

Etiologic Connections Among Substance Dependence, Antisocial Behavior, and Personality: Modeling the Externalizing Spectrum

Robert F. Krueger, Brian M. Hicks, Christopher J. Patrick, Scott R. Carlson,
William G. Iacono, and Matt McGue
University of Minnesota—Twin Cities Campus

A hierarchical biometric model is presented of the origins of comorbidity among substance dependence, antisocial behavior, and a disinhibited personality style. The model posits a spectrum of personality and psychopathology, united by an externalizing factor linked to each phenotype within the spectrum, as well as specific factors that account for distinctions among phenotypes within the spectrum. This model fit self-report and mother-report data from 1,048 male and female 17-year-old twins. The variance of the externalizing factor was mostly genetic, but both genetic and environmental factors accounted for distinctions among phenotypes within the spectrum. These results reconcile evidence for general and specific causal factors within the externalizing spectrum and offer the externalizing factor as a novel target for future research.

Common mental disorders are often correlated with each other, co-occurring at greater than chance rates in both clinical and epidemiological samples (Clark, Watson, & Reynolds, 1995; Lilienfeld, Waldman, & Israel, 1994; Sher & Trull, 1996; Widiger & Sankis, 2000). What is the meaning of this “comorbidity” phenomenon? Krueger and colleagues (Krueger, 1999b, 2002; Krueger, Caspi, Moffitt, & Silva, 1998; Krueger, McGue, & Iacono, 2001) have proposed that this phenomenon may result from common mental disorders acting as reliable indicators of latent factors, or hypothetical core psychopathological processes, that underlie putatively separate disorders.

To date, this hypothesis has been supported by data gathered from unrelated persons. Such data have allowed for multivariate analyses of observed, phenotypic correlations among mental disorders. These analyses have revealed a broad, latent factor linking substance dependence and antisocial behavior disorders in late adolescence and adulthood. Following the lead provided by multivariate analyses of emotional and behavioral problems in children (Achenbach & Edelbrock, 1978, 1984), this factor has been labeled *externalizing* (cf. Kendler, Davis, & Kessler, 1997).

In the analyses presented herein, we extended this line of research by addressing three specific questions in a genetically informative sample. First, what is the etiologic basis for the phenotypic externalizing factor? Second, are there etiologic factors that distinguish among specific externalizing disorders? Third, are disinhibitory personality traits part of the externalizing spectrum?

The Etiologic Basis of the Externalizing Factor

Recent research suggests the hypothesis that genetic factors play an important role in the etiology of the externalizing factor in adolescence and adulthood. First, many large-scale, well-conducted studies now point to genetic factors in the etiology of specific antisocial behavior disorders (Bock & Goode, 1996; Carey & Goldman, 1997; DiLalla & Gottesman, 1989; Gottesman & Goldsmith, 1994; Krueger, Hicks, & McGue, 2001; Lyons et al., 1995; Rutter, 1997; van den Bree, Svikis, & Pickens, 1998) and substance use disorders (Heath et al., 1997; McGue, Pickens, & Svikis, 1992; Pickens et al., 1991; Prescott & Kendler, 1999; Tsuang et al., 1996). Second, in contrast to earlier adoption studies that suggested genetic differentiation of antisocial and substance use disorders (Bohman, Sigvardsson, & Cloninger, 1981; Cadoret, O’Gorman, Troughton, & Heywood, 1985; Cadoret, Troughton, & O’Gorman, 1987; Cloninger, Bohman, & Sigvardsson, 1981; Crowe, 1974; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1973), a number of recent twin studies have begun to point to common genetic factors linking antisocial behavior and substance use disorders. Grove et al. (1990) presented evidence for substantial genetic overlap between antisocial and alcohol problem symptom counts in a small sample of identical, or monozygotic (MZ), twins reared apart. Pickens, Svikis, McGue, and LaBuda (1995) compared cross-twin correlations between alcohol dependence and antisocial personality in small samples of both MZ and fraternal, or dizygotic (DZ), twins. For male pairs, the MZ cross-twin, cross-trait correlation was similar to the within-person correlation between alcohol dependence and antisocial personality but higher than the DZ cross-twin, cross-trait correlation, suggesting that the phenotypic correlation was partially due to genetic factors shared between alcohol dependence and antisocial personality.

The most extensive and thorough study documenting significant genetic links between antisocial behavior and substance use disorders was reported by Slutske et al. (1998). A sample of 2,682 adult Australian twin pairs retrospectively reported symptoms of childhood conduct disorder and alcohol dependence. Both disorders were substantially heritable; in addition, genetic influences

Robert F. Krueger, Brian M. Hicks, Christopher J. Patrick, Scott R. Carlson, William G. Iacono, and Matt McGue, Department of Psychology, University of Minnesota—Twin Cities Campus.

The Minnesota Twin Family Study is supported in part by U.S. Public Health Service Grants AA00175, AA09367, DA05147, and MH65137.

Correspondence concerning this article should be addressed to Robert F. Krueger, Department of Psychology, University of Minnesota, Twin Cities Campus, N414 Elliott Hall, 75 East River Road, Minneapolis, Minnesota 55455-0344. E-mail: krueg038@umn.edu

accounted for 76% and 71% of the phenotypic, observed association between conduct disorder and alcohol dependence in men and women, respectively.

These twin studies have made fundamental contributions to our understanding of the meaning of comorbidity by suggesting that a significant portion of the covariance between substance dependence and antisocial behavior disorders can be traced to common genetic factors. This finding is compatible with the idea of a heritable factor that connects multiple substance use and antisocial behavior disorders (cf. Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Krueger, 1999b; Tarter, 1988). Nevertheless, we are aware of only one study to directly examine genetic and environmental contributions to such a factor (Young, Stallings, Corley, Krauter, & Hewitt, 2000). Young et al. (2000) modeled genetic and environmental contributions to a latent factor linking child-reported symptoms of conduct disorder, attention-deficit hyperactivity disorder, substance experimentation (number of substances used on more than five occasions), and the personality trait of novelty seeking in 334 twin pairs ages 12–18 years. The majority of the variance in the latent factor (84%) was attributed to genetic factors.

The current study therefore endeavored to extend the existing literature by assessing both conduct disorder and adolescent antisocial behavior symptoms, along with alcohol and illicit substance dependence, simultaneously in a sample of 524 male and female 17-year-old twin pairs who were assessed with both maternal and self-report. Thus, our study extends the existing literature by fitting multivariate models to a range of severe externalizing problems that are observed in older adolescents and young adults. In addition, as evidence suggests that parents and children both contribute unique information regarding children's maladjustment (Achenbach, McConaughy, & Howell, 1987), we were able to extend existing work by using information provided by both mothers and children in determining the presence of externalizing symptoms in our participants.

Distinct Etiologic Factors Linked to Distinct Externalizing Syndromes

The externalizing factor accounts for the variance shared among substance dependence and antisocial behavior disorders. Yet when this shared variance is taken into account, significant variance remains uniquely associated with each disorder (Krueger, 1999b; Krueger et al., 1998; Krueger, McGue, & Iacono, 2001). In addition, as noted earlier, adoption studies suggest greater genetic specificity for antisocial behavior and substance use disorders in comparison with twin studies. How might we account for evidence of a broad externalizing factor, unique variance in specific substance use and antisocial disorders, and distinctive findings from adoption and twin studies? These observations might be reconciled if at least a portion of the unique variance in each externalizing syndrome reflects unique etiologic factors, distinct from the etiology of the broad externalizing factor. That is, it may be the case that there are broader factors that impact on the risk for externalizing disorder in general, along with specific factors that differentiate among specific disorders in the externalizing realm.

This hypothesis has considerable appeal because it can accommodate evidence from both adoption and twin studies, that is, evidence for both genetic generality and specificity. Along

these lines, the hypothesis also has the potential to provide an ecumenical resolution to ongoing debates between nosologists who posit that a few broad syndromes can account for most psychopathologic variation ("lumpers"), and those who believe that there are many mental disorders, each with unique etiologies and pathophysiologies ("splitters"). If the unique variance in each externalizing syndrome can be shown to have an etiologic basis not in common with the etiologic basis for the broad externalizing factor, then lumping and splitting positions might be reconciled. Rather than arguing principally for a lumping versus a splitting position, or for genetic generality versus specificity, such data would instead support a hierarchical model of the externalizing disorders.

A hierarchical model organizes individual difference variables from those that are narrow, more specific, and at lower levels of a hierarchy to those that are broader, more general, and at higher levels of a hierarchy (Krueger & Finger, 2001). In this way, comorbidity among mental disorders can be explicitly modeled through the influence of variables at higher hierarchical levels on variables at lower levels. For example, Mineka, Watson, and Clark (1998) proposed a hierarchical model to account for patterns of comorbidity among unipolar mood and anxiety disorders. This model posits a broad, higher order dimension of temperament, namely, negative affect, that influences all disorders within this realm. However, in this model, each separate disorder also has its own unique component of variance. Thus, anxiety and unipolar mood disorders are significantly influenced by negative affect, thereby accounting for their comorbidity. Yet each disorder also contains unique variance, thereby explaining why negative affect can be manifested in diverse ways, that is, as distinguishable, but often comorbid, unipolar mood and anxiety disorders.

