using a sample of MZ and DZ twin pairs in which both members of the pair are raised in the same home, the degree of genetic *and* shared family environment is known. Biometric modeling can then use the concordance (agreement) between twins on a phenotype of interest to decompose the variance in that phenotype into genetic and environmental components. As a first step, one can estimate correlations between twin pairs and compare differences in the magnitude of correlations between MZ and DZ twins to obtain a general indication of the size of genetic and environmental influences. For instance, when the MZ correlation is greater than the DZ correlation, this indicates the presence of genetic influences on the trait. Formal biometric modeling of twin data is done using structural modeling software (e.g., Mx; Neale, Boker, Xie, & Maes, 2003), by comparing the similarity (i.e., the covariance) within MZ and DZ twin pairs on the phenotype, resulting in estimates for genetic and environmental influences. In this section, we focus on univariate twin studies, which decompose the variance of one phenotype (i.e., one personality trait); however, structural modeling is readily extended to the multivariate case (e.g., the structure of multiple personality traits considered together, discussed below).

A univariate ("one variable") biometric model with twin pairs is used to separate the variation in a phenotype into three sources that collectively account for the total variance in the population (Plomin, DeFries, McClearn, & McGuffin, 2008). This is an important point, and often lost when discussing biometric models; these models explain variation in the population from which the sample is drawn, not the relative influence of genes and environment on any one person's outcome. The first source of influence on variation is heritability (abbreviated h^2), which reflects how much of the variation in a personality phenotype is due to genetic differences between people in a population. The heritability estimate is actually a ratio, or a proportion of genetic variation over total variation (the sum of genetic and environmental variation). Importantly, the heritability statistic and the commensurate estimates for environmental influences are population parameters; when we conclude that the heritability of a personality trait is 50%, what we are actually saying is that genetic differences *among people in the sample drawn from that population* account for 50% of the variance in that trait; again, we are not saying that genes account for 50% of any one individual's personality. In other words, among any population of individuals, some will be more extroverted and some will be more introverted and most will cluster around the mean, and 50% of the reason for this variation is that genetic influences also vary among people. Most biometric models of personality assume that genetic influences are additive, meaning that personality variation is due to the influence of many genes of small effect size located at different places (loci) on the genome. There is

evidence for nonadditive genetic effects on normal personality traits (e.g., Keller, Coventry, Heath, & Martin, 2005) as well.

Beyond genetic influences, a second source of variation in a phenotype is the effect of the shared or common environment, abbreviated c^2. This component of variance captures the extent to which twins are similar by virtue of growing up in the same household. Examples of the shared environment include neighborhood influences, socioeconomic status, having similar friends or peer groups, customs, habits, and the extent to which siblings have similar interactions with their parents. A final source of phenotypic variance is the unique or nonshared environment, abbreviated e^2. This component of variance indexes the extent to which twins are different from each other despite having grown up in the same household and sharing genes. Examples of nonshared environmental experience include traumatic events and stressors, having different friends and life experiences from one's sibling, events in utero, and the extent to which each sibling has a unique experience with their parents. It is important to note that measurement errors are included in the estimate of nonshared environmental influences, so any imprecision or bias in measurement among individuals will result in inflated estimates of e^2. The distinction between the two environmental sources of variance can often be subtle. As noted above, neighborhoods are often thought of as shared environmental influences, working to make siblings within the family more similar to each other; however, one sibling's experience or perception of the environment may be quite unique to him or her and thus work to make siblings growing up within the same family less similar to each other (and this would be accounted for under the nonshared environmental component of variance).

Findings From Univariate Twin Studies of Personality

For decades, researchers, the media, and the lay public debated the relative influence of nature versus nurture on the development of individual differences in human behavior. Thanks to decades of work from the field of behavior genetics, we can now definitely say that *both* nature and nurture are at work when we consider the etiology of almost any phenotype that differs between people and would be of interest to psychologists. Indeed, virtually every phenotype that can be said to differ in meaningful ways between individuals in the population has a detectable genetic component, a finding so well-replicated that it has been called the "First Law" of behavior genetics (Turkheimer, 2000). This is certainly true for almost every major personality trait or domain, as research across different populations, cultures, and personality measures has found heritability estimates of approximately 50%, with the rest of the variance primarily attributed to nonshared environmental influences. Given the prominence of trait models of personality in general and the Five-Factor Model/Big Five Model in particular (McCrae, Gaines, & Wellington, this volume), it is not surprising that behavior genetic modeling has focused on these personality domains (extraversion, openness, agreeableness, conscientiousness, and neuroticism). The heritability of all five domains range from 40 to 50%, with a majority of the rest of the variance accounted for by nonshared environmental influences (Bouchard & Loehlin, 2001). It is compelling that parameter estimates of genetic and environmental influences are so consistent across these major personality domains, which obviously differ widely in the aspects of human behavior that they capture.

There are several aspects of these key findings that bear further discussion. First, all biometric models with twin data are built on the equal environments assumption (EEA). This assumption derives from the following logic: biometric modeling compares the similarities between MZ twin pairs to the similarities between DZ twin pairs to arrive at estimates of genetic and environmental components of variance. We infer that greater similarities between MZ twin pairs is due to greater genetic similarity, since in both MZ and DZ pairs, twins are raised in the same environment (if they are not reared apart). But what if MZ twin pairs are more similar because their parents impose more similar treatment on them, compared with parents of DZ twin pairs, in ways that do not reflect the contributions of the MZ twins' genes to parental treatment? If this were true, it could result in biased estimates of genetic effects. However, there is now substantial evidence supporting the EEA (Goodman & Stevenson, 1991; Loehlin & Nichols, 1976; Scarr & Carter-Saltzman, 1979); even when the environment does seem biased to making MZ twins more similar (i.e., they are dressed the same by their parents), this does not appear to have a major influence on phenotypic similarity for individual difference phenotypes like personality.

Second, there is a great deal of consistency in the heritability estimates for a wide range of personality traits. As noted above, the domains of the Five-Factor Model (FFM) of personality, which encompass very different and nonoverlapping aspects of personality, show robustly similar levels of genetic influences (Jang, Livesley, Angleitner, Riemann, & Vernon, 2002; Jang, Livesley, & Vernon, 1996; Jang, McCrae, Angleitner, Riemann, & Livesley,

1998; Yamagata et al., 2006). Further, significant estimates of genetic influence have been found in both adults (Livesley & Jang, 2008) and children (Coolidge, Thede, & Jang, 2001) for dimensional measures of pathological personality and categorical personality disorder diagnoses, It is important to note that most heritability estimates of personality traits are "additive," that is the contributions of many genes of small effect "add up" in their influence on phenotypic variation. Certainly it is possible that more complex nonadditive genetic effects (such as the interaction of measured genes with each other) are at work, and may in part explain the difficulty with finding measured genes for personality.

Third, estimates of shared environment influences are often negligible, which would suggest that one's family has little to no impact on personality above and beyond shared genetics. Many have concluded from this finding that the family a person grows up in has no influence on personality, or even that it serves to make siblings from the same family less similar, not more. There are some exceptions to this finding, including evidence of significant shared environment on the personality trait of altruism (Krueger, Hicks, & McGue, 2001) or when personality traits are rated by previously unacquainted observers (as opposed to self-report or report by knowledgeable informants; Borkenau, Riemann, Angleitner, & Spinath, 2001). Further, it is possible that elements of the environment that might look shared from the outside actually have an interactive influence on personality, such that family-level factors (e.g., the kinds of relationships people have with their parents) either enhance or suppress genetic effects on personality. With standard biometric models, these interactive effects would be captured in heritability or nonshared environmental estimates; newer biometric moderation models have started to capture greater levels of shared environment at extreme ends of certain environments (a topic we return to later, below).

Finally, it is important to remember that most of the work thus far on the behavior genetics of personality has utilized self-report methods of personality in adult twin samples. The reliance on self-report, in particular, has methodological implications for estimates of heritability. Reliance on a single observer enhances measurement error; when self-reports are supplemented by peer or observer reports, heritability estimates increase (e.g., Wolf, Angleitner, Spinath, Riemann, & Strelau, 2004). This work is now being extended to child and adolescent samples, where both self- and observer report of personality is collected. Isen, Baker, Raine, and Bezdjian (2009) reported substantial heritability estimates for only two (self-directedness and harm avoidance) of the scales from the Junior Character and Temperament Inventory, using a sample of 9- to 10-year-old twins. What was also striking about their findings was the presence of substantial shared environmental effects for two other scales—novelty seeking and cooperativeness. In a different study that utilized a sample of toddlers, the authors found that variation in the temperament dimension of inhibitory control (IC) was 38% genetic and 62% nonshared environment when observer ratings were used, but 58% genetic, 26% shared environment, and 16% nonshared environment when parent ratings were used in the analyses (Gagne & Saudino, 2010). It remains to be seen whether these effects of rater and developmental period on estimates of genetic and environmental influences will replicate in future studies.

