Genetic and Environmental Influences on the Familial Transmission of Externalizing Disorders in Adoptive and Twin Offspring

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**IMPORTANCE** Twin-family studies have shown that parent-child resemblance on substance use disorders and antisocial behavior can be accounted for by the transmission of a general liability to a spectrum of externalizing disorders. Most studies, however, include only biological parents and offspring, which confound genetic and environmental transmission effects.

**OBJECTIVE** To examine the familial transmission of externalizing disorders among both adoptive (genetically unrelated) and biological relatives to better distinguish genetic and environmental mechanisms of transmission.

**DESIGN** Family study design wherein each family included the mother, father, and 2 offspring, including monozygotic twin, dizygotic twin, non-twin biological, and adoptive offspring. Structural equation modeling was used to estimate familial transmission effects and their genetic and environmental influences.

**SETTING** Participants were recruited from the community and assessed at a university laboratory.

**PARTICIPANTS** A total of 1590 families with biological offspring and 409 families with adoptive offspring. Offspring participants were young adults (mean age, 26.2 years).

**MAIN OUTCOMES AND MEASURES** Symptom counts of conduct disorder, adult antisocial behavior, and alcohol, nicotine, and drug dependence.

**RESULTS** There was a medium effect for the transmission of the general externalizing liability for biological parents ($r = 0.27$-$0.30$) but not for adoptive parents ($r = 0.03$-$0.07$). In contrast, adoptive siblings exhibited significant similarity on the general externalizing liability ($r = 0.21$). Biometric analyses revealed that the general externalizing liability was highly heritable ($a^2 = 0.61$) but also exhibited significant shared environmental influences ($c^2 = 0.20$).

**CONCLUSIONS AND RELEVANCE** Parent-child resemblance for substance use disorders and antisocial behavior is primarily due to the genetic transmission of a general liability to a spectrum of externalizing disorders. Including adoptive siblings revealed a greater role of shared environmental influences on the general externalizing liability than previously detected in twin studies and indicates that sibling rather than parent-child similarity indexes important environmental risk factors for externalizing disorders.
Determining whether a disorder is transmitted within families—and if so, the genetic and environmental mechanisms of transmission—is fundamental to understanding its etiology, as a familial disorder suggests specific theoretical models by which to investigate risk mechanisms. Antisocial behavior and substance use disorders, collectively referred to as *externalizing disorders*, exhibit substantial familial transmission.\(^1\)\(^2\) Externalizing disorders not only run in families but also tend to run together in the same individual; that is, they exhibit high levels of comorbidity or co-occurrence.\(^4\) As such, one theory is that rather than risk for specific disorders, what is transmitted from parents to offspring is a broad liability that increases risk for a spectrum of externalizing disorders.\(^5\)

Consistent with this model, we previously showed that the similarity between parents and their 17-year-old twin offspring for child and adult antisocial behavior, alcohol dependence, and drug dependence could be accounted for by the transmission of a general externalizing factor \(r = 0.30\).\(^6\) There were, however, disorder-specific effects across siblings, that is, effects that contributed to sibling similarity for a specific disorder over and above the general externalizing factor. Results from twin studies indicated that genetic influences accounted for most familial transmission, as the heritability of the externalizing factor in late adolescence was estimated at 0.80 to 0.85 with no shared environmental influences (environmental influences that contribute to similarity among relatives).\(^5\)\(^6\)

We extended this finding to childhood manifestations of the externalizing liability by examining the link between substance use disorders and antisocial behavior in parents and disruptive behavior disorders (attention-deficit/hyperactivity disorder, conduct disorder, oppositional defiant disorder) in their preadolescent offspring.\(^9\) Again, we found that the transmission of a general externalizing factor could account for the similarity between adult externalizing disorders in parents and childhood disruptive behavior disorders in their 11-year-old twin offspring \(r = 0.23\). Disorder-specific effects were again detected for sibling—but not parent-child—similarity. The childhood externalizing factor was also highly familial, exhibiting strong genetic \(a^2 = 0.65\) and significant shared environmental \(c^2 = 0.23\) influences.