Recently, Widiger and Clark (2000) reviewed research on the classification of psychopathology in anticipation of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-V)*. Referring to the potential of the Mineka et al. (1998) model to inform the classification of mood and anxiety (internalizing) disorders, Widiger and Clark (2000) also noted that "on the basis of Krueger et al.'s (1998) results . . . researchers will need a parallel model to account for the externalizing disorders" (p. 954). The work reported herein represents an attempt to develop this type of model. In addition to examining genetic and environmental contributions to the externalizing factor (i.e., to the variance shared among conduct disorder, adolescent antisocial behavior, alcohol dependence, and illicit substance dependence), we were able to examine the genetic and environmental etiology of the residual variance in each of these syndromes. Thus, we were able to evaluate the level of empirical support for a hierarchical model of the externalizing disorders—a model including etiologic factors influencing both the broad, higher order externalizing factor and the residual aspects of specific syndromes within the externalizing realm.

We have discussed how antisocial behavior and substance dependence might define an etiologically coherent spectrum of externalizing disorders. Yet Widiger and Clark (2000) also noted that, like negative affect in the realm of internalizing disorders, the bipolar personality trait of disinhibition–constraint is pervasively linked with disorders in the externalizing spectrum. Thus, in the

research presented here, we also examined how this trait fits into the externalizing spectrum.

Linking Externalizing Disorders and Disinhibitory Personality Traits

The idea that disorders involving substance dependence and antisocial behavior represent syndromes of disinhibition is not new (Gorenstein & Newman, 1980; Zuckerman, 1979). Extensive research documents correlations between externalizing disorders and personality traits such as novelty seeking, impulsivity, and disinhibition (Howard, Kivlahan, & Walker, 1997; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; McGue, Slutske, & Iacono, 1999; McGue, Slutske, Taylor, & Iacono, 1997; Patrick & Zempolich, 1998; Sher & Trull, 1994; Verona & Patrick, 2000; Watson & Clark, 1993). However, most research in this area has examined cross-sectional, phenotypic correlations between mental disorders and personality traits. The problem with this design is that it is ambiguous regarding the causal direction of the personality–psychopathology correlation. That is, a cross-sectional correlation between a disinhibited personality style and psychopathology might be observed because an antisocial, substance-abusing lifestyle leads to impulsivity and disregard for the future consequences of one's actions (cf. Nathan, 1988) or because impulsivity leads to involvement with criminal behavior and substance use (cf. Tarter, 1988). Determining which of these two models is the more plausible requires either longitudinal or genetically informative data.

Longitudinal studies support the latter model. Higher novelty seeking in children is associated with subsequent substance use and abuse (Cloninger, Sigvardsson, & Bohman, 1988; Masse & Tremblay, 1997) as well as subsequent delinquency (Tremblay, Pihl, Vitaro, & Dobkin, 1994). Indeed, impulsivity observed as early as age 3 foretells alcohol dependence and criminal behavior in early adulthood (Caspi, Moffitt, Newman, & Silva, 1996). Moreover, a lack of constraint in late adolescence predicts substance dependence and antisocial behavior in early adulthood, even after controlling for contemporaneous levels of substance dependence and antisocial behavior in late adolescence (Krueger, 1999a).

Genetically informative studies (e.g., twin studies) can also evaluate the possibility that disinhibitory personality traits are causally linked to externalizing disorders because they can discern the extent to which etiologic (genetic and environmental) contributions to personality and psychopathology are shared versus distinctive. For example, twin studies have indicated that a significant portion of the phenotypic relationship between the personality trait of neuroticism and the diagnosis of major depression can be traced to genetic factors shared between these variables (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Roberts & Kendler, 1999). Nevertheless, the twin study approach has rarely been used to examine the etiologic basis for phenotypic connections between personality traits and externalizing disorders. Jang, Vernon, and Livesley (2000) recently reported an investigation documenting substantial genetic correlations between a four-item measure of self-reported alcohol misuse (excessive consumption and alcohol-related problems) and dissocial behavior (a self-reported, continuous personality factor resembling the antisocial personality diagnosis from the *DSM*). However, this study was somewhat limited

by its reliance on a sample of volunteer twin pairs and assessment conducted solely by mailed self-report questionnaire. The study by Young et al. (2000) also supports genetic connections between the personality trait of novelty seeking, involvement in illicit substance use, and childhood symptoms of attention-deficit and conduct disorder, but this study is limited by its sole reliance on self-report data and limitation to milder symptoms characteristic of younger children.

The current research therefore endeavored to extend the existing literature by modeling the personality trait of disinhibition–constraint (Tellegen, 1985; Watson & Clark, 1993) as a potential indicator of the externalizing factor in genetically informative data. Our sample consisted of 17-year-old twins from the Minnesota Twin Family Study (MTFS), a birth record-based epidemiological study of twins born in the state of Minnesota. Twins and their mothers were interviewed in person to assess the twins' childhood antisocial behavior and alcohol and illicit substance dependence, and twins were also interviewed regarding their adolescent antisocial behavior. Twins also completed a self-report index of disinhibition. The fit of a model postulating that these measures were valid indicators of a hypothesized externalizing factor was evaluated. In addition, the genetically informative nature of the data allowed us to extend the existing literature by modeling genetic and environmental influences on both the externalizing factor, and the unique, residual variance in each of the measured indicators of the externalizing factor.

Method

Research Participants

Participants were twin pairs from the MTFS. A comprehensive description of the goals and design of the MTFS has been provided elsewhere (Iacono et al., 1999). Briefly, the MTFS is an ongoing epidemiological–longitudinal study designed to identify the genetic and environmental factors that contribute to substance abuse and related psychopathology. The study used a population-based ascertainment method in which all twins born in Minnesota were identified by public birth records. Initial assessment was conducted during the year the twins turned either 11 or 17 years old. The present investigation involved the 17-year-old cohort, identified from birth records for the years 1972–1978 in the case of male twins and 1975–1979 in the case of female twins. The study was able to locate at least 90% of all twin pairs born during these years in which both members were still living. Families were excluded from participation if they lived further than a day's drive from our Minneapolis laboratories, or if either twin had a physical or intellectual disability that would preclude his or her completing the day-long, in-person assessment. Of the eligible families, 17% declined to participate. A brief self-report survey or telephone interview was obtained from 83% of the nonassessed families. Socioeconomic status levels were slightly, albeit significantly, lower for nonparticipating families, in that parents who participated had 0.25 more years of education, on average, than parents from families that did not participate. However, participating and nonparticipating families did not differ significantly on a brief screening measure of psychopathology, indicating that the MTFS sample is likely representative of twins born in Minnesota during the target years. Consistent with the demographics of Minnesota, 98% of the twins were Caucasian.

Zygosity was determined by agreement of questionnaires completed by (a) parents and (b) MTFS staff regarding the physical similarity of the twins as well as (c) an algorithm that compared twins on ponderal and cephalic indices and fingerprint ridge count. If the three estimates did not agree, a serological analysis was conducted. After intake, the sample size

of the 17-year-old cohort consisted of 626 (223 female MZ, 188 male MZ, 114 female DZ, 101 male DZ) twin pairs. The preponderance of MZ twins reflects both an excess of MZ over same-sex DZ twins in the population from which the sample was drawn (Hur, McGue, & Iacono, 1995), as well as a slightly increased likelihood of MZ relative to DZ agreement to participate.

Measures

Clinical assessment. All twins were interviewed separately and concurrently by different interviewers to assess lifetime mental disorders according to criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (3rd edition, revised; *DSM-III-R*; American Psychiatric Association, 1987). (The *DSM-III-R* was the current diagnostic system at the time of intake.) Mothers were also interviewed about their children's psychopathology. Interviewers had either a bachelor's or master's degree in psychology and underwent extensive training. Maternal reports of child antisocial behavior and substance disorder symptoms were obtained with the use of the parent version of the Diagnostic Interview for Children and Adolescents—Revised (DICA-R; Welner, Reich, Herjanic, Jung, & Amado, 1987). Twins were assessed for child (before age 15) and adolescent (after age 15) antisocial behavior with a structured interview developed by MTFs staff (Holdcraft, Iacono, & McGue, 1998). Twins were assessed for substance abuse and dependence with the Substance Abuse Module (SAM) of the Composite International Diagnostic Interview (Robins, Babor, & Cottler, 1987).

Interview data were then reviewed in a clinical case conference by at least two graduate students with advanced training in descriptive psychopathology and differential diagnosis. All items that scored positive, or about which there were any questions regarding scoring, were reviewed. Symptoms were confirmed on the basis of consensus between the two diagnosticians and were tracked by informant (child or mother). A symptom was considered present if reported by either the twin or the mother, with the exception of adolescent antisocial behavior symptoms, for which only the twin reported.

The current investigation made use of four symptom count variables: adolescent antisocial behavior, conduct disorder, alcohol dependence, and drug dependence. Adolescent antisocial behavior consists of 9 of the 10 Criterion C symptoms of antisocial personality disorder. Symptom 9 ("has never sustained a totally monogamous relationship for more than 1 year") was not assessed due to the young age of the participants. Adolescent antisocial behavior was used instead of antisocial personality disorder because of the *DSM* requirement that an individual must be at least 18 years old to receive the latter diagnosis. In addition, this investigation sought to distinguish between child and adolescent symptoms of antisocial behavior, a distinction confounded by the antisocial personality disorder diagnostic requirement that at least three symptoms of conduct disorder be present before the age of 15 (Elkins, Iacono, Doyle, & McGue, 1997; Iacono et al., 1999). Conduct disorder, alcohol dependence, and drug dependence consist of the Criterion A symptoms of their respective disorders. In the case of conduct disorder, Symptom 9 ("has forced someone into sexual activity with him or her") was not assessed to avoid potential mandated reporting. Drug assessment covered amphetamines, cannabis, cocaine, hallucinogens, inhalants, opioids, phencyclidine, and sedatives. The substance for which the participant had the greatest number of symptoms was used as their drug dependence variable.