Multivariate and Longitudinal Twin Studies

Establishing the relative influence of genetics, shared environment, and nonshared environment on different kinds of personality traits was an important contribution of behavior genetics. However, the utility of this method does not stop once we establish heritability for a personality trait. The univariate biometric model can be readily extended to encompass multiple phenotypes, or personality traits, a technique known as *multivariate* biometric modeling. Multivariate biometric modeling has many uses, which we will discuss in more depth below. First, it can be used to examine the etiological structure of the architecture of multivariate personality space. That is, just as univariate biometric modeling decomposes the variance of a trait into genetic and environmental components, multivariate biometric modeling can decompose the covariance between two or more personality traits. This type of modeling results in estimates of the genetic and environmental contributions to each individual phenotype and the amount of overlap between these sources of variance. Typically, this overlap is represented by a correlation; for instance, a genetic correlation (varying from -1.0 to $+1.0$) indexes the degree to which the genetic influences on Personality Trait A overlap with the genetic influences on Personality Trait B. Similar correlations are computed for shared and nonshared environmental influences. Of note, these correlations can be subjected to factor analysis in the same way that phenotypic correlations between personality variables can, and resulting analysis provide insights into the structure of etiological influences.

The second extension of multivariate modeling would be to include an environmental variable in this multivariate modeling, so that it is possible to determine the

etiological overlap between personality and putatively causal environmental variables. This type of modeling has important implications for finding the nonshared environmental factors that make up approximately 50% of personality variance. Third, the etiology of personality over time can be examined by including measures of the same personality traits collected over multiple points in time. If, for instance, the genetic correlations among personality traits assessed at different ages were high, this would suggest that stability of personality is due to genetic influences. Finally, there are relatively new extensions of multivariate modeling that allow for examination of not just etiological overlap between personality traits, but interactive effects between personality and the environment.

There are several multivariate models that researchers can utilize for this work, discussed below in order of increasing complexity (Neale & Cardon, 1992). The simplest model is a Cholesky decomposition, which decomposes the genetic and environmental variance shared in common between two or more phenotypes. Figure 1.1 presents an illustration of a Cholesky model for three phenotypes (e.g., three different personality traits). For simplicity, only the genetic (A) and nonshared environmental (E) components of variance are presented in Figure 1.1, but shared environmental (C) influences are easily incorporated as well. This model is most useful for estimating the amount of shared influences between multiple phenotypes (i.e., the genetic and environmental correlations), but tells us relatively little about why those influences might be shared, in the sense of delineating latent constructs

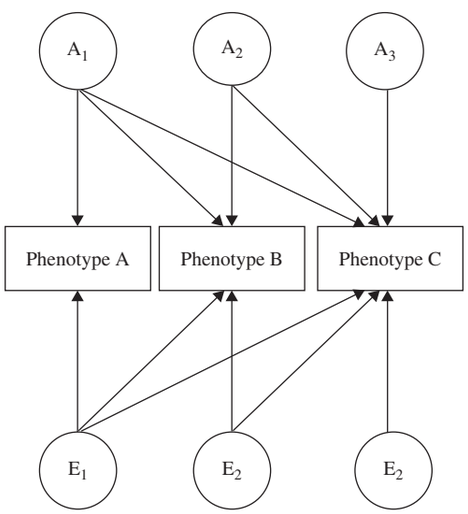

Figure 1.1 Path model for a bivariate (Cholesky) decomposition of variance into additive genetic (A) and nonshared environmental (E) sources

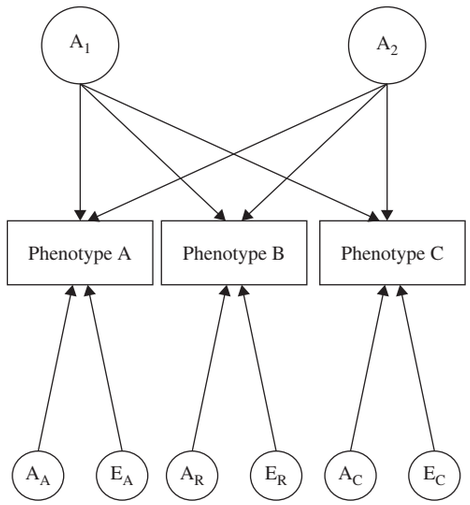

Figure 1.2 An independent pathways model

that connect multiple manifest phenotypes. For that, we turn to an additional multivariate model, the *independent pathways* (IP) model (see Figure 1.2), which differs from a Cholesky by positing direct paths from a common latent genetic factor to all of the observed variables, as well as allowing for specific genetic influences that are unique to each variable. There are commensurate general and specific factors for shared and nonshared environmental influences as well. Both the IP model and the Cholesky model are able to estimate shared etiological influences among multiple personality traits. The IP model, however, imposes a greater amount of structure on the etiological influences on personality, allowing each observed personality variable to have both general and specific genetic and environmental influences. The final multivariate model, the *common pathways* (CP) model (see Figure 1.3), goes even farther than the IP model in positing a known structure for the etiology of multiple personality domains. The CP model suggests that there is a single latent construct that accounts for the covariance among multiple personality traits, and the variance in this latent factor can be decomposed into additive genetic, shared environmental, and nonshared environmental influences. The strictest of the three models, the CP model is the closest biometric extension of a phenotypic factor analysis, and should provide the best fit to the data if the personality domain is truly an etiologically coherent underlying dimension.

Covariation Among Personality Traits

One of the most important uses of multivariate biometric models is to investigate the etiological structure of personality. On a phenotypic level, factor analysis has been

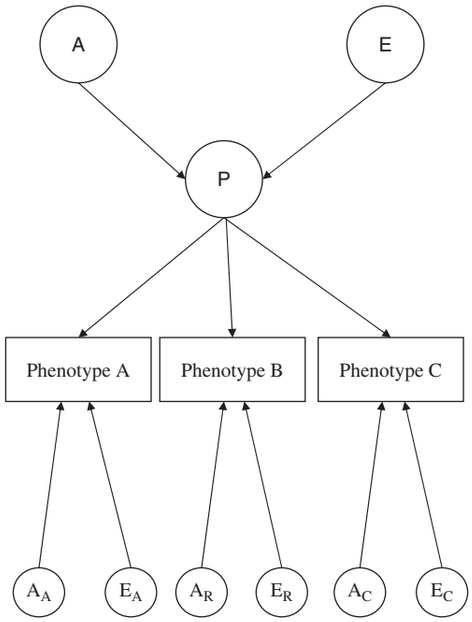

Figure 1.3 A common pathways model

widely used to examine how many and which personality traits are needed to capture all of the multivariate space. Multivariate biometric modeling can be used to determine if the genetic structure of personality parallels the phenotypic structure. The FFM, for example, is often posited to represent basic biological tendencies that account for all of the variation in normal and pathological personality. If multivariate biometric modeling found that the genetic and environmental structure of the FFM facets and domains was best represented by a biometric common pathways model, this would be strong support for Costa and McCrae's conceptualization of the FFM domains as biologically based unitary latent constructs (McCrae & Costa, 2008). Although multivariate biometric modeling has been applied to other models of personality structure, the FFM has certainly dominated this work so we concentrate our review on biometric modeling applied to the FFM.

The results from this work suggest that the etiological influences on the structure of personality are quite complex. Jang and colleagues (2002) used a combined German and Canadian adult twin sample to compare the IP and CP models when applied to lower-order personality traits subsumed under the FFM domains. They found that the lower-order facets of the NEO-PI-R (Costa & McCrae, 1992) did not load cleanly onto five genetic factors (i.e., one for each of the FFM domains, as would be expected for the CP model). Instead, the IP model provided the best fit to the data, and each of the FFM higher-order domains required two genetic and two nonshared environmental factors. This led the authors to conclude that the FFM domains "do not exist as veridical psychological entities per se, but rather they exist as useful heuristic devices" (Jang et al., 2002, p. 99). Johnson and Krueger (2004) later examined the multivariate structure of the Big Five domains in a nationwide sample of American twins using adjectives taken from existing trait inventories. They found that the CP model best fit the data for extraversion and neuroticism, suggesting that of the FFM domains these two come closest to being unitary latent personality constructs. In contrast, the IP model best fit conscientiousness and openness, while the Cholesky model provided the best fit to the data for Agreeableness, perhaps reflecting a looser organizational etiological structure for these domains. However, in contrast to these earlier findings, a later study supported the robustness of the etiological structure of the FFM. The authors found that factor analysis of genetic and environmental correlations among the NEO-PI-R facets resulted in five, genetically robust domains that paralleled the FFM higher-order traits (Yamagata et al., 2006).

One possible interpretation of these somewhat contradictory findings is that the FFM does not completely capture the true nature of multivariate personality space. However, the FFM does exhibit greater etiological coherence than other personality models. For instance, Ando and colleagues (2004) found that there was little correspondence between the phenotypic and genetic structure of the Temperament and Character Inventory (TCI; Cloninger, Svrakic, & Przybeck, 1993), challenging the distinction between temperament and character as posited by Cloninger's model of personality. A more optimistic interpretation of these findings is that personality is truly hierarchically structured, and that the facet-level traits of the FFM are innate tendencies that have specific genetic and environmental influences of their own (Jang et al., 1998). A consequence of this conclusion is that current personality inventories may not adequately capture etiologically coherent personality constructs. In fact, when a multiple-rater model is used to explicitly account for rater-bias in measurement, greater genetic variance is found in NEO-PI-R domains and facets (Kandler, Riemann, Spinath, & Angleitner, 2010b). In future research, it may be necessary to use an iterative process whereby the results of biometric modeling are used to inform personality inventories and assessment instruments that can then capture and identify more etiologically "pure" personality traits. Only by better refining personality domains and the measures used to assess them will it be possible to identify "genetically crisp categories" (Farone, Tsuang, & Tsuang,

1999) of personality variation that may finally solve the problem that molecular geneticists face of identifying replicable measured gene-personality trait associations (Ebstein, 2006).