Adoption studies can also help to delineate the genetic and environmental influences on the familial transmission of externalizing disorders. The strength of the adoption design is the power to detect shared environmental effects; because family members are genetically unrelated, any similarity must be due to environmental influences. Adoption studies have made fundamental contributions to understanding genetic and environmental risk for externalizing disorders by showing that adoptees tend to more closely resemble their biological parents than their adoptive parents.\(^10\)\(^12\) However, few adoption studies have been large enough to provide sufficient statistical power to adequately parse general vs specific transmission effects. Also, adoption studies have often focused on parent-child similarity (vertical transmission) rather than similarity among siblings (horizontal transmission). The few that have, however, found moderate similarity among adoptive siblings \(r = 0.26\) for adolescent substance use,\(^13\)\(^15\) suggesting that sibling rather than parent influences may be an important environmental risk factor for externalizing disorders.

We sought to further our understanding of the familial transmission of externalizing disorders by combining the strengths of the twin (high power to detect genetic influences) and adoption (high power to detect shared environmental influences) family designs. We were particularly interested in the extent to which the inclusion of adoptive families affected findings for (1) general vs specific transmission of externalizing disorders and (2) estimates of the genetic and environmental influences on general and specific transmission effects. Based on previous findings,\(^10\)\(^14\) we anticipated little similarity between adoptive parents and offspring but small to medium effects for the similarity between adoptive siblings, indicating a greater role of shared environmental influences than previously found in twin studies.\(^5\)\(^7\)\(^16\)\(^19\) We also included nicotine dependence, a disorder that is part of the externalizing spectrum in late adolescence and young adulthood,\(^15\)\(^20\)\(^21\) but one for which we had not examined the general vs specific nature of familial transmission. Also, unlike our previous studies that examined familial transmission when the offspring were children or adolescents,\(^5\)\(^9\) the offspring in this study were adults, being on average 26 years old. As such, the vast majority of offspring had reached or passed the period of greatest risk for antisocial behavior and substance use disorders,\(^23\)\(^24\) allowing us to account for most of the lifetime risk and familial resemblance for externalizing disorders.

**Methods**

Additional details of sample recruitment, demographic characteristics, and measures are provided in the eAppendix in the Supplement. All participants provided informed consent and all study protocols were reviewed by an institutional review board.

**Sample**

Participants were members of the longitudinal Minnesota Twin and Family Study\(^25\)\(^26\) or Sibling Interaction and Behavior Study,\(^25\)\(^26\) both of which used a family design that included the mother, father, and 2 siblings. Offspring from the Minnesota Twin and Family Study were either monozygotic (MZ) or dizygotic (DZ) twins, while the Sibling Interaction and Behavior Study included nontwin biological or adoptive offspring. The mean (SD) age of placement for the adoptive offspring was 4.7 (3.4) months. The total sample included 1999 families: 902 families with MZ twins, 480 families with DZ twins, 208 families with 2 nontwin biological offspring, 124 families with 1 biological and 1 adoptive offspring, and 285 families with 2 adoptive offspring. All twin pairs were same sex; nontwin biological and adoptive siblings were a mix of same sex (61.0%) and opposite sex (39.0%). The mean (SD) age was 44.0 (6.2) years for mothers \(n = 1993\), 45.4 (8.2) years for fathers \(n = 1790\), and 26.2 (3.8) years (range, 16.2-32.4 years) for offspring \(n = 3877\); 53.3% female) at their most recent assessment. More than 90%
of the combined Minnesota Twin and Family Study and Sibling Interaction and Behavior Study sample reported European American ancestry, although a large number of the adoptive offspring reported Korean ancestry (n = 433).

**Assessment**

Externalizing disorders were assessed via structured interviews administered by trained staff. We used DSM-III-R criteria in all analyses as they were assessed at intake and all follow-ups. Symptoms of alcohol, nicotine, and illicit drug abuse and dependence were assessed using the Substance Abuse Module of the World Health Organization's Composite International Diagnostic Interview.** Conduct disorder and adult antisocial behavior (the adult criteria for antisocial personality disorder) were assessed using a structured interview comparable to the Structured Clinical Interview for DSM-III-R, Axis II. Rates of lifetime diagnoses for the full sample were 21.3% with alcohol dependence, 11.6% with drug dependence, 31.0% with nicotine dependence, 10.8% with conduct disorder, and 7.5% with adult antisocial behavior. Externalizing disorders exhibit age and sex effects and even secular trends for drug use that might affect comparisons of familial similarity. Therefore, all symptom counts were regressed on age, age squared, sex, the interactions between the age and sex variables, and whether the participant was a member of the parent or offspring generation. The mean correlation among the symptom count variables was 0.42 (range, 0.30-0.55).

