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Confirming the Etiology of Adolescent Acting-out Behaviors: An Examination of Observer-ratings in a Sample of Adoptive and Biological Siblings

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Abstract

Background—A recent meta-analysis revealed moderate shared environmental influences (C) on most forms of child and adolescent psychopathology (Burt, 2009), including antisocial behavior. Critically, however, the research analyzed in this meta-analysis relied largely on specific informant-reports (and particularly parent and child reports), each of which are subject to various sources of rater bias. Observer-ratings of children's behaviors avoid many of these biases, and are thus well-suited to verify the presence of C. Given this, we sought to buttress the evidence supporting C in two key ways. First, we sought to confirm that C contributes to observer-ratings in a sample of adoptive siblings, as similarity between adoptive siblings constitutes a “direct” estimate of C. Second, we sought to confirm that these shared environmental influences persist across informants (i.e., the effects are not specific to the rater or the context in question).

Methods—The current study examined the etiology of observer-ratings of acting-out behaviors, as well as sources of etiological overlap across observer-ratings, adolescent self-report and maternal-report in sample of over 600 biological and adoptive sibling pairs from the Sibling Interaction and Behavior Study (SIBS).

Results—Results revealed moderate and significant shared environmental influences on observer-ratings (31%), as well as on the other informant-reports (20-23%). Moreover, a portion of these effects overlapped across measures (C correlations ranged from .32-.34).

Conclusions—Such findings argue against passive rGE and rater bias as primary explanations for earlier findings of C on antisocial behavior, and in this way, offer a critical extension of prior work indicating that the role of shared environmental influences on child and adolescent antisocial behavior was dismissed too soon.

Keywords

shared environment; delinquency; antisocial behavior; adoption design; observer-ratings

A recent meta-analysis revealed the presence of moderate shared environmental influences (C; or those familial and contextual experiences that increase sibling similarity regardless of

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the proportion of genes shared) on most forms of child and adolescent psychopathology (Burt, 2009), including antisocial behavior. This finding was somewhat surprising, as prior theory held that meaningful environmental influences were primarily non-shared or child-specific in nature (Plomin & Daniels, 1987). Burt (2009) also examined a number of potential moderators of these effects, including informant. C effects were identified for multiple individual informant-reports, thereby bolstering conclusions regarding the importance of these effects.

Even so, behavioral genetic research on psychopathology to date (and thus, the research included in the meta-analysis) has relied almost exclusively on individual informant-reports (and particularly parent and child reports), each of which are subject to various sources of rater bias (as reviewed in Burt, 2009; De Los Reyes & Kazdin, 2005). Depending on the child's age, his or her self-report may be relatively unreliable, thereby serving to reduce twin correlations and artifactually increasing estimates of the non-shared environment (as seen in Burt, 2009). Alternately, parents may have limited knowledge regarding normative child behavior, and thus may interpret specific behaviors as more or less pathological accordingly. Parents may also over- or under-estimate the similarity of their twins (referred to as "sibling contrast"), thereby artifactually inflating heritability estimates. Teachers, by contrast, have extensive knowledge of normative child behavior, although this knowledge is largely context-specific. Moreover, all informant-reports may be influenced by recency effects or by the rater's own personality/psychological background. For example, a parent may report that a child acts out more or less than he/she objectively does based on the parent's own psychological state, or based on a recent incident. In short, results obtained through particular individual informant-reports are somewhat less conclusive than one would like.

Some researchers have attempted to circumvent reliance on individual informant-reports with the use of informant composites, in which multiple informant-reports are combined prior to analysis. This procedure has been found to improve the validity of the assessment (Kraemer et al., 2003). However, because it is difficult to tease apart error and true score variance even when combining informants, this solution is unlikely to fully ameliorate issues of potential rater bias. Observer-ratings of children's behaviors offer another compelling strategy for avoiding most forms of rater bias. As with teacher-reports, children are rated in comparison to other children in the same developmental stage, thereby ensuring that children are evaluated in reference to normative child behaviors. Coders also receive extensive manual-based training to reach and then maintain reliability with other coders. Scores obtained this way thus allow researchers to obtain data that are largely (though, of course, not entirely) free of personological interference. Given these two clear advantages, we would thus argue that the use of observer reports can meaningfully supplement our understanding of the etiology of adolescent antisocial behavior.