Reliability of the assessment process was estimated by an independent review of over 600 cases representative of the entire MTFs sample and yielded the following kappa statistics: .95 for adolescent antisocial behavior, .81 for conduct disorder, and greater than .91 for substance dependence disorders.

Personality. Personality was assessed with a shortened (198-item) version of the Multidimensional Personality Questionnaire (MPQ; Tellegen, in press). The current investigation focused on behavioral disinhibition as indexed by the higher order MPQ factor of Constraint. Persons high

on Constraint tend to inhibit behavioral impulses, to prefer boring but safe activities to exciting but dangerous activities, and to endorse conventional values. The primary MPQ scales Control, Harm Avoidance, and Traditionalism load principally on Constraint (Krueger, 2000; Tellegen, 1985). In this investigation, Constraint was reverse scored so that high scorers tended to exhibit greater behavioral disinhibition. This was done to ease interpretation of results, as all predicted relationships among variables would then be positive.

MPQs were mailed to families prior to their on-site, intake assessment. Participants were asked to bring their completed MPQ with them to their in-person visit. If a completed MPQ was not obtained by the end of the day-long intake assessment, participants were asked to complete it at home and return it by mail. One telephone prompt was made if a completed MPQ was still not received. Complete MPQs were available for 524 (188 female MZ, 156 male MZ, 103 female DZ, 77 male DZ) twin pairs. Female twins were more likely than male twins to complete the MPQ (91% vs. 86%).

To determine whether the final sample was representative, we compared returners and nonreturners on the four *DSM-III-R* symptom count scales in separate analyses for male and female adolescents. Because of the non-normal distributions of the symptom count variables, we used the Mann-Whitney (a nonparametric test) rather than *t* tests to compare groups. Female nonreturners ($n = 64$) did not differ from female returners ($n = 610$) on any of the symptom count scales. Male nonreturners ($n = 82$), however, exhibited more symptoms than male returners ($n = 496$) for adolescent antisocial behavior, conduct disorder, alcohol dependence, and drug dependence (Mann-Whitney $Z_s = -2.98, -2.90, -2.44, \text{ and } -2.57$, respectively, all $p_s < .02$, two-tailed). To provide an estimate of the impact of the higher levels of psychopathology in the male nonreturners, we fit the final best-fitting model without the Constraint variable on the total sample of persons observed on the symptom count variables. These analyses yielded nearly identical parameter estimates to those that included Constraint (i.e., the median absolute standardized parameter estimate discrepancy was .02).

Data Analysis

We used structural equation modeling to determine the genetic and environmental structure of the externalizing disorders and Constraint. The phenotypic variance of any trait can be decomposed into three causal latent factors—additive genetic effects, shared or common environmental effects, and nonshared or unique environmental effects. Twin methodology allows the estimation of these effects by comparing the similarity of MZ and DZ twins. Because MZ twins share all their genetic material, and DZ twins share on average 50% of their segregating genes, additive genetic effects have a correlation of 1.0 for MZ twins and 0.5 for DZ twins. Twin similarity may also be due to shared environmental effects. Because all twin pairs participating in the MTFs were reared together, shared environmental effects have a correlation of 1.0 for both MZ and DZ twins. Nonshared environmental effects are factors whose influences are unique to an individual and therefore are uncorrelated for both MZ and DZ twins. Nonshared environmental effects also include random and unsystematic variance (e.g., measurement error).

Structural equation modeling can be used to model the MZ and DZ correlations in order to estimate genetic and environmental effects and test relationships among multiple variables. We examined the fit of three multivariate biometric models: the Cholesky or triangular decomposition model, the independent pathway model, and the common pathway model (Neale & Cardon, 1992; Waldman & Slutske, 2000). In the Cholesky model, the phenotypic, observed variances and covariances among the five phenotypes (each of the four disorders evaluated plus Constraint) are decomposed into genetic, shared environmental, and nonshared environmental variances and covariances. The Cholesky model is the least parsimonious of the three models because it allows for all possible genetic and environmental variances and covariances to be freely estimated. That is,

the Cholesky model does not impose a particular structure on the genetic and environmental variances and covariances.

The independent pathway model, in contrast, is more parsimonious than the Cholesky model because it imposes a structure on the genetic and environmental variances and covariances. In this model, genetic and environmental effects are of two types: general and specific. This model specifies general latent genetic, shared environmental, and nonshared environmental factors that load on each of the five phenotypes as well as specific genetic and environmental factors that are specific to each of the five phenotypes.

The common pathway model is the most parsimonious of the three models. This model augments the independent pathway model by hypothesizing that the general genetic, shared environmental, and nonshared environmental effects of the independent pathway model are mediated by a latent phenotype. In this model, rather than loading directly on each measured phenotype, the general effects are mediated through a latent phenotype that represents the variance shared among the measured phenotypes. As in the independent pathway model, the common pathway model also allows for additional effects that are specific to each observed phenotype.

Symptom counts were used rather than diagnoses for the following reasons. First, symptom counts retain information that is lost when collapsing into a dichotomous variable (cf. Krueger & Finger, 2001). For example, the Developmental Trends Study reported that over a 4-year period, the number of conduct disorder symptoms fluctuated above and below the number necessary for a definite diagnosis, suggesting that some persons who would be included in a negative diagnostic category are actually more similar to individuals who meet full criteria for the disorder (Lahey et al., 1995). Second, symptom counts provide greater statistical power, especially in a community-based sample such as the MTFs where diagnostic prevalence rates are lower than in a clinically referred sample. Third, there is empirical evidence to support measuring at least some forms of externalizing psychopathology as a quantitative trait (Doyle, 1998). For example, there is a linear relationship between the number of symptoms of conduct disorder and impairment criteria (Robins & Price, 1991). Finally, other investigations have shown that the patterns of genetic and environmental influence are similar for categorical and dimensional models of adolescent antisocial behavior and conduct disorder as well as other forms of psychopathology (Doyle, 1998; Livesley, Jang, Jackson, & Vernon, 1993).

As is typical in a population-based sample, the symptom count variables were positively skewed. In order to better approximate normality, variables were Blom transformed and rank normalized prior to model fitting. A Blom transformation replaces each raw score with its rank value. Ties were resolved by assigning the mean of the ranks being contested. The ranks were then referenced to the normal distribution and expressed in z -score units. A simulation study by van den Oord et al. (2000) has shown that of the available procedures for behavioral genetic analysis of psychiatric symptom count data, this procedure resulted more often in the selection of the true model from a set of alternative models. Though not markedly skewed, reversed Constraint scores were also transformed to maintain consistency across variables. Transformations were conducted by sex but without regard to zygosity. In addition, the data were double entered, a procedure that constrains the variance of Twin A and Twin B to be equal in order to remove any variance associated with this arbitrary designation. However, equating the variances reduces the degrees of freedom because some statistics in the variance-covariance matrix are no longer free to vary.

Model fitting to the variance-covariance matrices for the transformed symptom count scales and Constraint (reversed) was carried out by maximum likelihood estimation with the statistical modeling program (Neale, 1997). One standard index of model fit is the root-mean-square error of approximation (RMSEA), which is used to evaluate the absolute fit of a model. That is, RMSEA is used to determine whether a specific, isolated

model fits the data, but it is not used to select the most optimal model from among a series of competing models. RMSEA values less than .05 indicate a close fit of the model (Browne & Cudeck, 1993). To evaluate the comparative fit of competing models within the present study, we report the Bayesian information criterion ($BIC = \chi^2 - df \ln N$; Raftery, 1995). BIC provides a quantitative index of the extent to which each model maximizes correspondence between the observed and model predicted variances and covariances while minimizing the number of parameters. Better fitting models have more negative values, and the difference in BIC values relates to the posterior odds—the odds ratio formed by taking the probability that the second model is correct, given the data, over the probability that the first model is correct given the data. When comparing models, a difference in BIC of 10 corresponds to the odds being 150:1 that the model with the more negative value is the better fitting model and is considered “very strong” evidence in favor of the model with the more negative BIC value (Raftery, 1995).

Results

Descriptive Statistics

Prevalence rates for the *DSM-III-R* defined disorders were calculated separately for men ($n = 466$) and women ($n = 582$) in order to provide an estimate of the level of psychopathology in the final sample. *DSM-III-R* requires the presence of three or more Criterion A symptoms for a diagnosis of conduct disorder, alcohol dependence, or drug dependence. A clinically significant level of adolescent antisocial behavior was operationalized as the presence of four or more Criterion C symptoms of antisocial personality disorder, as is required by *DSM-III-R* for the latter diagnosis. Conduct disorder symptoms are not included in the adolescent antisocial behavior symptom count. Table 1 provides lifetime prevalence rates at the definite level (all criteria satisfied) and the probable level (all but one symptom present).

Table 1 also contains the means, standard deviations, and range of the symptom count scales for male and female participants. Male participants exhibited significantly more symptoms for adolescent antisocial behavior, conduct disorder, and alcohol dependence (Mann-Whitney $z = -6.10, -10.83, \text{ and } -2.71$, respectively, all $ps < .01$) but not for drug dependence ($z = -.73, ns$). The mean value of reversed Constraint was also significantly higher for male participants. That is, male participants exhibited greater behavioral disinhibition than female participants, $t(1046) = -7.86, p < .001$, two-tailed. The range of the symptom count scales was broad and similar for both genders. These results show that the MTFs sample covers a wide spectrum of behavioral adjustment and maladjustment including a number of persons with clinical levels of psychopathology.

