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Are there sex differences in the etiology of youth antisocial behavior?

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Abstract

Sex differences in the etiology of youth antisocial behavior are an intuitively appealing hypothesis given the consistently higher prevalence of antisocial behavior in boys versus girls. Although a few early studies supported this possibility, reporting stronger genetic influences in females and stronger environmental influences in males, subsequent meta-analyses found that antisocial behavior was equally heritable in males and females. Critically however, none of the meta-analyses evaluated whether sex differences in etiology might be enhanced in particular sub-populations or contexts. The current study sought to do just this. We examined 1,030 child twin pairs from the Michigan State University Twin Registry (MSUTR), half of whom were over-sampled for neighborhood disadvantage, thereby allowing us to meaningfully evaluate whether sex differences in etiology were enhanced in disadvantaged contexts. We also directly evaluated the possibility of sex differences in the etiology of teacher- versus maternal-informant reports of antisocial behavior, evaluating each informant-report for possible sex differences. Results were not consistent with differential effects of sex on etiology in disadvantaged versus advantaged contexts, but did suggest moderation by informant-report. Namely, genetic influences were stronger in girls, and environmental influences were stronger in boys, when antisocial behavior was assessed using teacher informant-reports, but not when assessed using maternal informant-reports. Critically, these findings were confirmed when we re-analyzed meta-analytic data from Burt (2009a) separately by informant. Such findings suggest that, at least in school contexts, the etiology of antisocial behavior does indeed vary across sex. Implications are discussed.

Keywords

antisocial behavior; sex differences; GxE; meta-analysis

Antisocial behavior is defined as actions that violate societal norms and the personal or property rights of others, and includes at least two correlated symptom dimensions (see review by Tremblay, 2010), an overt or aggressive and oppositional dimension (fighting, hitting, bullying, anger, defiance) and a covert or non-aggressive/rule-breaking dimension (stealing, lying, vandalism). Males engage in notably higher rates of antisocial behavior than females, with a 2:1 to 10:1 male-female ratio beginning in the toddler years and continuing throughout the lifespan (see, for example, Moffitt, 2003). Although sex differences are

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observed for rule-breaking forms of antisocial behavior, including theft, vandalism, and status violations (Bongers, Koot, van der Ende, & Verhulst, 2004; Li & Lee, 2010), there is evidence that the magnitude of sex differences in prevalence may be especially pronounced for overt physical aggression (Monuteaux, Fitzmaurice, Blacker, Buka, & Biederman, 2004; van Lier, Vitaro, Barker, Koot, & Tremblay, 2009). Moreover, these sex differences in aggression persist across numerous human societies (Archer, 2004; Ramirez, Andreu, & Fujihara, 2001) and across most mammalian species, including humans' nearest phylogenetic cousin, the chimpanzee (Gray, 1971; Maccoby & Jacklin, 1980; Manson et al., 1991). Such results collectively suggest that natural selection may have favored aggression in males at some time in our evolutionary history (Archer, 2009), perhaps to promote protection of kin and social group.

Given these unambiguous sex differences at the level of actual behavior, one might expect equally robust differences in genetic and environmental etiology across sex. These differences in etiology, if they exist, could be either qualitative or quantitative in nature. Findings of qualitative sex differences would indicate that different etiologic factors (e.g., different genes, different environments) influence the development of antisocial behavior in boys versus girls. Findings of quantitative sex differences, by contrast, would indicate that, although the genetic and environmental influences underlying antisocial behavior do not differ across sex, the magnitude of those influences does differ, such that genetic and/or environmental influences are more important in one sex versus the other.

A number of genetically-informed studies have sought to uncover qualitative or quantitative sex differences in the etiology of antisocial behavior. A recent genome-wide association study, for example, concluded that some of the specific genes associated with antisocial behavior varied across sex (Tielbeck et al., in press). Critically, however, no twin and adoption study to our knowledge has provided compelling evidence of qualitative sex differences in etiology (e.g., Cloninger, Christiansen, Reich, & Gottesman, 1978; Jacobson, Prescott, & Kendler, 2002), an important set of null findings given that twin designs are able to disambiguate and simultaneously evaluate both genetic and environmental influences (something that cannot be done using molecular genetic designs). Jacobson and colleagues (2002), for example, searched for qualitative sex differences in a large longitudinal study with over 1,000 opposite sex twin pairs (the informative unit of analysis in twin studies of qualitative sex differences), and found no evidence for qualitative sex differences. Extant data thus largely suggested that the specific genetic and environmental influences contributing to antisocial behavior are the same in males and females.