Etiological Links Between Personality and Psychopathology and Other Outcomes of Public Health Relevance

A second important use of multivariate biometric modeling is to examine the shared etiology between personality and important health-relevant outcomes. One of the most important of these outcomes is psychopathology. There is strong phenotypic evidence of associations between various personality traits and many different forms of psychopathology. For instance, the personality trait of neuroticism is a correlate of many different disorders, particularly mood and anxiety disorders (e.g., Griffith et al., 2010). Biometric models are an important method for determining whether these links are the result of shared genetic or environmental influences between personality and psychopathology.

There are several different models that attempt to explain the association between personality and psychopathology (for a recent review, see South, 2011, p. 509). Briefly, these models differ in the causal ordering of the associations between personality and psychopathology syndromes. The vulnerability model, for instance, presumes that certain personality traits convey vulnerability toward certain types of mental illness. However, the model that has been arguably most well supported in both the child and adult literatures posits that different psychopathology syndromes and personality traits are related by virtue of being components of a higher-order spectrum. Research is now converging on a model that posits that mood and anxiety disorders are indicators of a higher-order spectrum of Internalizing pathology, while antisocial, conduct-disordered behavior and substance use syndromes are indicators of a higher-order Externalizing spectrum (see Krueger & Markon, 2006). With regard to personality, neuroticism can be included in the internalizing factor, while disinhibtion fits well within the externalizing spectrum.

Importantly, multivariate biometric models can determine whether the phenotypic structure found for the Internalizing-Externalizing model of psychopathology extends to the etiological structure. Krueger and colleagues (2002) used a twin sample of more than 600 adolescents and concluded that a CP model best explained the covariation among adolescent antisocial behavior, conduct disorder, alcohol dependence, drug dependence, and the personality trait of constraint (reverse scored). Singh and Waldman (2010) examined the structure of externalizing phenotypes (i.e., negative emotionality and childhood externalizing disorders) in a sample of 4- to 17-year-old twins. In contrast to Krueger et al., they found that the IP model provided a better fit to the data than the CP model. Differences in findings between these two studies have important implications for the changing etiology of externalizing psychopathology over time. Finally, an analysis of the internalizing spectrum including the trait of neuroticism found two overlapping genetic factors: one factor accounted for the covariation among major depression, generalized anxiety, and panic disorder, independent of neuroticism, while the second factor explained the variation between neuroticism, major depression, generalized anxiety, panic disorder, and the phobias (Hettema, Neale, Myers, Prescott, & Kendler, 2006).

In addition to further elucidating the relationship between personality and psychopathology, multivariate biometric models can also inform the etiology of the associations between personality and its well-known correlates. The basic Cholesky decomposition model can be used with one or more personality traits and an environmental variable that is phenotypically correlated with those specific personality variables. It is then possible to examine *why* a personality trait is associated with a certain outcome variable—is the correlation mediated genetically or environmentally? That is, we can ask whether the overlap between environment and personality is due to genetic influences or environmental influences shared between the two. This type of modeling is possible because evidence now shows that what we think of as putatively "environmental" variables often have a heritable component (Kendler & Baker, 2007; Rowe, 1981, 1983). That is, an adolescent's report of the quality of their relationship with one or both parents is partly heritable (Krueger, South, Johnson, & Iacono, 2008), possibly because genetically influenced personality traits influence the way in which people perceive, or experience, the environment. This is a question that can be directly tested using biometric models, in which a sizeable genetic correlation would suggest that personality and environment are related because of genetic variance shared between personality and the environmental measure. Indeed, there are now many examples of findings in this vein in the literature. For example, Spotts and colleagues (2005) showed that 32% of the total variance in wives' marital satisfaction was shared in common with a personality composite of aggression and optimism. Other work has found links between personality and measures of the current family environment

(Chipuer, Plomin, Pedersen, McClearn, & Nesselroade, 1993) and childhood family environment recalled by adults (Kandler, Riemann, & Kampfe, 2009; Krueger, Markon, & Bouchard, 2003), life events (Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997), parenting behaviors (Spinath & O'Connor, 2003), job satisfaction (Ilies & Judge, 2003), propensity to marry (Johnson, McGue, Krueger, & Bouchard, 2004), and leadership style (Johnson, Vernon, Harris, & Jang, 2004).

Longitudinal Modeling of Personality Over Time

Another important extension of multivariate biometric modeling is the ability to examine genetic and environmental influences on personality over time. Longitudinal biometric modeling can help in understanding whether the stability (or instability) of personality is due to genetic influences that stay constant over important developmental stages. A longitudinal behavior genetic study using a child sample studied from age 3 to 12 examined how genetic and environmental influences contributed to the stability and change in withdrawn behavior (Hoekstra, Bartels, Hudziak, Van Beijsterveldt, & Boomsma, 2008). The authors reported substantial heritability at all ages, particularly at age 3 and age 7; further, genetic correlations between personality traits measured at different ages indicated that similar genetic influences were operating across time. As is usually the case, only modest shared environmental influences were found at every age, but when present they better explained the stability of withdrawn behavior over time in girls (14%) as compared to boys (4%). The estimates of nonshared environment were large and increased from earlier to later ages, but the decreasing size of the nonshared environmental correlations indicated that new unique environmental effects were "coming online" at the different ages. Using an older adolescent and adult sample (age 16 and above at baseline), a recent study compared several different models of genetic and environmental influences on developing personality traits (Kandler et al., 2010a). Similar to prior studies (McGue, Bacon, & Lykken, 1993), they found that genetic influences primarily contributed to the stability of personality over time. Further, long-term changes in personality over young and middle adulthood were attributable to environmental factors. Finally, one longitudinal biometric study decomposed the variance in latent growth curves modeled from data available on twins from average age 17 to 29 (Hopwood et al., 2011). The authors found substantial genetic and nonshared environmental influences on the overall level (i.e., intercept factor) of higher-order personality constructs of negative emotionality and constraint; significant genetic influences were also found for change (i.e., slope growth factor) in constraint but not negative emotionality. This type of work is an exciting example of the possibilities for combining longitudinal and biometric models to explain the etiology of developmental phenomenon.

Biometric Moderation Models of Gene-Environment Interplay

As noted above, there is now abundant evidence of shared genetic overlap between personality and various measures of the environment. This invaluable finding was beneficial in demonstrating that people "make" their own environments; as such, the nature of parent-child, sibling, and marital relationships, for instance, can be explained, at least in part, by the genetically influenced traits of the individuals who comprise those relationships. This phenomenon is known as *gene-environment correlation* (rGE), or the degree to which a person's genotype influences a person's exposure to and experience of certain environments. The direct test of rGE is the magnitude of the genetic correlation between a personality trait and an environmental variable that is estimated in a multivariate biometric model. The presence of a significant and at least moderate genetic correlation from a biometric model would be evidence for rGE.

However, this is only one form of gene-environment interplay between personality and the environment. A different form of gene-environment interplay that has long been posited and studiedis known as *gene X* environment interaction (GxE), or the idea that different environments moderate (that is, enhance or suppress) the genetic and environmental influences on personality (Rutter, Moffitt, & Caspi, 2006). Influential adoption studies of conduct disorder were the first to suggest that pathogenic rearing environments may have a particularly detrimental effect on individuals possessing a genetic vulnerability (e.g., Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995). That is, a person might inherit genetic propensity toward certain personality traits, but the expression of those genetic influences would depend on the context or experience of certain environmental influences. Several well-known examples have now appeared in the literature reporting instances of measured gene X measured environment interactions, including Caspi and colleagues (2002) finding that a variant of the MAO-A gene is linked to antisocial behavior in boys only if they experienced maltreatment as children. Since the publication of a new, elegant biometric model for examining GxE in the presence of rGE (Purcell, 2002), there has been an explosion of

interest in research examining latent gene X environment interaction, or what also called heritability X environment interaction to differentiate it from measured GxE. These new biometric models are also called biometric moderation models, because they specifically allow for moderation of the ACE variance estimates. That is, they test for GxE by allowing heritability (and estimates for shared and nonshared environment) within a population to differ depending on a person's standing on an environmental variable of interest. If we think about a typical univariate biometric model, the resulting heritability statistic that we estimate is constant and specific to the population from which we sampled. In the same way that a mean value averages over differences within the population, a heritability estimate glosses over possible variations that might exist in subsamples of the population. The simplest example of this is gender—when gender is not accounted for, then heritability estimates are the same for men and women; both univariate and multivariate biometric models have been developed to explicitly test for quantitative and qualitative differences in heritability estimates by sex. Biometric moderation models are a logical and practical extension of this design, as they direct testing whether the presence of genetic and environmental influences on a personality trait depend on a person's level of experience of an environmental correlate.