To date, there are several twin studies examining etiological influences on observer-ratings of acting-out behaviors (Arsenault et al., 2003; Deater-Deckard, 2000; Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998; Marceau et al., submitted; O'Connor, Hetherington, Reiss, & Plomin, 1995; Plomin, Foch, & Rowe, 1981). Early work by Plomin et al. (1981), Leve et al. (1998), and Deater-Deckard (2000) estimated shared environmental influences on observer-ratings to be moderate-to-large in magnitude (25-42%). These findings were largely replicated in more recent work by Marceau et al. (submitted), who examined the etiology of observer-ratings in three independent samples. Estimates of C were prominent across all three samples (i.e., 44%, 77%, and 38%). Indeed, only two existing studies (Arsenault et al., 2003; O'Connor et al., 1995), to our knowledge, have not found evidence of significant shared environmental influences, although in both cases, the C effect was estimated to be "non-zero" (10-12%).

As there are no consistent differences in twin age, twin sex, or the severity of the coded behaviors, it remains unclear what may account for these differences across studies. Even so, it is noteworthy that *C* was uniformly estimated to be non-zero across all studies, and was typically moderate-to-large in magnitude. Given this, we would argue that the above studies collectively support the presence of *C* on observer-ratings, and in this way, argue against the notion that shared environmental influences on antisocial behavior are primarily a function of rater bias. Even so, additional research is needed to confirm this conclusion. First, available studies have exclusively examined twin/twin-sibling samples, which are not particularly well suited for examining the shared environment. In particular, *C* can be confounded by passive gene-environment correlations (i.e., passive rGE) when the origins are in fact a function of common parent-child genes. Simply put, the environment provided to one's biological children (including parental behavior towards the child) reflects the genetically influenced preferences and tendencies of the parent. And because parents share genes with their biological children, the child's genes are then correlated with his/her parent's behavior. Fortunately, the role of passive rGE in *C* can be circumvented by examining adoptive siblings. Because adoptive siblings do not share genes with their adoptive family members, passive rGE are entirely eliminated, providing a "direct" estimate of *C*. It would thus be critically important to extend the above findings to a sample of adoptive siblings, thereby firmly ruling out passive rGE as an explanation for prior results.

Second, informant-specific shared environmental influences have often been interpreted as suggestive of rater bias (Bartels et al., 2004) rather than other, more meaningful possibilities (e.g., situational specificity). This attribution may reflect the fact that shared environmental effects have historically been thought to have a minimal influence on outcome (Plomin & Daniels, 1987). Regardless of the reason, because of this history, it is important to confirm that shared environmental effects are not confined to particular informants. Shared environmental influences that overlap with those on observer-ratings would constitute particularly strong evidence for persistence across informants, given both the very different assessment strategies (e.g., lifetime assessment versus a 10-minute slice of observation) as well as the fact that observer-ratings should be largely free from rater bias. Should shared environmental influences on observer-ratings overlap with those on other informant-reports, it would provide convincing evidence against rater bias as a primary explanation for observed shared environmental influences.

The current study sought to fill these gaps in the literature, so as to more definitely support or refute claims of meaningful *C* on adolescent antisocial behavior. To do so, we examined the sources of etiological influence on observer-ratings of acting-out behaviors, as well as the origins of overlap between these ratings and parent- and child-reports, in a sample of adoptive and biological siblings. Should there be evidence of significant *C* on observer-ratings and evidence of shared environmental overlap with the other informants (as we expected), the results would provide compelling support for prior claims that shared environmental influences constitute a meaningful source of variance within adolescent antisocial behavior.