**Statistical Analysis**

Structural equation modeling was used to estimate the general and specific transmission effects for externalizing disorders. The general liability to externalizing disorders was operationalized as a latent externalizing factor, defined by the covariance among symptoms of conduct disorder, adult antisocial behavior, and alcohol, nicotine, and drug dependence. General transmission effects were operationalized as the correlations among the latent externalizing phenotypes between parents and offspring (vertical transmission) and between siblings (horizontal transmission). We estimated disorder-specific transmission effects by allowing the residual variance of the parent symptom counts (ie, the variance of each disorder that was unrelated to the general externalizing liability) to covary with the residual variance of the corresponding offspring symptom counts (eg, the residual variance of the mother’s alcohol dependence was allowed to correlate with the residual variance of the offspring’s alcohol dependence). The same procedure was used to estimate specific transmission effects between siblings.

Models of familial transmission were fit using Mplus version 5.0 statistical software (Muthén and Muthén). We used the MLR estimator that adjusts for the nonindependence of the family-level data and the nonnormal symptom count variables and accommodates missing data. Model fit was evaluated using the mean-adjusted $\chi^2$, the Bayesian information criterion (BIC, $\chi^2 - df$ [In N]), and the root mean square error of approximation. The mean adjusted $\chi^2$ provides an estimate of overall fit for nonnormal data. The BIC balances overall fit with parsimony such that model fit is penalized for the inclusion of unnecessary parameters. Negative BIC values indicate a good fit, with lower (more negative) values indicative of better fit. The root mean square error of approximation provides an estimate of discrepancy in model fit per df; values less than 0.080 and 0.050 indicate adequate fit and very good fit, respectively. Nested models can be compared by conducting a likelihood ratio test calculated as the difference in the $\chi^2$ values and df between the 2 models. Such tests, however, are overpowered for large samples, especially with nonnormal data. Therefore, we used differences in BIC values to compare the relative goodness of fit of competing models given its greater weight to model parsimony. A difference in BIC of 0 to 2 is considered weak evidence of support for the model with the lower BIC value, a difference of 2 to 6 is considered positive evidence, a difference of 6 to 10 is considered strong evidence, and a difference greater than 10 is considered very strong evidence.

We also fit biometric models to obtain estimates of the genetic and environmental influences on the general and specific transmission effects using the offspring data. These models conceptualize the variance of phenotypes as attributable to additive genetic (A), shared environmental (C), and nonshared environmental (E) influences. Additive genetic influences refer to the cumulative effects of genes summed across loci and are inferred if the correlation between MZ twins is greater than the correlation between DZ twin and non-twin biological siblings, which in turn should be greater than the correlation between adoptive siblings. Shared environmental influences refer to environmental influences that contribute to similarity among relatives. As adoptive siblings have no genes in common, the correlation between adoptive siblings provides a direct estimate of shared environmental influences. Shared environmental influences are also inferred if the correlation between DZ twins and non-twin biological siblings is greater than one-half the correlation between MZ twins. Nonshared environmental influences refer to environmental influences that contribute to differences among relatives, including measurement error. Any MZ twin correlations less than 1.0 are evidence of nonshared environmental influences. Biometric models were fit using the computer program Mx with a full-information maximum-likelihood estimator that accommodates missing data. Symptom counts were log($x + 1$) transformed to reduce skew and kurtosis.

**Results**

**General and Specific Transmission of Externalizing Disorders**

First, a model of general transmission only was fit to the 5 family types. To simplify the model, the general transmission effects across siblings were constrained to be the same for the families with (1) DZ twins and non-twin biological offspring and (2) families with 2 adoptive children and families with both an adoptive child and a biological child. This general transmission model provided a good fit to the data, with values for the fit indices listed in Table 1. This model served as the baseline model for all subsequent models that incorporated disorder-specific transmission effects.
Next, we tested for any disorder-specific transmission effects by allowing the residual variance of each parental symptom count to covary with residual variances of the corresponding offspring symptom count variable. Parent-child transmission effects were allowed to vary for biological and adoptive parent-child relationships. To maximize power to detect effects, we tested for 1 disorder at a time (2-\(df\) test). No father-child disorder-specific effects resulted in a lower BIC value, the criterion by which we judged as significant improvement in model fit. For mother-child disorder-specific effects, only the effect for nicotine dependence resulted in a lower BIC value. The effect was small but statistically significant for biological mothers (\(r = 0.11\); 95% CI, 0.06 to 0.17) but not adoptive mothers (\(r = 0.03\); 95% CI, −0.06 to 0.13).