The use of these moderation models for the study of psychopathology is growing. For instance, research has shown that genetic influences on internalizing psychopathology are increased for people with extremely poor marital relationships (South & Krueger, 2008) or decreased for people living in extremely high SES levels (South & Krueger, 2011). The use of moderation models for personality is still relatively new but increasing. For example, in a recent study using a sample of adolescent twins, the authors found that the genetic and environmental influences on higher-order domains of personality (i.e., positive and negative emotionality) varied depending on parent-adolescent relationship quality (Krueger et al., 2008). These results suggest that the etiological influences on personality differ as a function of the context of the family. In similar work, researchers found that genetic and environmental influences on emotional instability (akin to neuroticism) were impacted by perceived level of family conflict and maternal indulgence (Jang, Dick, Wolf, Livesley, & Paris, 2005). Not only did heritability differ depending on level of family variables, but moderation was also found for shared environmental effects, offering the possibility that the lack of c^2 found in many studies may partly result from the effect of GxE. That is not to say that moderation models are a panacea that will always explain the variation in personality. Kendler and colleagues (2003a), for example, found that the genetic and environmental effects on neuroticism were not moderated by aspects of the family environment. Again, this work is relatively new, and more will need to be done and replicated before we can conclude that gene-environment interactions are vital part of understanding the developmental etiology of personality. However, it does offer the possibility of better modeling the richness and complexity of personality; it also may explain why the robust 50% heritability of personality has failed to generate replicable and reliable associations between specific alleles and personality traits.

Adoption Studies of Personality

As noted earlier, a large portion of behavior genetic research on personality has utilized biometric modeling with twin samples. Adoption studies, however, are also an excellent way of taking advantage of genetically informative samples to understand the etiology of personality. Like the twin design, families with an adopted child (or children) are a natural experiment by which it is possible to tease apart genetic and environmental influences on variation in personality. As an example, consider a family that consists of one or more biological children of the parents as well as one or more adopted, nonbiologically related children (i.e., not a kinship adoption). The parents and nonbiological adopted siblings share 100% of the rearing environment, but 0% of their genes, while the parents and biological siblings share 100% of environment and approximately 50% of their genes. As with the twin design, these known degrees of genetic and environmental relatedness can be used to calculate estimates of genetic and environmental influences on personality.

The findings from adoption studies are commensurate with the results of twin studies in finding little influence of the shared environment and substantial nonshared environmental effects; where they differ, however, is that adoption studies generally find smaller heritability estimates than twin studies (Loehlin, Willerman, & Horn, 1987; Plomin, Corley, Caspi, Fulker, & DeFries, 1998). Generally two explanations have been proposed to explain the disconnect in findings between adoption and twin studies. First, it is possible that too much faith is placed in the equal environments assumption (EEA), and that in fact identical twins reared together are more similar to each other because environmental influences encourage it. However, the powerful twins reared apart design has found heritability estimates generally comparable in

magnitude to those found with samples of twins reared together (Bouchard, 1994; Pedersen, Plomin, McClearn, & Friberg, 1988), further refuting possible violation of the EEA.

A more likely possibility to explain the difference between findings from these two different methods is the relative influence of nonadditive genetic effects on personality. Most biometric modeling of twin data operates under the assumption that genetic effects are additive, and among those who test for dominant (nonadditive) genetic effects there are few significant findings. Twin-only designs may in fact be limited in their ability to identify nonadditive genetic effects (i.e., effects due to dominant genetic effects or interactions among specific genes). Keller and colleagues (2005), for instance, found evidence of nonadditive genetic effects using a twin-plus-sibling design. The Nonshared Environment in Adolescent Development (NEAD) study went even further, combining information from MZ and DZ twins, full siblings, half siblings, and genetically unrelated children in the same family resulting from remarriage. When examining broad domains of adjustment, the authors found high heritability estimates, significant shared environmental estimates, and little effect of nonshared environment (Reiss, Neiderhiser, Hetherington, & Plomin, 2000). A later follow-up study from the same sample reported slightly lower heritabilities than were found in the original study, but again reported significant nonadditive genetic effects (Loehlin, Neiderhiser, & Reiss, 2003). In fact, extensions of twin-only or adopted-only family designs may reveal more than just nonadditive influences. A recent adoption study took advantage of a large sample of families consisting of parents and two adolescent siblings, either all biologically related or, in the adoptive families, two adopted siblings or one adopted sibling and one biological sibling (Buchanan, McGue, Keyes, & Iacono, 2009). The authors found that variation in negative emotionality was largely due to nonshared environment and genetic effects; however, when examining the variance in disinhibition, the authors found evidence of significant shared environmental effects (20%). Thus, large samples that include siblings of varying genetic relatedness may be invaluable in finding systematic family influences on personality.

Biometric Modeling of Pathological Personality

Next, we review findings from biometric modeling of pathological personality, including both dimensional measures of pathological traits from different assessment instruments and *DSM*-defined personality disorders.

Univariate Studies

So far, our review has focused primarily on "normative" personality traits; in this section we review behavior genetic findings with regard to more maladaptive personality. Pathological personality traits are incorporated into the formal diagnostic nomenclature through personality disorder (PD) diagnoses on Axis II of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*; American Psychiatric Association, 2000). The *DSM* system specifies 10 PDs grouped into three clusters, A, B, and C, called the odd/eccentric, dramatic/emotional and the anxious/fearful, respectively. Behavior genetic studies have been conducted with the *DSM* PDs using both categorical and dimensional approaches. Heritability estimates vary considerably depending on methodological differences, including population sampled (e.g., clinical versus general population), method of assessment (e.g., questionnaire versus structured interview), and how broadly the disorder is defined. A strong genetic influence on all *DSM-IV* PDs was found in the only twin study of children that has been published (Coolidge et al., 2001). Heritability estimates ranged from 50% (paranoid) to 81% (schizotypal, dependent), and there were no substantial shared environmental effects found for any of the disorders. In a twin study of categorical PD diagnoses based on structured interview in clinical samples of adults, most of whom had severe mental disorders, heritability estimates ranged from 28% (paranoid, avoidant) to 77% (narcissistic and obsessive-compulsive; Torgersen et al., 2000). Again, the best-fitting models did not include shared environmental effects, a finding consistent with behavior genetic work with normal personality traits.

Antisocial PD was not analyzed in any of these studies. However, this disorder has been examined in a number of studies using different phenotypes, and two meta-analytic studies have been published (Ferguson, 2010; Rhee & Waldman, 2002). In both analyses, genetic influences were found to play a major role (heritability 41% and 56%, respectively). Interestingly, both reviews found that shared environmental influences contributed significantly, 16% and 11% respectively. Nonadditive effects explained 9% of the variance in the Rhee and Waldman study.

The heritabilities of dimensional representations of *DSM-IV* PD traits, based on structured interviews, have also been estimated in a population-based study of young adult Norwegian twins. These estimates may be a closer parallel to the behavior genetic findings from models using dimensional ratings of normal personality. Genetic influences were found to be modest to moderate, ranging from 21% (paranoid) to 28% (schizoid) in Cluster A

(Kendler et al., 2006a), from 38% (antisocial) to 24% (narcissistic) in Cluster B (Torgersen et al., 2008), and from 27% (obsessive-compulsive) to 35% (avoidant) in Cluster C (Reichborn-Kjennerud et al., 2007b). No shared environmental effects were identified, and all genetic effects were additive. Thus, heritability of dimensional PD scores tend to be lower, on average, than heritability estimates for broad domains of normal personality.

The studies reviewed above primarily used data from diagnostic clinical interviews. Other studies have also used self-report data. For example, one study using self-report questionnaire data found that schizotypal personality traits had a heritability of approximately 50%, and the role played by shared environmental factors was negligible (Linney et al., 2003). Distel and colleagues studied the heritability of borderline PD traits using questionnaire data in both a twin and a twin-family design. In the twin study, additive genetic factors explained 42% of the variance (Distel et al., 2008), and in the extended twin model additive and nonadditive genetic factors each explained 24% of the variance (Distel, Rebollo-Mesa, et al., 2009). Kendler, Myers, Torgersen, Neale, and Reichborn-Kjennerud, (2007) directly compared data from both questionnaire and structured interview at different points in time to estimate the heritability of the latent liabilities to Cluster A PDs. Heritabilities were substantially higher using both methods (55 to 72%) than when structured interview data alone was utilized. Another method that can reduce measurement error is to use a model in which a common latent factor influences lower order traits of a PD. This has been done for borderline PD by two groups using different self-report data (Distel et al., 2010; Kendler, Myers, & Reichborn-Kjennerud, 2010). As expected, heritability estimates were higher, 51% and 60% respectively.

In addition to the *DSM* categorical system of classification, a number of dimensional classification systems have been proposed for abnormal personality traits, including models which integrate personality disorders with general personality structure (Widiger & Simonsen, 2005). Biometric modeling of pathological personality traits has primarily been conducted with the Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ; Livesley & Jackson, 2001), which includes 18 primary traits and 69 defining facets. The heritability estimates of the primary traits range from 56 to 35%, with the rest of the variance primarily attributable to nonshared environmental effects and no evidence of common environmental effects (Jang, Livesley, Vernon, & Jackson, 1996; Livesley & Jang, 2008). These results resemble those found for normal personality traits (see above).