In terms of quantitative sex differences, large-scale meta-analyses of twin and adoption studies have consistently suggested that antisocial behavior is equally heritable in males and females (Burt, 2009a, 2009b; Rhee & Waldman, 2002). Burt (2009a), for example, collected 103 twin and adoption studies examining the etiology of youth antisocial behavior. After adjusting for non-independence and other possible confounders, aggression data were available on 14,373 pairs in 19 samples, and rule-breaking data were available on 8,103 pairs in 15 samples. Analyses revealed that, although aggression was more heritable than non-aggressive antisocial behavior in both boys and girls, neither phenotype was differentially heritable across sex.

Could there still be sex differences in the etiology of antisocial behavior?

Given the very large sample sizes seen in most meta-analyses, null results are generally considered robust (i.e., they are unlikely to reflect Type 2 error). Critically, however, it is worth noting that more than half of the twin studies examined in above meta-analyses regressed sex out of their data prior to analysis (a very common practice in BG studies; McGue & Bouchard, 1984). As these studies are not able to inform our understanding of sex differences, they were excluded when examining sex as a moderator of heritability estimates. For example, only ~45% of the studies examined in Burt (2009a) reported intraclass correlations separately across sex (N's in the sex difference analyses were 6,350 for aggression and 3,826 for rule-breaking). And although these sample sizes are still large enough to draw firm inferences, the exclusion of so many studies/sibling pairs does somewhat reduce confidence in the conclusions.

The other salient observation is that, although they did not reach significance in the various meta-analyses, quantitative (but not qualitative) sex differences have emerged in a number of empirical studies (Eley, Lichtenstein, & Stevenson, 1999b; Jacobson et al., 2002; Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998; Silberg et al., 1994; E. J. Van den Oord, D. I. Boomsma, & F. C. Verhulst, 1994). Although some studies have reported higher genetic influences in boys as compared to girls (Silberg et al., 1994; E. J. Van den Oord et al., 1994), the opposite pattern has generally received more support (Eley et al., 1999b; Jacobson et al., 2002; Langbehn et al., 1998). Eley and colleagues (1999) examined the etiology of parental-reports of child antisocial behavior in a sample of 1,022 Swedish twin pairs, and in an independent sample of 501 British twin pairs. Results in both samples pointed squarely to significant sex differences in the etiology of non-aggressive antisocial behavior, such that shared environmental influences were larger in boys while genetic influences were larger in girls. The same pattern of results emerged for aggression, although the differences were not statistically significant. Jacobson and colleagues (2002) examined retrospective self-reports of antisocial behavior in a large sample (N = 6,806 twins) during late childhood, adolescence, and adulthood. As noted above, they found no evidence of qualitative sex differences, but they did find evidence of significant quantitative sex differences in childhood. As with Eley et al. (1999), results again pointed to higher shared environmental in boys and higher genetic influences in girls. A yoked adoption study by Langbehn and colleagues (1998) came to a similar conclusion. They found that Conduct Disorder in adopted males was predicted by adoptive family environment alone whereas biological background (as assessed via antisocial behavior in the biological parents) and interactions between biological background and adoptive family environment predicted Conduct Disorder in adopted females.

A possible role for joint moderation

The above empirical studies thus collectively suggest that the etiology of antisocial behavior might vary across sex. And yet, multiple meta-analyses have failed to find evidence of quantitative sex differences. How do we rectify these seemingly opposed results? The positive findings for specific studies could simply represent sampling variation, whereby the results of each study vary around the true score. Another possibility is that etiologic sex

differences in antisocial behavior are confined to specific sub-populations or contexts (which were collapsed together for the meta-analyses, thereby obscuring sex differences in etiology). This latter possibility is somewhat more feasible that it might initially seem. None of the meta-analytic examinations of sex differences considered joint etiologic moderation by other moderators (Burt, 2009a, 2009b; Rhee & Waldman, 2002). This is a potentially problematic omission, since multiple studies have documented robust and replicable changes in the heritability of antisocial behavior across a number of moderators. As one key example, prior meta-analyses have confirmed notable informant effects on heritability estimates, such that genetic influences are larger when examining teacher or maternal informant-reports of their twins than when examining twin self-reports (Burt, 2009a, 2009b). Shared environmental influences, for their part, are stronger for maternal informant-reports than for teacher-informant reports or twin self-reports. These results may partially reflect situational specificity, or differences in informant perspective and/or exposure to the children's behavior (Achenbach, McConaughy, & Howell, 1987). In other words, it may be that antisocial behaviors are expressed differently and for different reasons in various contexts, and accordingly appear more or less heritable/environmental across those contexts (Burt, McGue, Krueger, & Iacono, 2005). If true, it is certainly possible that, just as antisocial behavior is more or less heritable across specific informants, sex differences in etiology emerge for one informant and not another.