Methods

Participants

Participants were from the Sibling Interaction and Behavior Study (SIBS), a population-based study of adoptive and biological adolescent siblings and their parents. Adoptive families living in the Twin Cities greater metropolitan area were contacted based on records for the three largest adoption agencies in Minnesota (averaging between 600 and 700 placements a year), and were selected to have 1) an adopted adolescent placed as an infant and first assessed between the ages of 11 and 19 years, and 2) a second non-biologically

related adolescent sibling falling within the same approximate age range. Biological families were ascertained from Minnesota birth records and located using public databases. Other eligibility requirements for adoptive and biological families included living within driving distance of our Minneapolis-based laboratory, participating siblings no more than 5 years apart in age, and the absence of cognitive or physical handicaps that would preclude completion of our daylong assessment (McGue et al., 2007). Among eligible families, 63% of adoptive and 57% of biological families participated. Although participating biological mothers were significantly more likely to have a college degree (44%) than non-participating biological mothers (29%), participating and non-participating adoptive and biological families otherwise did not differ. Detailed information on sample recruitment and participation is available in McGue et al. (2007). Informed consent or assent was obtained from all participants.

The current sample consisted of the 406 biologically-unrelated and 204 biologically-related families where at least one of the two adolescents was younger than 19 ($n=1,199$ adolescent siblings, 604 mothers, and 541 fathers). Adolescents ranged in age from 10 to 18 years (average 14). A little over half of the sample was female (55%). The adoptive and biological parents (and therefore, the biological adolescents) were broadly representative of the ethnic composition of the Minnesota population at the time they were born; approximately 95% were Caucasian. However, due to predominantly international adoptions in Minnesota, the adopted adolescents were 67% Asian-American, 21% Caucasian, 2% African-American, 2% East Indian, 3% Hispanic/Latino, 1% South or Central American Indian, 4% mixed race, and 0.1% other ethnicities.

Measures

Observer-ratings—Two 5-minute videotaped family interactions took place in a room decorated to look like a living room/dining room, with family members seated around a dining table (as detailed in Rueter, Keyes, Iacono, & McGue, 2009). The video camera was inconspicuously placed in a bookcase, although family members were aware that they were being videotaped. For the first task, families were presented with a Rorschach inkblot (Exner, 2002) and asked to come to a consensus about what it resembled. For the second task, families were presented with a moral dilemma (Kohlberg, 1981), in which a man's wife has been diagnosed with a fatal disease but he cannot afford to buy the only drug that can save her life. Families were asked to decide: (a) whether the man should steal the drug for his wife? and (b) whether he should also steal the drug for a stranger in need?

Trained observers then globally rated twelve family interaction characteristics using the Sibling Interaction and Behavior Study Rating Scales, adapted from the well-known Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). Each family member's behavior toward each of the other family members was rated using a 9-point scale ranging from 1 (*not at all characteristic of the person*) to 9 (*mainly characteristic of the person*). Each observer received approximately 100 hours of training and was required to pass written and observation examinations before coding videotapes. Observers attended biweekly coder meetings for ongoing training and to prevent "rater drift." Observer reliability was assessed by randomly assigning 25% of all tapes to be rated by a second observer, and then comparing the primary and secondary ratings using intraclass correlations. Inter-rater intraclass correlations ranged from .58 to .78 across all scales.

For the current study, we made use of the Antisocial (ANTI) scale. The ANTI scale assesses the extent to which an individual's behavior was characterized by anger, hostility, aggression, and contempt, as well as socially irresponsible or age-inappropriate behaviors. An adolescent scoring 9 (*mainly characteristic of the person*) was described as demonstrating "immaturity, non-compliance, irritability, whining, or may 'talk back to' or

threaten the other or actively refuse to participate in the task". In addition, he/she may "exhibit signs of physical aggression, out-of-control behavior, lack of constraint in his/her behavior, or pleasure in actively resisting the other. The focal may go out of his/her way to instigate conflict. He/she may be actively rebellious, cruel, or try to cause distress for the other". For the current study, we averaged ratings of the adolescent's behavior towards his/her mother (available on 1,154 participants), father (available on 920 participants), and sibling (available on 1,173 participants). As we allowed up to one missing rating per person, composite ANTI ratings were available on 1,174 participants (mean ANTI was 3.40 with a standard deviation of 1.74; the observed range was 1 through 9 with 5.3% of the sample scoring 7 or higher).