Few behavior genetic studies investigating the genetic overlap between dimensional measures of pathological personality and normal personality traits as defined by the FFM have been conducted. In a twin study of the 18 DAPP-BQ traits and the FFM traits, the largest genetic correlations were observed with *neuroticism* (median = 0.48), and the lowest with *openness* (median −0.04; Jang & Livesley, 1999). Two more recent studies have examined the etiological overlap between dimensional measures of Borderline PD and the FFM (Distel, Trull, et al., 2009; Kendler, Myers, & Reichborn-Kjennerud, 2010). Both found strong genetic correlations with the FFM traits. Indeed Distel, Trull, et al. concluded that "all genetic variation for Borderline PD is shared with normal personality traits." Further studies are needed to clarify the genetic overlap between the *DSM-IV* PDs and normal personality traits.

Multivariate Studies

As with normal personality traits, behavior genetic modeling can be used with pathological personality traits to achieve several goals, including determining the etiological structure of pathological personality, the genetic and environmental overlap between pathological personality, normal personality, and psychopathology, and the stability of pathological traits over time.

Phenotypic analyses of pathological personality traits assessed by different systems typically identify four factors (Livesley & Jang, 2008). Using principal component analyses, Livesley and colleagues (1998) identified four components in the DAPP-BQ: *emotional dysregulation*, *dissocial behavior*, *inhibitedness*, and *compulsivity*. These higher-order traits strongly resemble dimensions of normal personality. Emotional dysregulation is a broad domain with substantial loadings on 11 of the 18 primary traits. It resembles the normal personality trait neuroticism but is more extensive, including primary traits like identity problems, cognitive dysregulation, oppositionality, suspiciousness, and narcissism. Dissocial behavior resembles the negative pole of agreeableness in the five-factor approach, inhibitedness is similar to introversion, and compulsivity resembles the conscientiousness domain of the five-factor model. Multivariate genetic analyses yielded four genetic and four environmental factors that were remarkably similar to the phenotypic factors. The heritability of the secondary domains were 53%, 50%, 51%, and 38% for emotional dysregulation, dissocial behavior, inhibitedness, and compulsivity, respectively.

There has been only one population-based multivariate twin study including all 10 *DSM-IV* PDs (Kendler et al.,

2008). The best fitting model included three common additive genetic and three common individual-specific environmental factors in addition to disorder-specific genetic and environmental factors. The first common genetic factor had high loadings ($> +0.28$) on PDs from all three clusters, including histrionic, borderline, narcissistic, dependent, obsessive-compulsive, and paranoid, PD. One interpretation of this factor is that it reflects a broad vulnerability to PD pathology and/or negative emotionality; as such it is likely related to genetic liability to the normal personality trait *neuroticism*. In meta-analytic reviews of the phenotypic relationship between *DSM-IV* PDs and the FFM of personality, three of the five PDs with high loadings on this factor (paranoid, borderline, and dependent) were found to be closely linked phenotypically to neuroticism (Samuel & Widiger, 2008; Saulsman & Page, 2004). This factor also resembles the genetic factor reflecting the higher-order trait emotional dysregulation in the DAPP-BQ (Livesley et al., 1998). The second common genetic factor was quite specific, with substantial loadings only on borderline and antisocial PD. This suggests genetic liability to a broad phenotype for impulsive/aggressive behavior. It resembles the second genetic factor identified by Livesley et al., 1998, dissocial behavior. From the perspective of the FFM, our second factor primarily reflects genetic risk for low conscientiousness and low agreeableness (Samuel & Widiger, 2008). Of interest, in a hierarchical analysis of normal and abnormal personality, Markon, Krueger, and Watson (2005) argue that the traits of agreeableness and conscientiousness—those that are indexed by the second genetic factor—are both reflections of a high-order construct they term *disinhibition*.

The third factor had high loadings only on schizoid and avoidant PD. There are several possible interpretations. This factor might, in part, reflect genetic risk for schizophrenia spectrum pathology (see below). From the perspective of the FFM, it reflects genetic liability for introversion (low extraversion). Indeed, avoidant and schizoid PDs are the two PDs most negatively associated with *extraversion* (Samuel & Widiger, 2008; Saulsman & Page, 2004). Obsessive-compulsive PD had the highest disorder-specific genetic loading, which is consistent with prior work demonstrating that this disorder shares little genetic and environmental liability with the other Cluster C PDs (Reichborn-Kjennerud et al., 2007b). These results again parallel findings from the Livesley et al. (1998) twin study, where the fourth genetic factor reflected their higher-order factor compulsivity, which had by far the strongest loading on the lower order trait of the same name, and which resembles the conscientiousness domain of the FFM. In sum, these findings indicate that genetic risk factors for *DSM-IV* PDs do not reflect the Cluster A, B, and C typology; however, the *DSM* cluster structure was well represented by the structure of the environmental risk factors, suggesting that the comorbidity of PDs within clusters is due to environmental experiences (Kendler et al., 2008).

Several lines of evidence indicate that common genetic or environmental liability factors might predispose to several disorders within clusters that transcend the Axis I/Axis II division (Andrews et al., 2009; Krueger & Markon, 2006; Siever & Davis, 1991). A number of family and adoption studies have found significantly increased risk for paranoid, schizoid, and schizotypal PDs in relatives of schizophrenic and control probands. These results suggest that schizotypal PD has the closest familial relationship to schizophrenia, followed by paranoid and schizoid PD. This is consistent with the hypothesis that a common genetic risk factor for Cluster A PDs reflects the liability to schizophrenia in the general population (Kendler et al., 2006a). The term *schizophrenia spectrum* is often used to describe the extended phenotype believed to reflect this genetic liability to (e.g., Siever & Davis, 2004). In a recent family study, Fogelson and colleagues (2007) showed that avoidant PD, currently classified in *DSM* Cluster C, also occurred more frequently in relatives of probands with schizophrenia even after controlling for schizotypal and paranoid PD, suggesting that avoidant PD could also be included in this spectrum. This finding is also in accordance with the results from the multivariate twin study described above, in which avoidant and schizoid PD share genetic liability (Kendler et al., 2008).

As noted above, mood and anxiety disorders, often grouped under the internalizing spectrum of psychopathology, share genetic and environmental liability factors with each other (Kendler, Prescott, Myers, & Neale, 2003b), and with normal personality traits (Hettema et al., 2006; Kendler, Gatz, Gardner, & Pedersen, 2006b). In a population-based multivariate twin study of major depression and *DSM-IV* PDs, Reichborn-Kjennerud and colleagues (2010) found that dimensional representations of borderline PD from Cluster B, avoidant PD from Cluster C, and paranoid PD from Cluster A were all independently and significantly associated with increased risk for major depression. Multivariate biometric modeling indicated that one common latent factor accounted for the genetic covariance between major depression and the three PDs. The genetic correlations between major depression and borderline, avoidant, and paranoid PD were

+0.56, +0.22, and +0.40, respectively. This indicates that vulnerability to general PD pathology and/or negative emotionality and major depression are closely related, consistent with results from a number of studies showing that the genetic liability factors for major depression and the personality trait neuroticism are strongly correlated (Kendler et al., 2006b). At the phenotypic level, neuroticism is also closely related to depressive PD, listed in the *DSM-IV* Appendix B. In a bivariate twin study, Ørstavik and colleagues (2007) found that a substantial part of the covariation between major depressive disorder and depressive PD was accounted for by genetic factors, with a genetic correlation of 0.56. Results from another population-based twin study, investigating the sources of co-occurrence between social phobia and of avoidant PD in females, indicated that the two disorders were influenced by identical genetic factors, whereas the environmental factors were uncorrelated (Reichborn-Kjennerud et al., 2007a). This suggests that whether an individual develops avoidant PD or social phobia is entirely the result of environmental risk factors unique to each disorder, which is in accordance with the hypothesis of underlying psychobiological dimensions cutting across the Axis I/Axis II classification system.

Numerous family, twin, and adoption studies have demonstrated that antisocial PD, conduct disorder, and substance use disorders (externalizing disorders) share a common genetic liability (e.g., Kendler et al., 2003b; Krueger et al., 2002). In a family-twin study, Hicks and colleagues (2004) found that a highly heritable (80%) general vulnerability to all the externalizing disorders accounted for most of the familial resemblance. Disorder-specific vulnerabilities were found for conduct disorder, drug dependence, and alcohol dependence, but not for antisocial PD. The same research group has reported an association between externalizing disorders and reduced amplitude of the P3 component of the brain event-related potential, suggesting that this could be a common biological marker for vulnerability to these disorders (Hicks et al., 2007).

Expanding earlier efforts that have provided consistent evidence that common Axis I disorders can be divided into the two broad categories, internalizing and externalizing disorders (e.g., Krueger & Markon, 2006), a recent study used data from the Norwegian Twin Panel to investigate the underlying genetic and environmental structure of 12 syndromal and subsyndromal common *DSM-IV* Axis I disorders and dimensional representations of all 10 Axis II PDs (Kendler et al., 2011). Four correlated genetic factors were identified: Axis I internalizing, Axis II internalizing, Axis I externalizing, and Axis II externalizing. From a genetic point of view, these results provide some support for the decision in *DSM* to distinguish between Axis I and Axis II disorders. The correlation between the two internalizing factors was 0.49 and between the two externalizing factors 0.38, supporting the internalizing-externalizing distinction. Consistent with results from previous studies, antisocial PD was strongly influenced by the Axis I externalizing factor. From a genetic perspective, it may therefore be placed with the Axis I disorders. Two Axis I disorders, dysthymia and social phobia, were included in the Axis II internalizing cluster, suggesting that from a genetic perspective they may be better placed with the PDs. Borderline PD loaded on both Axis I and Axis II externalizing genetic factors in addition to an environmental liability factor common to Axis I internalizing disorder, consistent with results from factor analytic studies showing associations with both the internalizing and externalizing dimension (Eaton et al., in press). Paranoid and dependent PD had substantial loadings on both the internalizing and externalizing Axis II factors. An important limitation in this study is that it only comprised common Axis I disorders and therefore did not include schizophrenia and other psychotic disorders.