Correlations

Correlations among the Blom-transformed variables were computed to provide initial indications of the magnitude of phenotypic covariation and the relative genetic and environmental contributions to their expression and covariation. Table 2 contains the intraclass correlation matrices for the transformed symptom count scales and reversed Constraint, considered separately for male and female adolescents, with MZ correlations above the diagonal and DZ correlations below the diagonal. Elements in the upper left-hand and lower right-hand portions of the matrices contain the within-twin, cross-trait correlations. These correlations describe

Table 1
Prevalence Rates for Lifetime Diagnoses and Descriptive Statistics for Symptom Count Scales

Disorder	Prevalence rate (%)		Symptom count scale			
	Definite	Probable	<i>M</i>	<i>SD</i>	Range	
					Min	Max
Male adolescents (<i>n</i> = 466)						
Adolescent antisocial behavior	4.7	9.2	0.73	1.16	0	6
Conduct disorder	19.7	33.3	1.37	1.77	0	10
Alcohol dependence	8.8	12.9	0.53	1.26	0	8
Drug dependence	3.2	4.9	0.22	1.03	0	8
Constraint (reversed)			52.6	9.40	23.0	97.0
Female adolescents (<i>n</i> = 582)						
Adolescent antisocial behavior	1.5	3.4	0.39	0.93	0	7
Conduct disorder	3.6	11.9	0.47	0.97	0	9
Alcohol dependence	5.8	8.9	0.41	1.26	0	9
Drug dependence	3.4	5.0	0.22	0.97	0	9
Constraint (reversed)			47.9	9.97	18.8	84.4

Note. A diagnosis at the *definite* level meets full criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (3rd edition, revised; American Psychiatric Association, 1987). A diagnosis at the *probable* level requires all but one symptom to be present. Hence, the probable group contains the definite group. Conduct disorder symptoms are not included in the adolescent antisocial behavior symptom count. Constraint (reversed) is scaled so that the total sample has a mean of 50 and a standard deviation of 10. Min = minimum; Max = maximum.

the phenotypic relationships among the disorders and Constraint, and therefore should be similar across zygosity. The magnitude of these correlations is evidence of the moderate phenotypic covariation among these variables.

The elements in the lower left-hand and upper right-hand portions of the matrices contain the cross-twin, within-trait (along the diagonal) and cross-twin, cross-trait correlations (off-diagonal elements). Cross-twin, within-trait correlations provide information about the status of Twin B if the status of Twin A on that trait is known. For example, the level of adolescent antisocial behavior in Twin B can be predicted if the level of the same disorder in Twin A is known. Cross-twin, cross-trait correlations allow for the prediction of Twin B's status on a trait if the status of Twin A on a *different* trait is known. For example, Twin B's level of adolescent antisocial behavior can be predicted if Twin A's level of alcohol dependence is known.

Cross-twin, within-trait correlations can be used to decompose the variance of a trait into its genetic, shared environmental, and nonshared environmental components, whereas the cross-twin, cross-trait correlations can be used to decompose the covariance between traits into those components. The magnitude of the difference between the MZ and DZ twin correlations describes the relative contribution of the genetic and environmental effects to the expression and covariation of the phenotypes. MZ correlations twice that of DZ correlations suggest that genetic factors are the primary cause of twin similarity and trait covariation. MZ correlations that are nearly equal to DZ correlations suggest that shared environmental factors are the primary cause of twin similarity and trait covariation.

The generally higher MZ, as compared with DZ, correlations in Table 2 suggest that genetic effects contribute substantially to the expression and covariation of the disorders and Constraint. The

similar pattern of correlations for males and female adolescents suggests that although the prevalence of the disorders is higher in men, the covariation and genetic–environmental structure of the disorders and Constraint are unlikely to differ across gender.

Model Fitting

Fitting an explicit statistical model to the data can better summarize the patterns visible in Table 2. We fit sex-variant and sex-invariant versions of Cholesky, independent pathway, and common pathway models to the data. Sex-variant models allowed parameters to differ for men and women, whereas sex-invariant models constrained the parameters to be equal for men and women. RMSEA was less than .05 for each of the models. Comparative fit indices for these models are presented in Table 3.

As is evident in Table 3, when the models are evaluated with a comparative index of fit (BIC), the sex-invariant models fit better than the sex-variant models. Whereas the prevalence for the disorders is higher in males (with the exception of drug dependence), the covariation and genetic–environmental structure of the disorders does not appear to differ by gender. In addition, the largest, negative BIC value was obtained for the sex-invariant common pathway model. That is, compared with the other models listed in Table 3, the sex-invariant common pathway model achieved the best balance of fit and parsimony. Moreover, the BIC value for the sex-invariant common pathway model was more than 10 points lower than the BIC value for its closest competitor (the sex-invariant independent pathway model), providing “very strong” (cf. Raftery, 1995) evidence in favor of the sex-invariant common pathway model. Specifically, the odds are greater than 150:1 that

Table 2
Correlation Matrices for Adolescent Antisocial Behavior, Conduct Disorder, Alcohol Dependence, Drug Dependence, and Constraint (Reversed)

Disorder	Twin A					Twin B				
	AAB	CD	ALD	DD	CON	AAB	CD	ALD	DD	CON
Male adolescents										
Twin A										
AAB	—	.49	.49	.39	.33	.51	.33	.46	.24	.28
CD	.57	—	.27	.25	.32	.33	.56	.23	.22	.22
ALD	.51	.49	—	.36	.22	.46	.23	.53	.36	.14
DD	.32	.39	.50	—	.17	.24	.22	.36	.48	.15
CON	.43	.37	.23	.22	—	.28	.22	.14	.15	.54
Twin B										
AAB	.24	.16	.28	.23	.17	—	.49	.49	.39	.33
CD	.16	.31	.30	.12	.09	.57	—	.27	.25	.32
ALD	.28	.30	.45	.21	.08	.51	.49	—	.36	.22
DD	.23	.12	.21	.28	.15	.32	.39	.50	—	.17
CON	.17	.09	.08	.15	.14	.43	.37	.23	.22	—
Female adolescents										
Twin A										
AAB	—	.36	.56	.54	.40	.35	.30	.43	.43	.27
CD	.50	—	.35	.36	.27	.30	.57	.29	.25	.20
ALD	.63	.41	—	.55	.30	.43	.29	.61	.44	.28
DD	.50	.31	.54	—	.33	.43	.25	.44	.48	.22
CON	.43	.25	.27	.31	—	.27	.20	.28	.22	.54
Twin B										
AAB	.18	.11	.13	.19	.01	—	.36	.56	.54	.40
CD	.11	.40	.12	.02	-.01	.50	—	.35	.36	.27
ALD	.13	.12	.25	.24	.08	.63	.41	—	.55	.30
DD	.19	.02	.24	.41	.07	.50	.31	.54	—	.33
CON	.01	-.01	.08	.07	.24	.43	.25	.27	.31	—

Note. Monozygotic twin correlations are above the diagonal; dizygotic twin correlations are below the diagonal. All variables in the table have been Blom transformed. Because of the double-entry procedure, corresponding elements in the upper left and lower right portions of the matrices (within-twin, cross-trait correlations), as well as corresponding elements above and below the diagonal of the lower left and upper right portions of the matrices (cross-twin, cross-trait correlations), are equal within zygosity. Correlations significant at $p < .01$ (two-tailed) are in boldface. AAB = adolescent antisocial behavior; CD = conduct disorder; ALD = alcohol dependence; DD = drug dependence; CON = Constraint (reversed).

the common pathway model provides a better balance of fit and parsimony than any of the other models listed in Table 3.¹

Figure 1 displays the standardized parameter estimates and 95% confidence intervals (bounded at 0) for the sex-invariant common

Table 3
Comparative Fit Indices for Sex-Variant and Sex-Invariant Confirmatory Biometric Models

Model	χ^2	df	BIC
Cholesky			
Sex variant	92.57	30	-95.23
Sex invariant	158.65	75	-310.85
Independent pathway			
Sex variant	142.06	60	-233.54
Sex invariant	180.79	90	-382.61
Common pathway			
Sex variant	184.14	76	-291.60
Sex invariant	216.61	98	-396.87

Note. For all chi-squares, $N = 524$. BIC = Bayesian information criterion; Sex variant = parameters were free to differ between sexes; Sex invariant = parameters were not allowed to differ between sexes.

pathway model. Path coefficients in the figure must be squared to determine the percentage of variance contributed by a given path. Because the parameter estimates are standardized, the sum of the squares of the paths pointing at a variable sum to 100% (with tolerance for rounding error). Thus, additive genetic factors accounted for 81% ($.90 \times .90$) of the variance of the latent phenotype, Externalizing, with the remaining variance ($.43 \times .43$, or 19%) attributable to nonshared environmental factors. In addition, all the disorders and reversed Constraint have significant loadings on Externalizing. Nevertheless, a model constraining the loadings

¹ Akaike's information criterion ($AIC = \chi^2 - 2 df$; Akaike, 1987), a statistic often used in behavior genetic modeling, ranks the models in Table 3 somewhat differently than does the BIC, preferring the sex-invariant independent pathway model to the sex-invariant common pathway model. In addition, a chi-square difference test comparing these two models indicates that the gain of 8 df in the sex-invariant common pathway model is associated with a significant increase ($p < .05$) in chi-square over the sex-invariant independent pathway model. We did not rely on chi-square difference tests to select the most optimal model from the models