Adolescent antisocial behavior—We examined two indices of adolescent antisocial behavior: the Delinquent Behavior Index (DBI) and a DSM-IV Conduct Disorder (CD) symptom count. The DBI (Burt & Donnellan, 2008; Farrington & West, 1971; Gibson, 1967) is a 36-item inventory of minor (e.g., skipping school) and more serious (e.g., using a weapon in a fight) delinquent behaviors (available on 1,181 adolescents). Participants were asked whether they had engaged in each behavior in their lifetime (0 = no; 1 = yes). Items were summed. If fewer than four items were missing, items were prorated and added to the scale score. The scale demonstrated good internal consistency reliability across the sample ($\alpha = .87$).

The maternal-reported lifetime DSM-IV "symptom count" variable corresponded to the sum of endorsed or partially-endorsed criterion A symptoms of CD in the adolescents (available on 1,193 adolescents). Mothers were assessed in-person by trained bachelor and masters-level interviewers using the Diagnostic Interview for Children and Adolescents-Revised (Reich, 2000). Supplementary probes and questions were added to ensure complete coverage of each symptom. Of the 13 possible symptoms, only symptom 9 ("has forced someone into sexual activity with him or her") was not assessed.

Following the interview, a clinical case conference was held in which the evidence for every symptom was discussed by at least two advanced clinical psychology doctoral students. Audio tapes from the interview were replayed as necessary. Symptoms judged to be definitely present (i.e., clinically significant in both severity and frequency) were counted as one full symptom. Symptoms judged to be probably present (i.e., clinically significant in either severity or frequency, but not both) were counted as half of a symptom. The reliability of the consensus process was good, with a kappa of 0.79 for diagnoses of CD.

All data were log-transformed prior to analysis to adjust for positive skew (skew following transformation ranged from -.31 to 1.98). Furthermore, because siblings often differed in age and sex, analyses incorporating age and sex would be unwieldy and underpowered. We thus statistically controlled sex and age effects in our biometric analyses via regression techniques (McGue & Bouchard, 1984). Of note, however, overall conclusions were unchanged when analyses were conducted on the raw data (results not shown). By contrast, effects of ethnicity were not regressed out of these data, as mean levels of ANTI, CD, and the DBI did not vary by ethnicity (all $p \geq .49$; Cohen's $d \leq .11$).

Statistical Analyses

Correlations between reared-together biological siblings (i.e., BIO) are a function of the 50% of additive genetic influences shared between them, as well as 100% of shared environmental influences. As noted, because reared-together adopted siblings (i.e., ADOP) do not share segregating genes, correlations between them are solely a function of their common familial and contextual environment, thereby functioning as a "direct estimate" of C. Sibling differences are a function of both genetic influences not shared by siblings and

non-shared environmental influences. Measurement error, which similarly acts to reduce sibling similarity, is also contained within non-shared environmental influences.

For our primary analyses, we made use of a full ACE biometric Cholesky decomposition structural equation model, which allowed us to explicitly parameterize A, C, and E (non-shared environmental) contributions to both the variance within and the covariance among the three measures. The genetic and environmental covariances were then standardized on their respective variances to produce genetic and environmental correlations. These statistics reveal the extent to which a specific effect (e.g., the genetic effect) on observer-ratings is correlated with the same effect on each of the other measures of antisocial behavior. In this way, the Cholesky model enabled us to offer focused conclusions on both the origins of observer-ratings individually, as well as the etiology of their overlap with the questionnaire and diagnostic interview assessments.

Mx, a structural-equation modeling program (Neale, Boker, Xie, & Maes, 2003), was used to perform the model-fitting analyses. Because of the small amount of missing data, we made use of Full-Information Maximum-Likelihood raw data techniques, which produce less biased and more efficient and consistent estimates than pairwise or listwise deletion in the face of missing data. When fitting models to raw data, their variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; $-2\ln L$). The $-2\ln L$ under this unrestricted baseline model is then compared with $-2\ln L$ under more restrictive biometric models. This comparison provides a likelihood-ratio chi-square test of goodness of fit for the model, which is then converted to the Akaike's information criterion (Akaike, 1987); $AIC = \chi^2 - 2df$, the traditional fit index of behavioral genetics research. The AIC measures model fit relative to parsimony. Better fitting models have more negative values.