Most of the genetic studies that have investigated changes in genetic influences on PDs and PD traits over time have used measures related to antisocial PD. For example, Lyons and colleagues (1995) demonstrated that the genetic influence on symptoms of *DSM-III-R* antisocial PD was much more prominent in adulthood than in adolescence. In another study, Eley, Lichtenstein, and Moffitt (2003) studied a large number of twin pairs at ages 8 to 9 years and again at 13 to 14 years. They found that genetic influences mediate the continuity in aggressive antisocial behavior from childhood to adolescence, whereas continuity in nonaggressive antisocial behavior was mediated by both shared environment and genetic influences. Results from a study of twins between 10 and 17 years of age demonstrated that a single genetic factor influenced antisocial behavior from age 10 through young adulthood, a shared environmental effect was present beginning in adolescence, there was a transient genetic effect at puberty, and there were genetic influences specific to adult antisocial behavior (Silberg, Rutter, Tracy, Maes, & Eaves, 2007). Finally, another recent twin study of externalizing disorders reported increasing genetic variation and heritability for men but a trend toward decreasing genetic variation and increasing environmental effects for women over the course of adolescence and young adulthood (Hicks et al., 2007b).

MOLECULAR GENETICS OF PERSONALITY

As we have seen, behavior genetic methods, such as twin and family studies, provide an excellent means by which to examine the heritability of personality. These investigations have consistently pointed to sizeable genetic influences on the variance of personality traits, thus largely resolving the question of "nature versus nurture" with an answer of "both." Behavior genetic analyses, however, can only provide broad estimates of genetics effects; they do not identify particular genes that contribute to this variation. To characterize the effects of individual genes—alone or in combination—molecular genetic methods must be used.

The past few decades have seen a major expansion of molecular genetic methods as well as increasingly frequent application of these approaches. In particular, molecular genetic methods have been used to investigate the genetic substrates of a number of medical disorders, sometimes yielding remarkable insights. For instance, molecular genetic studies have identified five gene variants that together account for more than half of the total risk for age-related macular degeneration in siblings (Manolio, 2010). Compelling findings such as this have raised awareness of these methods, and they are currently finding greater traction outside of medicine. Their application has also been bolstered by decreases in cost and continuous technological innovations. As such, psychological scientists have begun using molecular genetic approaches to clarify the role of particular genes in the development of phenotypes of interest and to address precisely which genes contribute to the total heritability of phenotypes identified by behavior genetic investigations.

The molecular genetics of personality is a broad and rapidly changing area of inquiry. In addition to the complexities of molecular genetic approaches, this field is further complicated by the nature of the phenotypes: Personality is a latent construct and thus not directly observable or definitively measurable. Further, the presence of multiple trait models, and associated assessment instruments, means that various constructs are being studied. Even when multiple studies focus on the same personality construct, findings frequently fail to replicate across samples. Thus, this section should be viewed as a snapshot of the current state of a field in flux. In addition, it should be noted that there is a growing literature on the molecular genetics of personality *disorder* as well (e.g., Tadic et al., 2008). Although beyond the scope of this chapter, interested readers are referred to this literature for a deeper understanding of the genes that might underlie variation in personality, whether normal or pathological.

We have organized this section around broad themes of the molecular genetics of personality. We begin with a simplified explication of three popular molecular genetic methods for studying personality: (1) candidate gene analysis, (2) linkage analysis, and (3) genome-wide association studies (GWAS). A full exposition of these methods is beyond the scope of this chapter, but a basic understanding is necessary to digest findings from molecular genetic studies; readers seeking more detail are referred to one of a number of excellent texts on statistical methods for molecular genetics (e.g., Neale, Ferreira, Medland, & Posthuma, 2008; Sham, 1997). Further, it should be noted that the boundaries separating these methods are somewhat artificial, so this tripartite organization serves as a basic explanatory rubric rather than as a representation of three fully distinct methods. After our methodological discussion, we go on to highlight some general findings from molecular genetic studies of personality as well as several novel approaches that may further our understanding of the genetic substrates underlying personality variation.

Three Methods of Molecular Genetic Analysis

We now turn to describing the methods used to examine the molecular genetic basis of personality.

Candidate Genes

The level of focus in molecular genetic studies ranges widely. Some studies focus on the entirety of the human genome, while others focus at the level of the chromosome or at specific points along the chromosome. At a finer level, an individual gene (or a handful of individual genes) may serve as the target of interest. In this latter sort of analysis, researchers typically evaluate the relation between a phenotype and alleles of a specific gene. Alleles are different sequences of DNA that occur at the same physical location on the genome, and can be conceptualized, on a basic level, as representing different forms of a gene, each of which may have a unique impact on the phenotype.

Let us consider eye color in the commonly studied animal model *Drosophila melanogaster*, better known as the fruit fly. These flies typically have red eyes in the wild, although some have unpigmented white eyes. Eye color variation in *Drosophila* is dictated by an allele for a particular gene located on the X chromosome. One allele, the dominant "wild type," produces red eyes; another allele, a mutation, produces white eyes. Thus, in this example, the gene for eye color has two alleles. Similarly, candidate gene

studies in humans typically investigate whether alleles of a particular gene are differentially associated with some phenotype of interest. This picture is complicated by the complexity of psychological phenotypes, which almost certainly result from the interplay of multiple genetic and environmental influences.

How are genes selected to serve as worthwhile "candidates" for study? There is no single method to identify candidate genes; rather, genes are typically selected as candidates for inquiry by several means ranging from the theoretical to the empirical. One common theoretical method is to focus on genes associated with brain-related variables, and particularly with neurotransmitters, as they seem likely to relate to psychological phenotypes such as personality. Some typical candidate genes for psychological and psychiatric genetic studies include those related to dopaminergic functioning (e.g., *DRD3* and *DRD4* genes) and to serotonergic functioning (e.g., *5HTTLPR* and *SLC6A4* genes). Enzymes that modulate the effects of neurotransmitters, such as monoamine oxidase A (MAOA) and catechol-*O*-methyltransferase (COMT), are another logical area of focus, and genes relating to these enzymes (e.g., *MAOA*, *COMT*) frequently serve as candidates for study.

Another means by which candidate genes are identified is through the animal literature. The genomes of many animals have been mapped and, as they frequently contain fewer genes than the human genome, the impact of particular genes on a phenotype is often comparatively easier to determine. Studies of naturally occurring mutations and rare alleles have led researchers toward a better understanding of particular genes in animal models, and analogous genes in humans can then serve as candidates for inquiry. Outside of naturally occurring mutations, experimental methods can be used in candidate gene studies to examine the effects of genes. For instance, geneticists often employ the "knock out" technique of gene modification to test hypotheses about specific gene functions. In this approach, animal models—frequently mice, due to their relatively fast development, low cost, and minimal required upkeep—are bred such that targeted genetic mutations have rendered specific genes inactive. The effects of deactivated genes, which are said to have been "knocked out," are then inferred from phenotypic differences observed between animals with and without the mutations.

A final common means by which candidate genes can be identified is through statistical genetic studies of humans. This is somewhat analogous to an atheoretical multiple regression, in which numerous variables can be used simultaneously to predict *Y* (the dependent variable) in a general sense; the significance of individual predictors, however, is a different, more specific question. The significance of individual predictors can be assessed, and it may be worthwhile to study the association between a single predictor and *Y*. Similarly, through the use of more broadband statistical molecular genetic methods, such as linkage and GWAS methods that examine large areas of the genome, researchers can uncover reasonable candidate genes for further, more direct investigation.

Candidate gene studies are an excellent means by which to tease apart genetic effects and identify the impact of an individual gene on a phenotype. However, for complex phenotypes such as personality traits, which certainly arise from the effects of multiple genes (i.e., personality traits are "polygenic") in addition to environmental influences, the relatively few reasonable candidate genes so far identified are inadequate. As such, methods that can take a more atheoretical approach to polygenic phenotypes and test multiple genes simultaneously hold a great deal of promise. We will now turn our attention to explication of two such methods: linkage analysis and GWAS.

Linkage Analysis

Candidate gene analysis examines the relations between a particular gene (or small set of genes) and a phenotype. To investigate multiple genes simultaneously, however, different approaches must be taken. One such method is linkage analysis. The results of linkage analyses indicate where on a chromosome genes possibly related to the phenotype may be located. Thus, linkage analysis suggests a *region* on a chromosome that seems probabilistically associated with the phenotype. In this way, linkage analysis has a broader focus than candidate gene analysis, but this focus comes at a cost: Resolution is lost to some degree. Within a given region of the chromosome, there can be numerous genes comprising thousands, or millions, of base pairs. Base pairs are pairs of nucleotides that code for the genetic information found in DNA; they are adenine-thymine and guanine-cytosine, often abbreviated as A-T and G-C, respectively. Ultimately, the sequence of these base pairs determines which allele of a gene an individual has. While linkage analysis yields areas of interest on the chromosome, researchers frequently are more concerned with the allelic status of individuals, defined at the level of base pairs (i.e., the genotype). As such, more fine-grained investigation, such as candidate gene analysis, of this chromosomal region can then be conducted to determine precisely which genes are associated with the phenotype of interest.