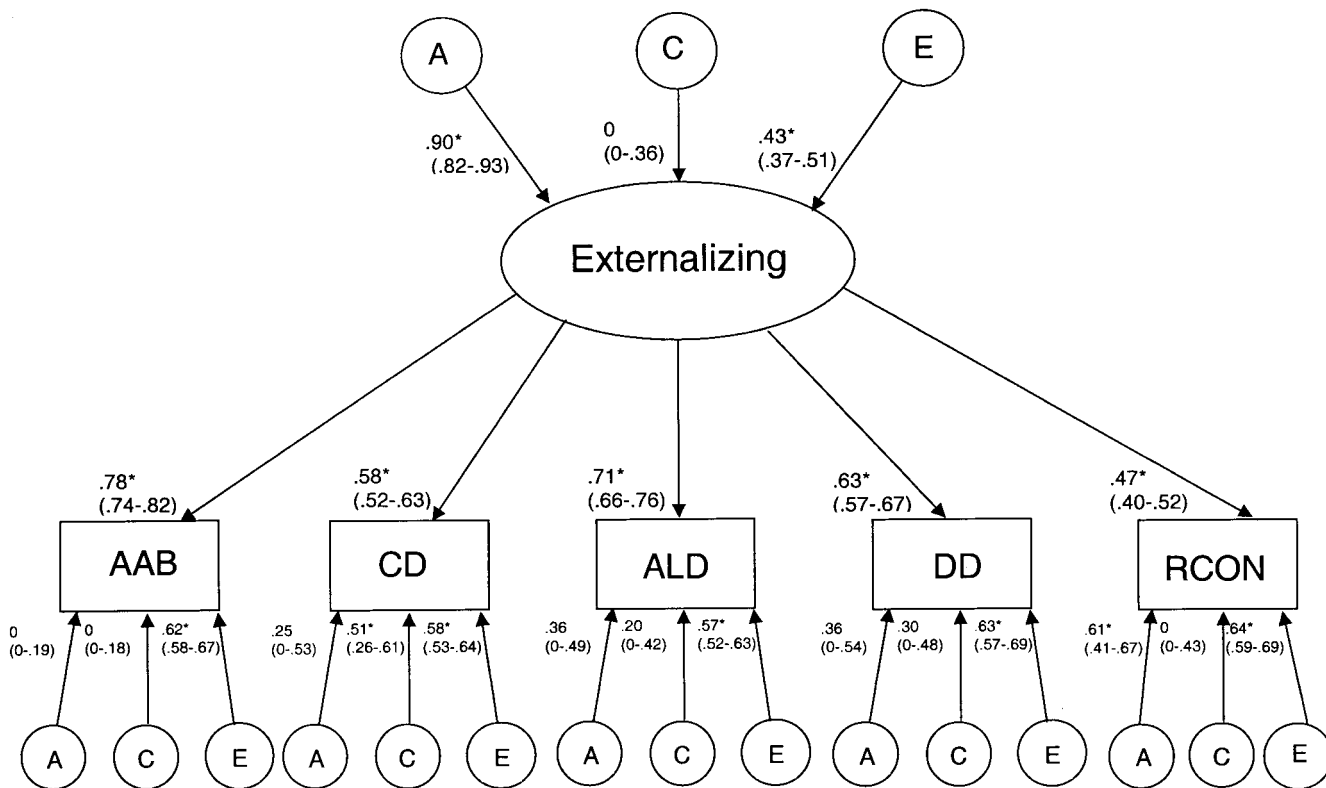


Figure 1. Common pathway model for externalizing phenotypes. Coefficients on the diagram are standardized, and 95% confidence intervals are presented in parentheses beneath each coefficient. Effects whose confidence intervals do not include zero are marked with an asterisk. The percentage of variance accounted for by a given variable in another variable can be determined by squaring the path coefficient on the path connecting the first with the second variable. A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; AAB = adolescent antisocial behavior; CD = conduct disorder; ALD = alcohol dependence; DD = drug dependence; RCON = constraint (reversed).

to be equal across the five variables resulted in a less optimal fit, $\chi^2(102, N = 524) = 279.18$, BIC = -359.34 (difference in BIC compared with the Figure 1 model = 37.53). Thus, the loadings are all significant but differ in magnitude across the five variables.

given in Table 3 because such tests are highly dependent on sample size. In larger samples, chi-square tests tend to prefer complex, "overparameterized" models to more straightforward models because there is more statistical power to detect even minor and substantively trivial differences between model-predicted and observed variances and covariances (Tabachnick & Fidell, 1996). Indeed, this is the reason statistical modelers have turned to indices such as AIC and BIC that attempt to overcome this problem. Both AIC and BIC attempt to identify the "most optimal" model from a competing set of models, where "most optimal" means the model that reproduces the observed variances and covariances with the greatest degree of parsimony (i.e., while invoking as few unknown, estimated parameters as possible). However, BIC differs from AIC in that it is interpreted in Bayesian terms, that is, in terms of the odds of one model being more optimal than another. Hence, BIC provides a very meaningful basis for comparing the degree of support for various models that is not provided by AIC, which is why we have chosen to use BIC to guide model selection in the research presented in this article.

Latent variables at the bottom of Figure 1 are specific or residual genetic, shared environmental, and nonshared environmental effects: factors that contribute to the expression of a particular observed phenotype but not to the expression of any other observed phenotype in the model. As such, specific genetic and environmental effects are etiologic factors that contribute to differences among the observed phenotypes. The common pathway model describes how these specific effects lead to the different phenotypic expressions of the underlying Externalizing factor. As with the loadings, the specific effects differed across the five variables, $\chi^2(110, N = 524) = 343.14$, BIC = -345.46 (difference in BIC compared with the Figure 1 model = 51.41).

Constraint (reversed) was the only variable for which the specific genetic loading (.61) was significant, indicating that there are genetic effects that contribute to the expression of Constraint but not to any of the disorders. Whereas the specific genetic loadings were not significant for any of the disorders, the confidence intervals were relatively large (with the exception of adolescent antisocial behavior). Conduct disorder was the only variable for which the specific shared environmental loading (.51) was significant, suggesting that there were shared environmental effects that were unique to the expression of conduct disorder. Specific non-

shared environmental effects were significant for all of the observed variables. This result suggests that there were nonshared environmental effects specific to the expression of a given variable and to the differentiation of that variable from the other variables included in the model.

Discussion

Substance dependence, antisocial behavior, and disinhibitory personality traits commonly co-occur, yet the reasons for these patterns of co-occurrence have not been fully elucidated. In the research presented here, we have proposed and evaluated a biometric model designed to provide a better understanding of patterns of comorbidity among these “externalizing” syndromes. Our model is hierarchical, involving a general factor linking externalizing syndromes, as well as distinct etiologic factors that differentiate among distinct externalizing syndromes.

This hierarchical model achieved a good fit to our data. Our analyses indicated that co-occurrence among alcohol dependence, drug dependence, conduct disorder, adolescent antisocial behavior, and a disinhibitory personality style assessed in late adolescence can be traced to a highly heritable externalizing factor. Yet this factor did not account for all of the variance in each of its indicators; significant causal variance in each specific syndrome remained after accounting for the general externalizing factor. Thus, our model accommodates evidence for both general and specific etiologic factors in the externalizing realm.

Nevertheless, some important limitations must be borne in mind when considering these results. First, our study is limited by the size of the confidence intervals around some of our parameter estimates (see Figure 1). Although our sample is large by most standards (1,048 individual members of complete twin pairs provided complete data for our study), and although the confidence intervals around most parameter estimates were reasonable, there were wider confidence intervals around our estimates of specific genetic and environmental effects on specific externalizing syndromes. In pursuing large-scale, population-based twin research, there are inevitable trade-offs among sample size, representativeness, and comprehensiveness of assessment. Along these lines, we note that strengths of our sample include its representativeness of the population from which it was drawn, and in-person assessments of mental disorder in which both mothers and their children provided data. Although information from multiple reporters is more difficult to obtain, the use of information from multiple reporters appears to enhance the validity of assessments of mental disorder. For example, combined mother and child reports are better predictors of teacher reports than either mother or child reports taken alone (e.g., Burt, Krueger, McGue, & Iacono, 2001). Nevertheless, future research could complement the work presented here by applying our model to data obtained from a larger sample assessed with the use of alternative data-collection strategies (e.g., mailed surveys completed by twins recruited from a wider range of birth cohorts or from a wider geographical area). In addition, we note that we have converged on our model through a Bayesian approach to model comparison that seeks the model that best reproduces the observed data while invoking the fewest number of unknown, estimated parameters. Although we feel that this is a compelling approach, in that it allowed us to compare models in terms of their odds of providing the most optimal fit to the data,

other approaches to model comparison are also possible. Ultimately, adoption of a model within a specific area of research depends on the model’s heuristic value, that is, the ability of a model to organize research and to lead to novel ideas and findings. We look forward to extensions of the work reported here that evaluate the heuristic value of our model in other contexts (e.g., in terms of specific biological and psychosocial factors that impact on risk of disorders within the externalizing spectrum).

Finally, our study is limited in its ability to delineate specific genetic and environmental causes of variance within the externalizing spectrum. In our study, genetic and environmental effects were inferred; such effects were not linked to specific genetic polymorphisms, nor to specific measured environmental variables. Future studies could endeavor to link the effects documented here to specific genes and environments by including more direct measures of genes and environments in models of the externalizing spectrum.

In spite of these limitations, our findings advance the existing literature. We have provided evidence supporting a specific model of co-occurrence among alcohol dependence, drug dependence, conduct disorder, adolescent antisocial behavior, and a disinhibitory personality style, assessed in late adolescence, with data from both genders and from multiple reporters, in a genetically informative sample. As such, our findings provide answers to the three questions we posed earlier regarding (a) the etiologic basis for the phenotypic externalizing factor, (b) etiologic factors that distinguish among specific externalizing syndromes, and (c) etiologic bases for phenotypic links between disinhibitory personality traits and externalizing disorders.