We also tested for disorder-specific transmission effects across siblings, allowing these effects to vary for MZ twins, DZ twins, and non-twin biological siblings, and adoptive siblings. Including specific transmission effects across siblings resulted in a lower BIC value for each disorder. The specific effect for adult antisocial behavior, however, was no longer significant when the other disorder-specific effects were included in the model; therefore, this effect was dropped. Specific transmission effects for siblings were medium to large for MZ twins (0.32 to 0.58), small to medium for DZ twins and non-twin biological siblings (0.08 to 0.36), and mostly nonsignificant for adoptive siblings (−0.05 to 0.13). The final best-fitting model is depicted in Figure 1.

The correlation between the mother and father latent externalizing factor was medium to large (\(r = 0.45\); 95% CI, 0.35 to 0.55), indicative of assortative mating. For the general transmission effects, the parent-child correlations for the latent externalizing factors were medium for biological mothers (\(r = 0.27\); 95% CI, 0.20 to 0.34) and fathers (\(r = 0.30\); 95% CI, 0.23 to 0.38) but were not significantly different from 0 for adoptive mothers (\(r = 0.07\); 95% CI, −0.01 to 0.15) and fathers (\(r = 0.03\); 95% CI, −0.07 to 0.12). Families that included both biological and adoptive offspring provided a particularly elegant demonstration of this pattern of familial transmission. The general transmission effects were significant for biological children (mother: \(r = 0.26\); 95% CI, 0.14 to 0.37; father: \(r = 0.35\); 95% CI, 0.25 to 0.46) but not for adoptive children (mother: \(r = 0.06\); 95% CI, −0.02 to 0.14; father: \(r = 0.02\); 95% CI, −0.07 to 0.11). These findings suggest that parent-child resemblance on externalizing disorders is almost entirely attributable to genetic transmission. We also tested whether the general transmission effects differed by mother and father. Equating the mother and father general transmission effects did not result in a higher BIC

<table>
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<th>Model</th>
<th>(\chi^2)</th>
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<th>RMSEA</th>
<th>BIC</th>
<th>(\Delta\chi^2)</th>
<th>(df)</th>
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<td>General transmission from parent to offspring, general transmission across siblings, specific effect for maternal nicotine dependence, cross-sibling specific effects for conduct disorder and alcohol, nicotine, and drug dependence</td>
<td>1648.22</td>
<td>960</td>
<td>0.042</td>
<td>−5648.16</td>
<td>647.64</td>
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<td>Equate maternal and paternal general transmission</td>
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<td>962</td>
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<td>−5649.14</td>
<td>633.41</td>
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Abbreviations: BIC, Bayesian information criterion; RMSEA, root mean square error of approximation.

* Lower values for fit indices are indicative of better fit. Lower (more negative) BIC values were used to select the best-fitting model.

\(b\) Significant at \(P < .001\).
value (Table 1), indicating that the correlations were not significantly different from each other.

The correlation between the 2 siblings on the latent externalizing factor was large for MZ twins ($r = 0.79$; 95% CI, 0.73 to 0.84), while the correlation was medium to large for DZ twins and nontwin biological siblings ($r = 0.45$; 95% CI, 0.36 to 0.55) and smaller but statistically significant for adoptive siblings ($r = 0.21$; 95% CI, 0.09 to 0.33). The adoptive sibling correlation is a direct estimate of shared environmental influences, indicating that sibling similarity was due to a combination of genetic and shared environmental influences.

### Genetic and Environmental Influences on General Externalizing Liability and Disorder-Specific Liabilities

Next, we fit standard biometric models to derive more precise estimates of the genetic and environmental influences on each externalizing disorder and the general externalizing liability. Table 2 provides the sibling correlations and univariate ACE parameter estimates for each externalizing disorder. Each externalizing disorder exhibited moderate heritability (0.35-0.49) and nonshared environmental influences (0.38-0.48). Each disorder except alcohol dependence also exhibited significant shared environmental influences (0.09-0.26).