Linkage analysis is technically more complex than candidate gene analysis in many ways, and understanding it requires a basic knowledge of molecular biology. Briefly, linkage analysis relies on the principles of DNA recombination to identify regions of the chromosome likely associated with a phenotype. When human sex cells—that is eggs and sperm, known as *gametes*—are produced, they contain 23 chromosomes. (The union of the egg and sperm at fertilization yields the full complement of 46 human chromosomes.) However, the 23 chromosomes in a gamete are not simply copies of half of an individual's set of chromosomes. Rather, in the production of gametes, the genetic information from a pair of chromosomes is exchanged (i.e., "recombined"), producing two unique chromosomes. It is this DNA recombination that ensures that two same-sex siblings from the same parents do not have the same genotype. During recombination, *sections* of genetic material from each chromosome "crossover" to the other chromosome, the result of which is a swapping of swaths of physically adjacent genes. Genes that are physically close ("linked") to one another are more likely to remain together on the chromosomal section, as their proximity decreases the probability that the movement of a DNA section to the other chromosome will separate them. For instance, two genes that are immediately adjacent will likely remain on the same section of DNA, because their separation would require a break in the chromosome precisely between them; two genes separated by multiple genes are more likely to be separated during recombination, because segmentation of the chromosome occurring at any of those genes would result in them being on different DNA sections.

Because DNA recombination involves the exchange of segments of DNA comprising multiple genes (rather than, say, the exchange of individual genes), researchers can draw inferences about the physical proximity of genes—and about regions of the chromosome that seem associated with phenotypes. Researchers using linkage analysis study members of families with a given phenotype and sequence marker genes throughout the genome. Then, statistical analysis of physical proximity of genes results in identification of chromosomal areas that appear associated with phenotypic status. In this way, linkage analysis draws on what we know about DNA recombination to permit inferences about which relatively broad chromosomal regions might contain genes that affect the phenotype of interest.

GWAS

GWAS (pronounced "GEE-wahz") is a third means to find how genes are associated with phenotypes and has recently risen to prominence in many areas of medicine, and is beginning to impact psychological science. Unlike candidate gene analysis, which focuses on one or a few genes of interest, and linkage analysis, which identifies chromosomal regions for further study, GWAS examines the entire genome at a very fine-grained level, thus combining breadth with resolution. GWAS evaluates the base pairs of the genetic code and determines if substitutions of one base pair for another in a gene is associated with an expressed phenotype. These substitutions of one nucleotide base for another (e.g., TAGCAT as compared with TAGCGT) are known as single-nucleotide polymorphisms (SNP, pronounced "snip"). GWAS characterizes each individual's genotype for many SNPs—some GWAS analysis microarray chips can sequence millions of SNPs from across the genome—and then compares the SNP frequencies of individuals with different forms of the phenotype (e.g., those high in neuroticism and those low in neuroticism) to determine if any SNP is significantly associated with phenotypic status. In this way, GWAS analysis essentially involves computing thousands, or millions, of *t*-tests to determine if different SNPs are associated with different phenotypes at a level higher than one would expect based on chance alone.

GWAS balances the broad focus inherent in genome-wide analysis with the precise focus of SNP-level analysis. For all its promise, however, GWAS does have limitations. First, it is often expensive to genotype individuals at the level necessary to conduct fine-grained GWAS (although costs of genotyping are decreasing steadily with improvements in technology). Second, it is often computationally intensive and is methodologically complex. Third, it typically requires large samples, both for identification of potential SNPs and for cross-validation purposes. Finally, it is atheoretical, so even replicable results can be ambiguous: It is unclear why a gene known to be expressed in foot development, for example, would reliably relate to personality phenotypes and by what biological mechanisms this association might occur. This being said, GWAS holds great promise as a means to atheoretically identify genes of interest, and in future studies, other methods, such as candidate gene analysis, can be used to explore promising genes with high precision.

Molecular Genetic Studies of Personality

The molecular genetics of personality present a complicated and quickly changing picture. New studies are published with increasing frequency, and it is not uncommon for previous findings to fail subsequent tests of replication by independent research groups. Indeed, the state of

the field is now beginning to shift from individual studies of candidate genes to more widespread use of GWAS, and the compatibility of the results from these different methodologies is not always completely clear. As such, many investigators have found it beneficial to focus at the level of literature review and meta-analysis, and several excellent reviews and meta-analyses of the molecular genetics of personality have been published. Rather than attempting to review the findings of this rapidly evolving and expanding field, we will instead focus our discussion on these reviews and meta-analytic results.

Theories and Measures

Numerous theories of personality have been proposed, each of which posits a unique set of traits and/or mechanisms to describe patterns of behavior and inner experience. Because the primary focus of molecular genetic studies of personality is personality *trait* models, our discussion will focus on personality traits as well. Many of the constructs elaborated in trait models of personality are operationalized in specific personality measures such that the theory and assessment are closely linked; however, notable areas of overlap exist, such as the inclusion of similar traits in some models (see Widiger & Simonsen, 2005). As such, the specific personality traits investigated in molecular genetic studies is often less a function of theoretical interest than of which personality assessment measure—and thus which personality model—was most available, convenient, and en vogue at the time of data collection. (It is noteworthy that some molecular genetic studies of personality resulted from personality data collected many years ago on participants who were more recently genotyped.)

An examination of reviews and meta-analyses (notably Ebstein, 2006; Munafò et al., 2003; Reif & Lesch, 2003; Sen, Burmeister, & Ghosh, 2004) indicates that two of the most commonly used measures in molecular genetic studies of personality are the Tridimensional Personality Questionnaire (TPQ) and the Temperament and Character Inventory (TCI), which operationalize personality theories proposed by Cloninger. The focus on these instruments might be surprising, given personality psychology's general shift toward the traits of the Big Five in the past decades. Cloninger's personality model is built on suggested links between the constructs of these models and biological systems (e.g., Cloninger et al., 1993); therefore, it is likely that such biologically oriented personality theories appeal to more biologically minded investigators (e.g., geneticists, researchers in medical school environments), who themselves went on to conduct many of the molecular genetic studies of personality. Regardless of the rationale, the constructs operationalized in the TPQ and/or TCI—most notably harm avoidance and novelty seeking—have received relatively more molecular genetic attention than many of the other major personality constructs. Of the studies that did not use the TPQ or TCI, most focused on the traits of the Five-Factor Model, operationalized in the NEO-PI-R or NEO-FFI (Costa & McCrae, 1992), the Eysenck Personality Questionnaire (EPQ) and Eysenck Personality Inventory (EPI; H. Eysenck & Eysenck, 1968, 1975), or the Multidimensional Personality Questionnaire (MPQ; Tellegen & Waller, in press).

The multiplicity of personality constructs and measures investigated has proved somewhat problematic for integrating molecular genetic results. For instance, if a study of NEO-PI-R extraversion were to indicate an association with gene X, while another study failed to find an association between EPQ extraversion and gene X, several possible explanations exist. One explanation, of course, is that the original finding simply failed to replicate—a common finding in the molecular genetic literature on personality and in the molecular genetics literature broadly as well. Another possible explanation is that the NEO-PI-R and EPQ operationalizations of trait extraversion are sufficiently dissimilar such that gene X is indeed related to NEO-PI-R extraversion and not significantly related to EPQ extraversion.

The use of different assessment instruments is a real and nontrivial complication in the molecular genetics literature on personality. As highlighted by one meta-analysis (Sen et al., 2004), a major source of variation in studies of the relation between anxiety-related personality traits and a serotonergic gene (*5-HTTLPR*) is the measure used. For instance, NEO-PI-R neuroticism showed a significant association with *5-HTTLPR* ($p = .000016$) while TCI/TPQ harm avoidance did not ($p = .166$). Findings such as these draw into question why some results fail to replicate and the extent to which imperfectly related personality constructs may have different molecular correlates.

Unfortunately, the number of similar, but nonisomorphic, traits considered in molecular genetic studies often precludes the drawing of strong inferences and the quick accumulation of congruent findings. This confound is not fatal, however, and researchers have developed ways to synthesize findings in a logical and defensible way. One method has been to aggregate studies focusing on putatively similar traits and to meta-analyze their results. While necessarily imperfect, reviews and meta-analyses taking this approach have proved informative, and they are often sufficiently large to parse out the effects of measurement instruments on findings (e.g., Sen et al., 2004).

Indeed, some clarity has been found using this approach, especially when the trait groupings seem to represent major sources of variation. For example, Munafò and colleagues (Munafò et al., 2003) parsed different traits into three groups reflecting basic personality constructs: approach behaviors, avoidance behaviors, and fight-or-flight/aggressive behaviors. When statistical control of variance contributed by different measures cannot be accomplished, this sorting approach appears to hold promise for establishing molecular genetic links with broadly defined individual differences in behavioral tendencies.