Heritability of the Externalizing Factor in Late Adolescence

Our results support the hypothesis of significant heritability of the externalizing factor in late adolescence. Previous research documented a phenotypic Externalizing factor linking substance use and antisocial behavior disorders in late adolescence and adulthood (Kendler et al., 1997; Krueger, 1999b; Krueger et al., 1998; Krueger, McGue, & Iacono, 2001). Only one prior study (Young et al., 2000) delineated genetic and environmental contributions to a similar latent factor, identified with a somewhat different set of variables (i.e., symptoms of conduct disorder, attention-deficit hyperactivity disorder, substance experimentation, and novelty seeking). Yet our findings and those reported by Young et al. (2000) are reassuringly similar. Indeed, we estimated the heritability of externalizing at 81%, and Young et al. (2000) estimated the heritability of their latent factor at 84%.

This finding of very high heritability of the latent externalizing factor, now demonstrated independently by two distinct research groups, has key implications for research on externalizing syndromes. The general tendency in this area (and in psychopathology research more generally) has been to study single syndromes in isolation from other syndromes, under the assumption that “pure,” single-disorder groups are more etiologically homogeneous than “impure,” multidisorder groups. The comorbidity phenomenon presents a challenge to this research strategy because pure cases tend to be rare and unrepresentative of individuals who meet criteria for the target disorder (Clark et al., 1995). An alternative strategy is to study “all

comers,” that is, persons who meet criteria for a disorder of interest, regardless of other disorders for which they meet criteria. However, this strategy is also problematic because, in studies of this kind, it is difficult to determine whether the findings are due to the target disorder or to the specific mix of comorbid disorders found in the study (Sher & Trull, 1996).

Our model offers a new perspective on how to design research on externalizing syndromes. Specifically, the high heritability of the externalizing factor makes it an attractive and novel target for research. Rather than focusing on individual disorders such as alcohol dependence or conduct disorder, research could instead focus on the variance shared among these syndromes, that is, the continuous externalizing factor that links the syndromes. From this perspective, comorbid cases are highly informative because they represent the high pole of the externalizing factor. This strategy circumvents problems inherent in comparing disorder-free controls with persons who meet criteria for specific disorders by conceiving of individual syndromes as facets of externalization. A *facet* is a variable that defines one aspect of a broader construct; for example, spatial and verbal talent are facets of intelligence (Jensen, 1980). Thus, alcohol dependence, drug dependence, conduct disorder, adolescent antisocial behavior, and a disinhibitory personality style can be viewed as facets of an externalizing factor, rather than as entirely separate and distinct phenomena. In this way, comorbidity among these disorders is accommodated, rather than ignored or controlled for, as in many contemporary research designs.

In addition to accommodating the comorbidity phenomenon, our model offers the externalizing factor as a highly heritable vulnerability dimension that can be directly measured in samples of unrelated persons. It therefore represents a logical target for future research on the psychobiology of the externalizing disorders. That is, by focusing on the externalizing factor per se, researchers working with samples of unrelated persons can study an individual difference variable closely linked to genetic differences among persons. Nevertheless, our results also indicate that specific facets of the externalizing factor contain unique etiologic variance, a topic to which we now turn.

Distinct Etiologic Bases for Distinct Externalizing Syndromes: Evidence Supporting a Hierarchical Model

Although the broad externalizing factor represents a promising target for continued research, our analyses also support etiologic distinctions among specific externalizing syndromes. The hierarchical nature of our model accommodates evidence for both etiologic generality and specificity by allowing for causal influences on the broad externalizing factor, as well as etiologic influences on each specific syndrome within the externalizing spectrum. As noted earlier, however, confidence intervals around estimates of specific genetic and environmental contributions to specific syndromes were wider than confidence intervals around other estimates. Hence, we focus our discussion on specific point estimates whose confidence intervals did not include zero. These estimates document (a) a unique, shared environmental effect on conduct disorder, (b) unique nonshared environmental effects on each facet

of externalizing, and (c) unique genetic effects on a disinhibitory personality style.

Shared environmental factors contributing uniquely to conduct disorder. Shared environmental effects on each of the five phenotypes we studied, as well as on the higher order externalizing factor, were generally small and not significantly different from zero. The sole exception was conduct disorder, for which the impact of unique, shared environmental factors (which might include influences such as neighborhoods or family dysfunction; Caspi, Taylor, Moffitt, & Plomin, 2000; Patterson, DeGarmo, & Knutson, 2000) was significant, accounting for 26% of the variance (i.e., $.51 \times .51$; see Figure 1). This finding dovetails well with findings from a number of other studies documenting shared environmental effects on conduct disorder and childhood antisocial behavior (Jacobson, Prescott, & Kendler, 2000; Lyons et al., 1995; Miles & Carey, 1997; Thapar & McGuffin, 1996; but see Slutske et al., 1997, for an exception). However, our findings show that the influence of the shared environment on conduct disorder is specific to this syndrome rather than a function of its comorbidity with other syndromes. Young et al. (2000) also found residual effects of the shared environment on conduct disorder, but in their study, these residual effects also influenced substance experimentation. Thus, findings from both groups emphasize the utility of a hierarchical model in understanding both specific and general etiological factors in the externalizing disorders. Overall, the shared environment has little impact within the externalizing spectrum, but it does appear to impact conduct disorder and experimentation with substances. In addition, conduct disorder and substance experimentation refer to behaviors earlier in the life-course (as opposed to adolescent antisocial behavior and substance dependence). Thus, shared environmental factors may be more important earlier in life (cf. Burt et al., 2001).

Unique nonshared environmental effects on each externalizing facet. Most of the unique variance in each externalizing syndrome was traced to nonshared environmental factors (i.e., factors that made our participants different, despite their shared genes and rearing within the same families; Turkheimer & Waldron, 2000). Indeed, each of the unique nonshared variance estimates in Figure 1 was significant (cf. Young et al., 2000).

One possible interpretation of these findings invokes unsystematic or random effects. Random and unsystematic effects mimic nonshared environmental effects because they create differences among relatives, such as twins. Thus, it may be that latent variables (which represent the systematic covariance among multiple indicators) are, in general, more heritable than measured variables (which are more saturated with the unsystematic or random effects specific to specific variables). The nonshared environment may represent such stochastic processes, rather than systematic linear relations between environmental events and phenotypes (Turkheimer & Waldron, 2000).

An alternative viewpoint on the finding of unique nonshared environmental contributions to each measured phenotype might be that nonshared environmental factors account for the differentiation of closely related disorders. That is, genetic factors may work in concert to influence the overall likelihood of developing a disorder in the externalizing spectrum, but what determines the way this liability is expressed are events whose impact is unique to a specific person at specific points in time.

For example, nonshared environmental factors contribute more to the variance of mental disorders measured on single occasions, compared with aggregate estimates of disorder status when disorders are measured on multiple occasions (Foley, Neale, & Kendler, 1998; Kendler, Karkowski, & Prescott, 1999). Thus, future research might extend the approach taken here by studying the externalizing spectrum longitudinally, attempting to link specific, transient environmental events not shared by twins (e.g., unique peer groups; Harris, 1995) to differences in their phenotypic externalizing propensities over time. Such an approach would allow for separation between the effects of temporal instability and random or unsystematic effects and, hence, could extend our understanding of the meaning of the unique nonshared environmental variance in each externalizing phenotype. In addition, this approach takes full advantage of a hierarchical conception of the externalizing spectrum in attempting to identify specific, unique environmental experiences that account for differential manifestations of the broad externalizing factor in different persons, at different times.

Unique genetic effects on disinhibitory personality. The heritability of each externalizing phenotype we studied could be traced to the heritability of the overarching externalizing factor, with one exception: a disinhibitory personality style. Young et al. (2000) also found residual genetic effects on their index of disinhibitory personality, the trait of novelty seeking. One interpretation of this finding is substantive, that is, it may be the case that there are genetic factors that impact uniquely on personality but do not influence overall risk for externalizing psychopathology. Another interpretation of this finding is methodological. Specifically, we measured personality and psychopathology in distinctive ways, using a self-report instrument and an in-person clinical interview, respectively. Although these measurement strategies reflect distinctive traditions in personality and psychopathology research, there is nothing inherent in either construct that demands measurement by interview vs. self-report questionnaire. For example, interviews have been developed to assess normal-range personality traits such as the "big 5" (Trull et al., 1998) and self-report instruments have been developed to assess *DSM*-defined psychopathology (Zimmerman & Mattia, 2001). Thus, future research could disentangle methodological and substantive interpretations of our finding of unique genetic contributions to a disinhibitory personality style by measuring both constructs (personality and psychopathology) using both approaches (interview and self-report questionnaire).

Etiologic Bases for the Link Between a Disinhibitory Personality Style and Externalizing Disorders

Although we found unique genetic variance in our measure of disinhibited personality, this variable also had a significant loading on the broad externalizing factor (cf. Jang et al., 2000; Young et al., 2000). Thus, personality and psychopathology are linked at an etiologic level. Part of the heritability of a disinhibitory personality style can be traced to its role as an indicator of the highly heritable latent externalizing factor, a factor also indicated by psychopathological syndromes.