Results

Observer-ratings were modestly correlated with questionnaire and diagnostic interviews of adolescent antisocial behavior ($r = .19$ and $.13$, respectively, both $p < .01$). Although small, the magnitude of these associations are consistent with the cross-measure/informant correlations seen in other studies (Achenbach, McConaughy, & Howell, 1987; Arseneault et al., 2003). The self-report DBI questionnaire and maternal-informant CD variables were similarly correlated $.22$ ($p < .01$). In short, although based on only 10 minutes of observation, observer-ratings of ANTI appear to evidence cross-informant associations similar to those across various lifetime informant-reports of antisocial behavior.

Sibling Similarity

Prior to multivariate model-fitting analyses, intraclass and cross-sibling, cross-informant correlations were calculated for BIO and ADOP siblings (see Table 1) using the double entry method. Genetic influences are preliminarily implied by BIO correlations that are larger than ADOP correlations, as tested via Fisher's z transformations. Shared environmental influences are implied by ADOP correlations that are greater than zero. The BIO sibling correlations for observer-ratings of ANTI was significantly larger than those for ADOP siblings (i.e., $.48$ and $.32$, respectively), offering early evidence of A on ANTI. The BIO correlations for the DBI and the CD diagnostic interview were also generally larger than their corresponding ADOP correlations, providing evidence of genetic influences on these measures as well. Evidence supporting C was unambiguous, as evidenced by ADOP intraclass correlations that were uniformly greater than zero (at $p < .01$).

The cross-sibling, cross-measure correlations permit us to preliminarily examine the origins of covariance across the measures. These correlations were calculated using, for example,

the CD symptom count of one sibling and the observer-ratings of his or her co-sibling. Results are clearly suggestive of shared environmental influences on the covariance across measures, as the cross-sibling, cross-measure correlations for ADOP siblings were uniformly greater than zero (though only marginally so for the association between CD and the DBI). By contrast, there was little evidence supporting genetic influences on measure covariance, as the BIO cross-sibling correlations were equivalent to their corresponding ADOP correlations.

Cholesky Trivariate Model

We initially estimated variances, covariances, and means for the raw data to get a baseline index of fit ($-2\ln L = 9561.570$, $df = 3442$), after which we fitted the trivariate Cholesky model ($-2\ln L = 9625.806$, $df = 3475$; $X^2 = 64.236$ on 33 df , $AIC = -1.764$). The negative AIC value implies that our model fit the data reasonably well. Results are presented in Table 2. As seen there, genetic influences appeared to comprise the largest source of variance in observer ratings of ANTI (45%), although C was also prominent (31%). These estimates were quite comparable to those for the DBI and CD. Such results thus suggest that observer-ratings of ANTI behave similarly to other informant-reports of adolescent antisocial behavior at the etiological level.

The sources of overlap across the measures highlighted the presence of (at least some) consistent shared environmental effects. The shared environmental correlations, while only moderate in magnitude, were consistently larger than zero, thereby suggesting that a portion of the C on observer-ratings overlap with those on the DBI and CD. C also overlapped somewhat across maternal-reports of CD and the self-report DBI. Genetic and non-shared environmental sources of overlap were uniformly non-significant, suggesting that these influences are largely unique to each measure.

Post-hoc analyses—Our observer-rating variable was an average of ANTI towards each of the other family members present in the interaction, including ANTI towards the sibling. As each sibling was ultimately a focal, however, the dyadic nature of their interaction could have introduced some element of bias in our findings. Critically, however, ANTI towards siblings is an important independent predictor of adolescent antisocial behavior. When ANTI towards parents and ANTI towards siblings were simultaneously entered into a regression of the DBI, both emerged as significant predictors (standardized beta weights = .11 and .09, respectively, both $p < .05$). The same general pattern held for maternal-reported CD, albeit with slightly weaker effects (standardized beta weights = .05 and .08, respectively; the latter was marginally significant at $p < .08$). Given these results, we elected to retain ANTI towards siblings in our observer-ratings variable.