Candidate Gene Findings

We will now turn our attention to discussing basic trends in the molecular genetics of personality literature. Most molecular genetic studies of personality have relied upon candidate gene analysis, which has had significant implications for the genes investigated (but see Ebstein, 2006, for discussion of other approaches). Notably, candidate gene analysis in psychiatric and psychological genetics has tended to focus on neurotransmitter-related genes, and molecular genetic personality research is no exception.

The first studies of the molecular genetics of personality, appearing in the literature in the mid-1990s, found associations between dopaminergic gene *DRD4* and extraversion/novelty seeking (see Ebstein, 2006, for a historical perspective). Subsequent studies then linked a promoter region of the serotonin transporter gene (i.e., *5-HTTLPR*) to harm avoidance. However, replication attempts of these findings by independent research groups frequently failed, leading to uncertainty about their accuracy. A set of recent meta-analyses, however, has settled these issues somewhat. For instance, two meta-analyses have supported significant associations between *5-HTTLPR* and avoidance-related traits (Munafò et al., 2003; Sen et al., 2004). Associations between *5-HTTLPR* and aggression traits, and between dopaminergic genes (e.g., *DRD3* and *DRD4*) and approach and avoidance traits, were significant ($p < .05$) in a meta-analysis of 46 studies, but most associations were reduced to nonsignificance when the effects of age, ethnicity, and sex were considered simultaneous in a multivariate context (Munafò et al., 2003). Indeed, it is worth noting that across many molecular genetic studies, ethnicity continues to be a complicating factor due to, for example, ethnicity differences in polymorphism occurrence rates, which can obscure subtle genetic effects. Thus, many studies sample only one ethnicity (typically white individuals), while others use statistical techniques, such as principal components analysis, to remove variation associated with ethnicity.

Outside of dopaminergic and serotonergic genetic links, other neurotransmitters and related enzymes have been associated with personality as well. Reviews of the literature by Reif and Lesch (2003) and Ebstein (2006) illustrate how multiple genes and traits have been investigated by researchers with varying results. In general, it has not been uncommon to find associations with genes relating to MAOA and brain-derived neurotrophic factor (BDNF). Thus, while investigation of genes regulating neurotransmitters and related enzymes has been profitable in identifying potentially important associations, the literature will remain something of a hodgepodge until more high-quality reviews and meta-analyses appear.

Gene-Gene and Gene-Environment Interactions

The effects of candidate genes on personality are further obscured by complex gene-gene interactions and epistasis. Epistasis is the phenomenon by which the effect of one gene is modulated by one or more other genes. As reviewed by Ebstein (2006), studies of interactions between candidate genes have proved somewhat fruitful. For instance, interactions have been observed between the serotonin transporter *SLC6A4* and genes relating to dopamine (e.g., *DRD4*) and to GABA (e.g., *GABA[A]*). Even more complex interactions, such as *DRD4* $\times$ *5-HTTLPR* $\times$ *COMT*, have been observed. These interactions highlight the intricate interplay between the effects of multiple genes.

Genes do not operate in a vacuum, and, as mentioned above, their effects can be modulated by those of other genes. Genetic effects may also be affected by environmental factors, a phenomenon known as gene X environment interaction and abbreviated GxE. For example, Caspi and colleagues (2002) investigated the interplay between a polymorphism in the *MAOA* gene, childhood maltreatment, and antisocial behaviors (e.g., disposition toward violence, antisocial personality disorder). The researchers found that *MAOA* genotype status moderated the impact of childhood maltreatment on subsequent antisocial behaviors, such that one genotype appeared protective against the deleterious effects of maltreatment. Numerous attempts at replicating this finding have been attempted with varying results, and a meta-analysis of these studies determined that *MAOA* status moderated the effect of maltreatment on mental health problems across studies (although moderation of the effect on antisocial behavior did not reach significance; Kim-Cohen et al., 2006). Gene X environment studies of this nature thus allow investigators to parse apart the main effects of, and interactions between, genes and environmental factors.

GWAS

In addition to candidate gene studies, some researchers have focused on genome-wide studies of personality. Similar to candidate gene analysis, GWAS attempts have produced varied results. To clarify this topic, researchers have recently completed a meta-analysis of GWAS analyses of the Five-Factor Model domains, bringing together data from around 2.4 million SNPs and more than 20,000 participants (de Moor et al., in press). The results suggested the presence of significant associations between SNPs and both openness to experience and conscientiousness, although these results failed to replicate completely across samples. Thus, the results of GWAS studies have yielded a similar picture to those of candidate gene studies, which appears simultaneously promising and ambiguous.

Novel Approaches

The failure of more "traditional" molecular genetic methods to further our understanding of personality has prompted some researchers to attempt novel approaches. Recent research that has combined molecular genetic investigations with functional neuroimaging—an approach referred to as "imaging genomics"—has produced some remarkable results, especially given the small sample sizes involved (e.g., Hariri & Holmes, 2006; Munafò, Brown, & Hariri, 2008). Other approaches are being explored as well, such as focusing on gene systems rather than individual genes. For example, Derringer and colleagues (2010) combined information from 273 SNPs, residing within eight dopaminergic genes, to test for associations between dopaminergic genes and sensation seeking. The development and use of methods such as these will likely lead to a clearer understanding of the molecular genetics of personality in the coming decades.

SUMMARY AND FUTURE DIRECTIONS

We finish our review of behavioral genetics of personality by considering why the twin method is still relevant for our understanding of the etiology of personality, and how it can inform molecular genetics as the field moves forward.

Why Twin Studies Remain Relevant: Drawing Causal Inferences

Personality genetics is a complex and constantly evolving field. Initial hopes that new technology will yield clear and lasting breakthroughs consistently encounter the reality that identifying the numerous specific polymorphisms associated with personality will be very challenging at best. A system for classifying personality variants based on specific molecular polymorphisms is in the distant future because molecular genetic research on personality is in an early phase of development. The first major international genome-wide effort to identify SNPs associated with the Five-Factor Model of personality yielded little in terms of the number of loci identified and the size of the corresponding effects (de Moor et al., in press). This result is not unique to personality, and is commonly encountered in the study of complex medical and behavioral phenotypes. Some have perhaps seen recent technical breakthroughs in human molecular genetics as a reason that twin studies are passé. This would be an unfortunate conclusion because, as we described throughout this chapter, twin research is valuable for many reasons that go well beyond the estimation of heritability. One recent realization, for example, has been the extent to which the classical twin study design provides a handle on establishing causality in nonexperimental design, that is, in situations where variables cannot be manipulated directly for practical or ethical reasons. McGue, Osler, and Christensen (2010) provide an excellent discussion of the potential for twin research to contribute to establishing specific exposures as causally linked to specific outcomes. Briefly, twin pairs discordant on exposure to an environmental risk factor are matched for both genetic background (if MZ pairs) and rearing environment. As a result, the difference between the twins on exposure may predict a difference in outcome, and such an effect is consistent with a direct causal impact of the exposure. This would be akin to a "counterfactual," that is, it allows one to ask the question "What would have happened to this person in the absence of the exposure?" The cotwin of the exposed twin provides the counterfactual example by virtue of being matched to the exposed twin on a host of relevant factors, allowing for this kind of inference. The reader is encouraged to consult the excellent paper by McGue and colleagues (2010) for a more thorough discussion of these important ideas, illustrating why twins continue to be central to inquiry in behavioral science.

Molecular Inquiry Is Hard But We Should Keep at It and Focus It on Personality

Throughout this chapter we have discussed how challenging it has been to link specific genetic polymorphisms with specific personality dispositions. One potential conclusion

is that the situation is nearly hopeless, such that further effort may be a poor use of time and resources. As with the idea that twin studies have outlived their usefulness, this is another situation where a pessimistic conclusion would be unfortunate. Human molecular genetic inquiry focused on disease is likely to continue, and our suggestion is that this kind of inquiry will be fundamentally assisted by a focus on personality assessment. The reason, as we have described throughout, is that personality is so fundamentally interwoven with so many outcomes of high public health relevance. For example, Lahey (2009) makes a very compelling case that neuroticism is probably the most important single variable in behavioral public health. Essentially, efforts to identify genetic polymorphisms associated with the numerous manifestations of neuroticism (e.g., specific mood or anxiety disorders) are likely less useful than efforts to identify the polymorphisms associated with neuroticism per se—particularly if those efforts are fragmented among different investigators. Our argument is that we need a comprehensive and highly collaborative endeavor designed to understand the genetics and psychobiology of neuroticism (and other trait domains of high public health relevance), as opposed to the fragmented approach of studying putatively distinct disorders that may be better conceived of as aspects of a broad spectrum of neuroticism-linked or "internalizing" disorders (Griffith et al., 2010; Krueger & Markon, 2006). We look forward to seeing whether the field can be galvanized around such a theme. We are optimistic that this kind of broad collaborative focus is possible because the GWAS literature has evolved in exactly this fashion, with large-scale consortia having formed to tackle the limitations of what is possible with specific studies. This kind of collaborative and cooperative approach to the psychobiology of personality is certain to ultimately yield findings that can help us to understand the dispositions underlying problem behavior, and thereby improve public health in transformative ways.

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