This finding extends the existing literature by documenting that the phenotypic association between disinhibited personality traits and externalizing disorders can be traced to etiologic factors in common between these phenotypes. Previous research in this area consists primarily of cross-sectional studies of unrelated persons (Sher & Trull, 1994), and such studies are open to multiple interpretations because they cannot establish the etiologic bases of the link between personality and psychopathology (cf. Nathan, 1988; Tarter, 1988). Our study, and the recent studies reported by Jang et al. (2000) and Young et al. (2000), are the first reports, to our knowledge, to document a genetic basis for the disinhibitory personality style–externalizing disorder link. Our study extends the work of Jang et al. (2000) to a population-based sample assessed with in-person interviews, and also extends the work of Young et al. (2000) to a larger, older sample showing more severe forms of externalizing disorder (such as substance dependence) assessed by multiple methods (both parent and child report). In addition, our study places the personality–externalizing disorder connection within the theoretical context of the externalizing spectrum. Disinhibitory personality, substance dependence, and antisocial behavior disorders are linked as indicators of the higher order, highly heritable externalizing factor that spans normal (personality) and abnormal (psychopathological) variation. These findings, now emerging from three independent research groups, thereby challenge the notion of a sharp dividing line between normal and abnormal variation.

In summary, we have presented evidence supporting a hierarchical model of the externalizing spectrum of disorder in late adolescence. Each phenotype we studied was significantly linked to a latent and highly heritable externalizing factor, yet each phenotype also contained unique variance traceable to etiologic factors impacting separately on each phenotype. Thus, our model accommodates evidence for both etiologic specificity and generality within the externalizing spectrum. Nevertheless, much work remains to be done in characterizing the specific genes and environments that account for shared and distinctive etiologic factors impacting on phenotypes in the externalizing spectrum. We hope our model serves a generative role in suggesting strategies for this next phase of research.

References

- Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin*, *85*, 1275–1301.
- Achenbach, T. M., & Edelbrock, C. S. (1984). Psychopathology of childhood. *Annual Review of Psychology*, *35*, 227–256.
- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*, 213–232.
- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- Bock, G. R., & Goode, J. A. (Eds.). (1996). *Genetics of criminal and antisocial behavior*. New York: Wiley.
- Bohman, M., Sigvardsson, S., & Cloninger, C. R. (1981). Maternal inheritance of alcohol abuse: Cross-fostering analysis of adopted women. *Archives of General Psychiatry*, *38*, 965–969.
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model

- fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.
- Burt, S. A., Krueger, R. F., McGue, M., & Iacono, W. G. (2001). Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of shared environment. *Journal of Abnormal Psychology, 110*, 516–525.
- Cadoret, R. J., O’Gorman T. W., Troughton, E. & Heywood, E. (1985). Alcoholism and antisocial personality: Interrelationships, genetic and environmental factors. *Archives of General Psychiatry, 42*, 161–167.
- Cadoret, R. J., Troughton, E., & O’Gorman, T. W. (1987). Genetic and environmental factors in alcohol abuse and antisocial personality. *Journal of Studies on Alcohol, 48*, 1–8.
- Carey, G., & Goldman, D. (1997). The genetics of antisocial behavior. In D. M. Stoff & J. Breiling (Eds.), *Handbook of antisocial behavior* (pp. 243–254). New York, NY: Wiley.
- Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry, 53*, 1033–1039.
- Caspi, A., Taylor, A., Moffitt, T. E., & Plomin, R. (2000). Neighborhood deprivation affects children’s mental health: Environmental risks identified in a genetic design. *Psychological Science, 11*, 338–342.
- Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology, 46*, 121–153.
- Cloninger, C. R., Bohman, M., & Sigvardsson, S. (1981). Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Archives of General Psychiatry, 38*, 861–868.
- Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. *Alcoholism: Clinical and Experimental Research, 12*, 494–505.
- Crowe, R. R. (1974). An adoption study of antisocial personality. *Archives of General Psychiatry, 31*, 785–791.
- DiLalla, L. F., & Gottesman, I. I. (1989). Heterogeneity of causes for delinquency and criminality: Lifespan perspectives. *Development and Psychopathology, 1*, 339–349.
- Doyle, A. E. (1998). *The familial transmission of antisocial behavior: Evidence from the Minnesota Twin Family Study*. Unpublished doctoral dissertation, University of Minnesota, Minneapolis.
- Elkins, I. J., Iacono, W. G., Doyle, A. E., & McGue, M. (1997). Characteristics associated with the persistence of antisocial behavior: Results from recent longitudinal research. *Aggression and Violent Behavior, 2*, 101–124.
- Foley, D. L., Neale, M. C., & Kendler, K. S. (1998). Reliability of a lifetime history of major depression: Implication for heritability and comorbidity. *Psychological Medicine, 28*, 857–870.
- Goodwin, D. W., Schulsinger, F., Hermansen, L., Guze, S. B., & Winokur, G. (1973). Alcohol problems in adoptees raised apart from alcoholic biological parents. *Archives of General Psychiatry, 28*, 238–243.
- Gorenstein, E. E. & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review, 87*, 303–315.
- Gottesman, I. I., & Goldsmith, H. H. (1994). Developmental psychopathology of antisocial behavior: Inserting genes into its ontogenesis and epigenesis. In C. A. Nelson (Ed.), *Minnesota Symposia on Child Psychology: Vol. 27. Threats to optimal development: Integrating biological, psychological, and social risk factors* (pp. 69–104). Hillsdale, NJ: Erlbaum.
- Grove, W. M., Eckert, E. D., Heston, L., Bouchard, T. J., Jr., Segal, N., & Lykken, D. T. (1990). Heritability of substance abuse and antisocial behavior: A study of monozygotic twins reared apart. *Biological Psychiatry, 27*, 1293–1304.
- Harris, J. R. (1995). Where is the child’s environment? A group socialization theory of development. *Psychological Review, 102*, 458–489.
- Heath, A. C., Bucholz, K. K., Madden, P. A. F., Dinwiddie, S. H., Slutske, W. S., Bierut, L. J., et al. (1997). Genetic and environmental contributions to alcohol dependence risk in a national twin sample: Consistency of findings in women and men. *Psychological Medicine, 27*, 1381–1396.
- Holdcraft, L. C., Iacono, W. G., & McGue, M. K. (1998). Antisocial personality disorder and depression in relation to alcoholism: A community-based sample. *Journal of Studies on Alcohol, 59*, 222–226.
- Howard, M. O., Kivlahan, D., & Walker, R. D. (1997). Cloninger’s tridimensional theory of personality and psychopathology: Applications to substance use disorders. *Journal of Studies on Alcohol, 58*, 48–66.
- Hur, Y-M., McGue, M., & Iacono, W. G. (1995). Unequal rate of monozygotic and like-sex dizygotic twin birth: Evidence from the Minnesota Twin Family Study. *Behavior Genetics, 25*, 337–340.
- Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral disinhibition and the development of substance-use disorders: Findings from the Minnesota Twin Family Study. *Development and Psychopathology, 11*, 869–900.
- Jacobson, K. C., Prescott, C. A., & Kendler, K. S. (2000). Genetic and environmental influences on juvenile antisocial behaviour assessed on two occasions. *Psychological Medicine, 30*, 1315–1325.
- Jang, K. L., Vernon, P. A., & Livesley, W. J. (2000). Personality disorder traits, family environment, and alcohol misuse: A multivariate behavioral genetic analysis. *Addiction, 95*, 873–888.
- Jensen, A. R. (1980). *Bias in mental testing*. New York: Free Press.
- Kendler, K. S., Davis, C. G., & Kessler, R. C. (1997). The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: A family history study. *British Journal of Psychiatry, 170*, 541–548.
- Kendler, K. S., Karkowski, L. M., & Prescott, C. (1999). Fears and phobias: Reliability and heritability. *Psychological Medicine, 29*, 539–553.
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A longitudinal twin study of personality and major depression in women. *Archives of General Psychiatry, 50*, 853–862.
- Krueger, R. F. (1999a). Personality traits in late adolescence predict mental disorders in early adulthood: A prospective–epidemiological study. *Journal of Personality, 67*, 39–65.
- Krueger, R. F. (1999b). The structure of common mental disorders. *Archives of General Psychiatry, 56*, 921–926.
- Krueger, R. F. (2000). Phenotypic, genetic, and nonshared environmental parallels in the structure of personality: A view from the Multidimensional Personality Questionnaire. *Journal of Personality and Social Psychology, 79*, 1057–1067.
- Krueger, R. F. (2002). Psychometric perspectives on comorbidity. In J. E. Helzer & J. J. Hudziak (Eds.), *Defining psychopathology in the 21st century: DSM–V and beyond* (pp. 41–54). Washington, DC: American Psychiatric Publishing.
- Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (DSM–III–R): A longitudinal–epidemiological study. *Journal of Abnormal Psychology, 107*, 216–227.
- Krueger, R. F., Caspi, A., Moffitt, T. E., Silva, P. A., & McGee, R. (1996). Personality traits are differentially linked to mental disorders: A multitrait–multidiagnosis study of an adolescent birth cohort. *Journal of Abnormal Psychology, 105*, 299–312.
- Krueger, R. F., & Finger, M. S. (2001). Using item response theory to understand comorbidity among anxiety and unipolar mood disorders. *Psychological Assessment, 13*, 140–151.
- Krueger, R. F., Hicks, B. M., & McGue, M. (2001). Altruism and antisocial behavior: Independent tendencies, unique personality correlates, distinct etiologies. *Psychological Science, 12*, 397–402.
- Krueger, R. F., McGue, M., & Iacono, W. G. (2001). The higher-order structure of common DSM mental disorders: Internalization, external-