Next, we fit a biometric factor model to estimate the genetic and environmental influences on the general externalizing liability as well as disorder-specific genetic and environmental liabilities (Figure 2). The general externalizing factor was highly heritable ($a^2 = 0.61$; 95% CI, 0.50-0.71) but also exhibited significant shared environmental influences ($c^2 = 0.20$; 95% CI, 0.16-0.23), with the remaining variance attributable to nonshared environmental influences ($e^2 = 0.19$; 95% CI, 0.16-0.23).

In terms of disorder-specific liabilities, each disorder exhibited specific genetic effects (drug dependence: $a^2 = 0.27$; 95% CI, 0.17-0.32; conduct disorder: $a^2 = 0.24$; 95% CI, 0.15-0.33; nicotine dependence: $a^2 = 0.21$; 95% CI, 0.13-0.28; and alcohol dependence: $a^2 = 0.16$; 95% CI, 0.09-0.20), although the effect for adult antisocial behavior was only marginally significant ($a^2 = 0.07$; 95% CI, 0.003-0.14). Only conduct disorder exhibited a specific shared environmental effect ($c^2 = 0.19$; 95% CI, 0.11-0.26). Each disorder also exhibited specific nonshared environmental influences (adult antisocial behavior: $e^2 = 0.30$; 95% CI, 0.27-0.34; conduct disorder: $e^2 = 0.39$; 95% CI, 0.35-0.43; alcohol dependence: $e^2 = 0.38$; 95% CI, 0.35-0.42; drug dependence: $e^2 = 0.40$; 95% CI, 0.36-0.44; and nicotine dependence: $e^2 = 0.32$; 95% CI, 0.29-0.36).

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**Figure 1. Family Transmission of Externalizing Disorders**
Discussion

Previously, we showed that most parent-child similarity on substance use disorders and antisocial behavior can be attributed to the transmission of a general liability to a spectrum of externalizing disorders. Herein, we extended this finding by leveraging the strengths of the twin and adoptive family designs in a single analysis, showing a medium effect for this general transmission among biological parents and offspring but a near 0 effect for adoptive parents and offspring. Especially persuasive was that this pattern was found in families that included both biological and adoptive offspring. That is, even among children who shared the same rearing environment, bio-

Table 2. Sibling Correlations and Univariate Parameter Estimates for Additive Genetic, Shared Environmental, and Nonshared Environmental Influences on Externalizing Disorders

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sibling Correlation</th>
<th>Univariate Parameter Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MZ Twin Pairs (n = 902)</td>
<td>DZ Twin Pairs (n = 480)</td>
</tr>
<tr>
<td>Adult antisocial behavior</td>
<td>Value 0.58</td>
<td>0.36</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.54 to 0.62</td>
<td>0.28 to 0.44</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>Value 0.62</td>
<td>0.49</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.57 to 0.65</td>
<td>0.32 to 0.55</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>Value 0.51</td>
<td>0.28</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.45 to 0.55</td>
<td>0.20 to 0.36</td>
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<tr>
<td>Drug dependence</td>
<td>Value 0.55</td>
<td>0.32</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.50 to 0.59</td>
<td>0.24 to 0.40</td>
</tr>
<tr>
<td>Nicotine dependence</td>
<td>Value 0.61</td>
<td>0.35</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.57 to 0.65</td>
<td>0.27 to 0.43</td>
</tr>
</tbody>
</table>

Abbreviations: A, additive genetic influence; C, shared environmental influence; DZ, dizygotic; E, nonshared environmental influence; MZ, monozygotic.