However, to ensure that our decision to combine behaviors towards all three family members did not unduly influence our results, we repeated our analyses restricting the observer-ratings variable to ANTI towards parents. Estimates of genetic, shared, and non-shared environmental influences on ANTI towards parents were 52%, 19%, and 29%, respectively (all $p < .05$), results that are quite similar to those reported above. Genetic and shared environmental correlations with CD and the DBI were also similar (i.e., genetic correlations were .16 and -.10, respectively; shared environmental correlations were .32 and .38, respectively). Our conclusions thus do not appear to be dependent on the specific operationalization of our observer-ratings variable, highlighting the robustness of our results.

Discussion

Results revealed that the shared environment made a meaningful contribution to observer-ratings of ANTI (contributing 31% of the variance), as did genetic and non-shared environmental influences (45% and 24%, respectively). Moreover, a portion of these shared environmental influences on observer-ratings overlapped with those on the self-report DBI questionnaire and the maternal-report CD symptom count. Such results provide an important extension of previous work (Burt, 2009), as they suggest that neither rater bias nor passive genotype-environment correlations serve as primary explanations for earlier findings of shared environmental influences on adolescent antisocial behavior. Moreover, because at least some of these shared environmental effects persist across other informant/assessment strategies, these results bolster prior claims (Burt, 2009) that shared environmental influences are tapping relatively persistent and systematic sources of variance.

There are several limitations to bear in mind when interpreting the results of this study. First, sex was regressed out of all three measures prior to analysis. Fortunately, prior meta-analyses (Burt, 2009; Rhee & Waldman, 2002) have indicated that heritability estimates for antisocial behavior do not generally vary across sex, suggesting that our decision to exclude sex is unlikely to have impacted our results. Next, because there is some suggestion that the heritability of antisocial behavior may vary by age/age-of-onset (Moffitt, 2003), the current results apply only to adolescence and not to other developmental periods. Also of note, ethnicity configurations varied across the families. In particular, although the biological siblings were necessarily the same ethnicity (and 95% Caucasian), the adoptive siblings were not. For example, both siblings were of Asian ancestry in 165 families, both were of Caucasian ancestry in 73 families, and the siblings had different ethnic backgrounds in 115 families. To evaluate the potential impact of same versus different sibling ethnicity on the observer-ratings, we computed intraclass correlations separately across these three adoptive family types. Results strongly suggest that sibling ethnicity had little to no impact on our results as correlations did not vary across sibling ethnicity configurations ($r_s = .36, .28$, and $.30$, respectively, for Asian-Asian families, Caucasian-Caucasian families, and different ethnicity families; all $p < .05$).

Another limitation concerns the Equal Environment Assumption (EEA), upon which biometric SEM analyses explicitly rely. When applied to the current design, the EEA assumes that BIO sib pairs are no more likely to share the environmental factors etiologically relevant to the phenotype under study than are ADOP sib pairs. There is no empirical testing of this assumption, to our knowledge, in the adoptive/biological sibling design, making its applicability uncertain. Critically, however, violations of the EEA would not affect our estimates of shared environment, as adoptive sibling correlations provide a “direct” estimate of shared environmental effects. As such, it is highly unlikely that our core findings are an artifact of the EEA.

Finally, our observer-rating variable included ANTI towards the sibling. As noted previously, because each sibling was ultimately a focal, the dyadic nature of their interaction could have inflated our estimates of the shared environment for the observer-ratings. The somewhat higher estimates of the shared environment obtained for the overall ANTI composite (31%) as compared to the composite of ANTI towards parents only (19%) lend some support for this notion. Importantly, however, our ultimate conclusions, and particularly those suggesting that shared environmental influences overlap somewhat across informants, remained entirely unchanged. Even so, it would be important for future research to replicate these findings using observer-ratings that are obtained separately for each sibling.