- ization, and their connections to personality. *Personality and Individual Differences*, 30, 1245–1259.
- Lahey, B. B., Loeber, R., Hart, E. L., Frick, P. J., Applegate, B., Zhang, Q., et al. (1995). Four-year longitudinal study of conduct disorder in boys: Patterns and predictors of persistence. *Journal of Abnormal Psychology*, 104, 83–93.
- Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical examination of the use of the term and concept of comorbidity in psychopathology research. *Clinical Psychology: Science and Practice*, 1, 71–83.
- Livesley, W. J., Jang, K. L., Jackson, D. N., & Vernon, P. A. (1993). Genetic and environmental contributions to dimensions of personality disorder. *American Journal of Psychiatry*, 150, 1826–1831.
- Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., et al. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry*, 52, 906–915.
- Masse, L. C., & Tremblay, R. E. (1997). Behavior of boys in kindergarten and the onset of substance use during adolescence. *Archives of General Psychiatry*, 54, 62–68.
- McGue, M., Pickens, R. W., & Svikis, D. S. (1992). Sex and age effects on the inheritance of alcohol problems: A twin study. *Journal of Abnormal Psychology*, 101, 3–17.
- McGue, M., Slutske, W., & Iacono, W. G. (1999). Personality and substance use disorders: II. Alcoholism versus drug use disorders. *Journal of Consulting and Clinical Psychology*, 67, 394–404.
- McGue, M., Slutske, W., Taylor, J., & Iacono, W. G. (1997). Personality and substance use disorders: I. Effects of gender and alcoholism subtype. *Alcoholism: Clinical and Experimental Research*, 21, 513–520.
- Miles, D. R., & Carey, G. (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology*, 72, 207–217.
- Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and bipolar mood disorders. *Annual Review of Psychology*, 49, 377–412.
- Nathan, P. E. (1988). The addictive personality is the behavior of the addict. *Journal of Consulting and Clinical Psychology*, 56, 183–188.
- Neale, M. C. (1997). *Mx: Statistical modeling* (4th ed.). Unpublished computer program, Department of Psychiatry, Medical College of Virginia. (Available from M. C. Neale, Box 126, Medical College of Virginia, Richmond, VA 23298)
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic.
- Patrick, C. J., & Zempolich, K. A. (1998). Emotion and aggression in the psychopathic personality. *Aggression and Violent Behavior*, 3, 303–338.
- Patterson, G. R., DeGarmo, D. S., & Knutson, N. (2000). Hyperactive and antisocial behaviors: Comorbid or two points in the same process? *Development and Psychopathology*, 12, 91–106.
- Pickens, R. W., Svikis, D. S., McGue, M., & LaBuda, M. C. (1995). Common genetic mechanisms in alcohol, drug, and mental disorder comorbidity. *Drug and Alcohol Dependence*, 39, 129–138.
- Pickens, R. W., Svikis, D. S., McGue, M., Lykken, D. T., Heston, L. L., & Clayton, P. J. (1991). Heterogeneity in the inheritance of alcoholism: A study of male and female twins. *Archives of General Psychiatry*, 48, 19–28.
- Prescott, C. A., & Kendler, K. S. (1999). Genetic and environmental contributions to alcohol abuse and dependence in a population-based sample of male twins. *American Journal of Psychiatry*, 156, 34–40.
- Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, 25, 111–163.
- Roberts, S. B., & Kendler, K. S. (1999). Neuroticism and self-esteem as indices of the vulnerability to major depression in women. *Psychological Medicine*, 29, 1101–1109.
- Robins, L. M., Babor, T., & Cottler, L. B. (1987). *Composite International Diagnostic Interview: Expanded Substance Abuse Module*. Unpublished manuscript, Washington University, St. Louis.
- Robins, L. N., & Price, R. (1991). Adult disorders predicted by childhood conduct problems: Results from the NIMH Epidemiological Catchment Area Project. *Psychiatry*, 54, 116–132.
- Rutter, M. L. (1997). Nature–nurture integration: The example of antisocial behavior. *American Psychologist*, 52, 390–398.
- Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology*, 103, 92–102.
- Sher, K. J., & Trull, T. J. (1996). Methodological issues in psychopathology research. *Annual Review of Psychology*, 47, 371–400.
- Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P., Bucholz, K. K., Dunne, M. P., et al. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2,682 adult twin pairs. *Journal of Abnormal Psychology*, 106, 266–279.
- Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A. F., Bucholz, K. K., Dunne, M. P., et al. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology*, 107, 363–374.
- Tabachnick, B. G., & Fidell, L. S. (1996). *Using multivariate statistics* (3rd ed.). New York: HarperCollins.
- Tarter, R. E. (1988). Are there inherited behavioral traits that predispose to substance abuse? *Journal of Consulting and Clinical Psychology*, 56, 189–196.
- Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety with an emphasis on self-report. In A. H. Tuma & J. D. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681–706). Hillsdale, NJ: Erlbaum.
- Tellegen, A. (in press). *Manual for the Multidimensional Personality Questionnaire*. Minneapolis: University of Minnesota Press.
- Thapar, A., & McGuffin, P. (1996). A twin study of antisocial and neurotic symptoms in childhood. *Psychological Medicine*, 26, 1111–1118.
- Tremblay, R. E., Pihl, R. O., Vitaro, F., & Dobkin, P. L. (1994). Predicting early onset of male antisocial behavior from preschool behavior. *Archives of General Psychiatry*, 51, 732–739.
- Trull, T. J., Widiger, T. A., Ueda, J. D., Holcomb, J., Doan, B. T., Axelrod, S. R., et al. (1998). A structured interview for the assessment of the five-factor model of personality. *Psychological Assessment*, 10, 229–240.
- Tsuang, M. T., Lyons, M. J., Eisen, S. A., Goldberg, J., True, W., Lin, N., et al. (1996). Genetic influences on DSM–III–R drug abuse and dependence: A study of 3,372 twin pairs. *American Journal of Medical Genetics*, 67, 473–477.
- Turkheimer, E., & Waldron, M. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin*, 126, 78–108.
- van den Bree, M. B. M., Svikis, D. S., & Pickens, R. W. (1998). Genetic influences in antisocial personality and drug use disorders. *Drug and Alcohol Dependence*, 49, 177–187.
- van den Oord, E. J. C. G., Simonoff, E., Eaves, L. J., Pickles, A., Silberg, J., & Maes, H. (2000). An evaluation of different approaches for behavior genetic analyses with psychiatric symptom scores. *Behavior Genetics*, 30, 1–18.
- Verona, E., & Patrick, C. J. (2000). Suicide risk in externalizing syndromes: Temperamental and neurobiological underpinnings. In T. E. Joiner (Ed.), *Suicide science: Expanding the boundaries*. (pp. 137–173). Boston: Kluwer Academic.
- Waldman, I. D., & Slutske, W. S. (2000). Antisocial behavior and alcoholism: A behavioral genetic perspective on comorbidity. *Clinical Psychology Review*, 20, 255–287.
- Watson, D., & Clark, L. A. (1993). Behavioral disinhibition versus constraint: A dispositional perspective. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 506–527). New York: Prentice Hall.
- Welner, Z., Reich, W., Herjanic, B., Jung, K., & Amado, H. (1987).

- Reliability, validity, and parent-child agreement studies of the Diagnostic Interview for Children and Adolescents (DICA). *Journal of the American Academy of Child and Adolescent Psychiatry*, 26, 649-653.
- Widiger, T. A., & Clark, L. A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin*, 126, 946-963.
- Widiger, T. A., & Sankis, L. (2000). Adult psychopathology: Issues and controversies. *Annual Review of Psychology*, 51, 377-404.
- Young, S. E., Stallings, M. C., Corley, R. P., Krauter, K. S., & Hewitt, J. K. (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, 96, 684-695.
- Zimmerman, M., & Mattia, J. I. (2001). A self-report scale to help make psychiatric diagnoses: The Psychiatric Diagnostic Screening Questionnaire. *Archives of General Psychiatry*, 58, 787-794.
- Zuckerman, M. (1979). Sensation seeking and risk taking. In C. E. Izard (Ed.), *Emotions in personality and psychopathology* (pp. 163-197). New York: Plenum Press.

Received March 8, 2001

Revision received December 19, 2001

Accepted January 11, 2002 ■

Members of Underrepresented Groups: Reviewers for Journal Manuscripts Wanted

If you are interested in reviewing manuscripts for APA journals, the APA Publications and Communications Board would like to invite your participation. Manuscript reviewers are vital to the publications process. As a reviewer, you would gain valuable experience in publishing. The P&C Board is particularly interested in encouraging members of underrepresented groups to participate more in this process.

If you are interested in reviewing manuscripts, please write to Demarie Jackson at the address below. Please note the following important points:

- To be selected as a reviewer, you must have published articles in peer-reviewed journals. The experience of publishing provides a reviewer with the basis for preparing a thorough, objective review.
- To be selected, it is critical to be a regular reader of the five to six empirical journals that are most central to the area or journal for which you would like to review. Current knowledge of recently published research provides a reviewer with the knowledge base to evaluate a new submission within the context of existing research.
- To select the appropriate reviewers for each manuscript, the editor needs detailed information. Please include with your letter your vita. In your letter, please identify which APA journal(s) you are interested in, and describe your area of expertise. Be as specific as possible. For example, "social psychology" is not sufficient—you would need to specify "social cognition" or "attitude change" as well.
- Reviewing a manuscript takes time (1-4 hours per manuscript reviewed). If you are selected to review a manuscript, be prepared to invest the necessary time to evaluate the manuscript thoroughly.

Write to Demarie Jackson, Journals Office, American Psychological Association, 750 First Street, NE, Washington, DC 20002-4242.