Yet another potential moderator relates to the level of disadvantage in the child's broader environmental context. An emerging literature (Burt, Klump, Gorman-Smith, & Neiderhiser, 2016; Cleveland, 2003; Tuvblad, Grann, & Lichtenstein, 2006) has pointed to notable differences in etiology by disadvantage, such that genetic influences on antisocial behavior are suppressed, and environmental influences enhanced, in disadvantaged contexts relative to advantaged contexts. Given this, it is conceivable that sex differences in etiology are more pronounced in disadvantaged contexts than in advantaged contexts, and that these differences have been obscured in extant meta-analyses by the middle- to upper-class backgrounds of most twin study participants. Indeed, there is some data (Odgers, Donley, Caspi, Bates, & Moffitt, 2015) suggesting that neighborhood socioeconomic status and economic distance is differently associated with antisocial behavior in boys and girls. Odgers et al. (2015) found that low-income twins living in economically mixed neighborhoods engaged in more aggressive and nonaggressive antisocial behavior than those living in concentrated poverty (even after controlling for other neighborhood and family-level risk factors), but that this effect was limited to boys only. Findings such as these suggest that environmental context may be more influential in the development of antisocial behavior in boys as compared to girls.

Current Study

The current study evaluated informant and disadvantage as moderators of etiologic sex differences in a large (N = 1,030 twin pairs) and well-characterized sample of twins in middle childhood. We specifically focused on twins, since molecular genetic samples are only able to explore genetic influences, while traditional (non-twin) family samples are able to examine familial influence but cannot distinguish between genetic and environmental influences. We also focused on biological sex, rather than gender identity, as the latter data

were not collected. We specifically evaluated the possibility of sex differences in the etiology of teacher- and/or maternal-informant reports of antisocial behavior (the two most valid and widely-used informant-reports; Achenbach et al., 1987). We also leveraged our unique at-risk twin sample, in which twin families were over-sampled for neighborhood disadvantage, to evaluate whether sex differences in etiology were enhanced in disadvantaged contexts. Results should clarify whether there are sex differences in the etiology of antisocial behavior in specific sub-populations of children.

METHODS

PARTICIPANTS—Participants were drawn from the Twin Study of Behavioral and Emotional Development in Children (TBED-C), a study within the population-based Michigan State University Twin Registry (MSUTR) (Burt & Klump, 2013). The TBED-C includes both a population-based sample (N = 528 families) and an independent at-risk sample for which inclusion criteria also specified that participating twin families lived in neighborhoods with neighborhood poverty levels at or above the Census mean at study onset (10.5%) (N = 502 families). To be eligible for participation in the TBED-C, neither twin could have a cognitive or physical condition (as assessed via parental screen; e.g., a significant developmental delay) that would preclude completion of the assessment. The TBED-C was approved by the Michigan State University IRB (#04-887, entitled “Genotype-environment interactions in child conduct problems”). Children provided informed assent, while parents provided informed consent for themselves and their children.

The Department of Vital Records in the Michigan Department of Health and Human Services (formerly the Michigan Department of Community Health) identified twins in our specified age-range via the Michigan Twins Project, a large-scale population-based registry of twins in lower Michigan that were recruited via birth records. The Michigan Bureau of Integration, Information, and Planning Services database was used to locate family addresses no more than 90–120 miles of East Lansing, MI through parent drivers’ license information. Pre-made recruitment packets were then mailed on our behalf by the Michigan Department of Health and Human Services to parents. A reply postcard was included for parents to indicate their interest in participating. Interested families were contacted directly by project staff. Parents who did not respond to the first mailing were sent additional mailings approximately one month apart until either a reply was received or up to four letters had been mailed.

This recruitment strategy yielded an overall response rate of 57% for the at-risk sample and 63% for our population-based sample, which are similar to or better than those of population-based twin registries that use anonymous recruitment mailings (Baker, Barton, & Raine, 2002; Hay, McStephen, Levy, & Pearsall-Jones, 2002). A brief questionnaire was completed by families participating in the Michigan Twins Project, from which this sample was recruited, thereby allowing us to not only compare families in the at-risk and population-based samples at the time of recruitment, but perhaps more importantly, to compare families who chose to participate versus those who were recruited but did not participate. Compared to the population-based sample, the at-risk sample reported lower mean family incomes ($72,027 and $57,281, respectively; Cohen’s *d* effect size = −.38),

higher paternal felony convictions (d = .30), and higher rates of youth conduct problems and hyperactivity (d = .34 and .27, respectively), although they did not differ in youth emotional problems (d = .08, ns). However, both samples were largely representative of non-participating families. As compared to non-participating twins, participating twins were experiencing similar levels of conduct problems, emotional symptoms, or hyperactivity (d ranged from -.08 to .01 in the population-based sample and .01 to .09 in the at-risk sample; all ns). Participating families also did not differ from non-participating families in paternal felony convictions (d = −.01 and .13 for the population-based and the at-risk samples, respectively), rate of single parent homes (d = .10 and −.01 for the population-based and the at-risk samples, respectively), paternal years of education (both $d \leq .12$), or maternal and paternal alcohol problems (d ranged from .03 to .05 across the two samples). However, participating mothers in both samples reported slightly more years of education (d = .17 and .26, both $p < .05$) than non-participating mothers. Maternal felony convictions differed across participating and non-participating families in the population-based sample (d = −.20; $p < .05$) but not in the at-risk sample (d = .02). All told, we do not believe these differences significantly compromise the generalizability of these data.