Figure 2. Biometric Model of Genetic and Environmental Influences on Externalizing

All path coefficients are standardized and 95% CIs are given. All coefficients that do not include 0 are significant. 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 variable to the second variable. The sum of the squared loadings (effects from the general externalizing factor as well as the specific additive genetic [A], shared environmental [C], and nonshared environmental [E] influence loadings) equals 1.0. The total effect for A, C, and E can be calculated by summing the general effect (squared loading for a given disorder multiplied by the squared A, C, and E loadings on the externalizing factor) and the specific effect (squared A, C, or E specific effect on a given disorder). Using conduct disorder as an example, $a^2 = \text{general}(0.61^2 \times 0.50^2) + \text{specific}(0.49^2) = 0.33; c^2 = \text{general}(0.45^2 \times 0.50^2) + \text{specific}(0.43^2) = 0.24; e^2 = \text{general}(0.44^2 \times 0.50^2) + \text{specific}(0.62^2) = 0.43.$
logical offspring exhibited greater similarity than adoptive offspring to the same rearing parents. This indicates that, for the most part, the mechanism of parent to child transmission is not only general but also genetic in nature. Rather than disorder-specific risk, what parents pass on to their (biological) offspring is a nonspecific, genetic liability to multiple externalizing disorders.

In contrast to adoptive parents and offspring, we detected significant similarity between adoptive siblings on the general externalizing liability, which was solely attributable to environmental influences. This was reflected in the results of the biometric factor model, as we detected significant shared environmental influences on the general externalizing factor, more so than in previous twin studies.6,7 Previous Minnesota Twin and Family Study studies also identified shared environmental influences on a general externalizing factor in childhood8 and middle adolescence,9 but this is the first to do so in adulthood. Although the mechanism of these shared environmental effects is not yet known, it seems that sibling rather than parent-child similarity indexes shared environmental risk for multiple externalizing disorders.

We did detect a specific transmission effect for maternal nicotine dependence. The effect was small, however, and may be false-positive. The slightly stronger effect for biological mothers relative to adoptive mothers suggests either a specific genetic effect or a maternal smoking effect on fetal development. Regarding the former, a strong association was detected between the nicotinic receptor gene CHRNA3 and cigarettes smoked per day in a large genome-wide association study, but only among current smokers.10,11 This suggests that the CHRNA3 gene confers a specific genetic risk for nicotine addiction that is likely unrelated to externalizing, which might be evident in the specific effect we detected.

Disorder-specific transmission effects appear to be robust for sibling to sibling transmission. To some extent, this could be attributable to differences in assessment, that is, siblings are more similar in age, are assessed on multiple occasions, and are more actively engaging in externalizing behaviors relative to parents who were assessed for lifetime symptoms on a single occasion in middle adulthood. Conduct disorder was the most distinctive of the externalizing phenotypes exhibiting specific genetic and shared environmental influences. This distinctiveness is likely due to being a childhood disorder with multiple contributing etiological factors. That is, conduct disorder is a risk factor for almost all forms of adult psychopathology,12 suggesting that only a portion of conduct disorder cases are attributable to the general externalizing liability. The disorder-specific effects for the substance use disorders were primarily attributable to genetic factors, suggesting genes that confer increased risk for addiction to specific substances. Very little of the variance in adult antisocial behavior was attributable to specific genetic or shared environmental effects, indicating that it is the disorder most strongly determined by the general externalizing liability.

Some limitations should be noted. First, the sample had limited racial and ethnic diversity, which constrains generalizability to other populations. A second limitation regards restriction of range among adoptive families. Specifically, relative to biological families, mean levels of externalizing symptoms were lower among the adoptive parents but not among the adoptive offspring.22 However, a thorough analysis of restriction of range in the Sibling Interaction and Behavior Study sample22 showed that such effects were relatively small and would have little impact on our results. Third, our measure of drug dependence assessed illicit drug problems in general, when there are likely different genetic influences for different drug classes. Fourth, there was a notable age range among the offspring participants, leaving open the possibility that the results could differ slightly for different ages. Additionally, because data collection of the longitudinal data spanned 2 decades, there may be small cohort effects within the offspring sample. Finally, we used standard biometric models rather than more complex extended twin-family models35 (although they are unlikely to yield substantively different results) or models that incorporate the presence of gene × environment interactions that seem to be present for externalizing disorders.36

To conclude, we again demonstrated the primacy of general rather than disorder-specific transmission from parents to offspring for externalizing disorders and leveraged the adoption data to strengthen the case for genetic transmission. We also detected a greater role for shared environmental influences on the general externalizing liability in adulthood than previous twin studies and were able to link these environmental influences to sibling rather than parent similarity. An important future direction will be to identify more specific mechanisms of these shared environmental influences and their interplay with genetic risk in the development of externalizing disorders.