Conclusions

The current study has two important implications. Behavioral genetic research has historically concluded that the more important environmental influences on psychological and behavioral outcomes result in differences between siblings (Plomin & Daniels, 1987), a conclusion that continues to influence theory and interpretation of environmental influences up to the present day. More recent research, however, has suggested moderate influences of the shared environment on child and adolescent psychopathology, including antisocial behavior (Burt, 2009). The results of the current study offer robust additional support for this conclusion. Specifically, even though our analysis of adoptive sibling similarity definitely excludes passive rGE from our estimates of the shared environment, shared environmental influences continued to consistently emerge as a prominent source of variance in these data (20-31%). Passive rGE thus does not appear to explain the presence of shared environmental influences on adolescent antisocial behavior. The current results also indicated that shared environmental influences also cannot be solely attributed to the rater bias thought to impact parent and child informant-reports (e.g., lack of knowledge of normative behavior, idiosyncratic interpretation, etc.), as these effects were found for observer-ratings of the adolescents' behavior as well as for child and parent informant-reports. They also persisted, to some extent, across all three informant/assessment strategies, a rather remarkable finding given that the questionnaire and diagnostic interview reporting periods were lifetime whereas the observer-ratings were based on two brief family interactions. In sum, the current results offer a critical extension of prior work indicating that the role of the shared environment in child and adolescent antisocial behavior was dismissed too soon. Future research should thus meaningfully consider the role of the shared environment in theoretical and empirical work on antisocial behavior in particular, as well as on child and adolescent psychopathology more generally.

The prominence of genetic influences on observer-ratings of antisocial behavior is also worthy of additional discussion. Prior research has indicated that genetic influences on antisocial behavior appear to increase with the inclusion of multiple informants (Arsenault et al., 2003). The current results offer an important addition to this finding, as they indicate that genetic influences also have a potent influence on even very thin slices of behavior (accounting for 45% of the observed variance), and extend even to non-twin designs. In short, genetic influences should also be seriously considered in all etiological and intervention research on antisocial behavior.

Key Points

- Extant behavioral genetic research on antisocial behavior has relied largely on specific informant-reports (and particularly parent and child reports), each of which are subject to various sources of rater bias
- Observer-ratings of adolescents' behaviors avoid many forms of rater bias, and are thus particularly well-suited to verify findings regarding the etiology of behavior
- There are moderate and significant shared environmental influences on adolescent antisocial behavior regardless of informant or measurement strategy, a portion of which overlap with those on observer-ratings
- The role of the shared environment in child and adolescent antisocial behavior was dismissed too soon

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Table 1
Intraclass and Cross-Sibling, Cross-measure Correlations by Family Adoption Status

Measure	Observer-ratings of ANTL, sibling 2	Diagnostic interview of CD, sibling 2	Self-report DBI questionnaire, sibling 2
Observer-ratings of ANTL, sibling 1	.48 [*] /.32 [*]	.09 [*]	.09 [*]
Diagnostic interview of CD, sibling 1	.05	.38 [*] /.24 [*]	.07 [~]
Self-report DBI questionnaire, sibling 1	.11 [*]	.09 [~]	.43 [*] /.20 [*]

Note. Intraclass correlations are presented on the diagonal, first for BIO siblings (who share roughly 50% of their segregating genes) and then for ADOP siblings (who do not share any segregating genes). Cross-sibling, cross-informant correlations are presented on the off-diagonal (BIO correlations are below the diagonal, and ADOP correlations are above the diagonal).

*
p < .05

~
p < .10

Table 2
Standardized parameter estimates from the Biometric Cholesky Decomposition Model

Component of variance	Observer-ratings (ANTI)	Diagnostic interview (CD)	Self-report questionnaire (DBI)	$r_{\text{ANTI-CD}}$	$r_{\text{ANTI-DBI}}$	$r_{\text{CD-DBI}}$
A	45%* (18, 69)	48%* (18, 75)	44%* (14, 72)	-.01 (-.52, .44)	.16 (-.37, .62)	.03 (-.59, .47)
C	31%* (22, 39)	23%* (14, 31)	20%* (10, 30)	.32* (.07, .58)	.34* (.06, .63)	.33~ (.00, .65)
E	24%* (5, 48)	29%* (7, 56)	36%* (14, 61)	.12 (-.55, .76)	.11 (-.52, .61)	.34 (-.21, .86)

Note. A, C, and E represent genetic, shared, and non-shared environmental influences, respectively. Univariate variance estimates are presented in columns 2-4. Genetic and environmental correlations are presented in columns 5-7. 95% confidence intervals are presented below the variance estimates and the correlations.

* $p < .05$

~ $p = .05$