The twins in the TBED-C ranged in age from 6 to 10 years (mean = 7.7, SD = 1.51; although 27 pairs had turned 11 by the time the family participated) and were 48.7% female. Families were somewhat more racially diverse than the local area population (e.g., 10% Black and 82% White versus 5% Black and 85% White). Zygosity was established using physical similarity questionnaires administered to the twins' primary caregiver (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of at least 95% when compared to DNA. The current study included 224 monozygotic (MZ) male pairs, 211 dizygotic (DZ) male pairs, 202 MZ female pairs, 206 DZ female pairs, and 187 DZ opposite-sex pairs.

MEASURES

Child Antisocial Behavior: Mothers completed the Achenbach Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) separately for each twin, while the twins' teacher(s) completed the corresponding Achenbach Teacher Report Form (TRF; Achenbach & Rescorla, 2001), a commonly used and well-validated family of instruments for assessing aggressive and rule-breaking antisocial behaviors prior to adulthood. For the current study, we examined both the Rule-breaking Behavior (RB) (e.g., lies, breaks rules, steals, truant; 17 items on the CBCL and 12 items on the TRF) and the Aggressive Behavior (AGG) (e.g., destroys others' things, fights, threatens others, argues, suspicious, temper; 18 items on the CBCL and 20 items on the TRF) scales. Maternal-reports were available on nearly all twins (98.7%; N = 2,034). The teachers of 119 twins were not available for assessment (because the twins were home-schooled, because teacher contact information was incorrect, etc.). Our final teacher participation rate was 83%, with TRF data available for 1,551 participants. To adjust for positive skew, all scales were log-transformed prior to analysis to better approximate normality.

Disadvantage: We examined three dichotomous indicators (i.e., family poverty, neighborhood poverty, and exposure to community violence; detailed below). These various

indices of disadvantage were only modestly-to-moderately associated with one another, with correlations ranging from .21 to .32. Given that, we combined our three dichotomous indicators to operationalize disadvantage as a broader construct, requiring that families qualify as disadvantaged across at least 2 of the 3 indicators to be coded as 'disadvantaged' in our analyses. For those families missing data on one indicator (i.e., 6%), we required that families qualify as disadvantaged on only 1 of the 2 remaining indicators. More than 40% of our families (41.4%, or 426 of the 1,030 families) met these criteria.

Family poverty was indexed via maternal reports of combined family income. Because the living wage for one parent and two children (the minimum number of family members in the household) in Michigan is $56,693 (http://livingwage.mit.edu/states/26), we used $55K as our cut-point for familial poverty (0 = $60K or more; 1 = $55K or less). Just under half (45.6%) of families reported annual incomes at or below living wages.

For neighborhood poverty, we collected information on the proportion of Census tract residents living below the poverty line from www.Census.gov. Given that all families were recruited from 2008 onwards, we focused here on the 2008–2012 census data. In these data, 2008–2012 neighborhood poverty rates ranged from 0 to 93%, with a mean of 17.7%. As in prior publications (Burt et al., 2016), neighborhood disadvantage was coded as either "low" (0–19.9%; N = 690 families) or "high" (20+%; N = 329 families). This cut-point was chosen in accordance with recent work indicating that the effects of neighborhood poverty on youth outcomes are very small until poverty reaches 20% (Galster, 2010).

Our final indicator of disadvantage centered on exposure to community violence (ECV), as assessed using maternal reports on the KID-SAVE (Flowers, Hastings, & Kelley, 2000). For the current study, we focused on the 17-item indirect violence scale. Items are specifically assessed with regards to the family's current neighborhood, and include such items as "I run for cover when people start shooting", "I have seen the police arrest someone", "I have seen someone pull a gun on someone else". To qualify as experiencing significant ECV, we required an endorsement of at least ~30% of the items in the scale (i.e., 5 or more). 44% of the sample met this criterion.

ANALYSES—Classical twin studies leverage the difference in the proportion of genes shared between MZ twins (who share 100% of their genes) and DZ twins (who share an average of 50% of their segregating genes) to estimate additive genetic (A), shared environmental (i.e., environmental factors that make twins similar to each other; C), and non-shared environmental (i.e., factors that make twins different from each other, including measurement error; E) contributions to a given phenotype. More information on twin studies is provided elsewhere (Neale & Cardon, 1992).

For our first set of analyses, we evaluated whether child sex moderated the etiology of maternal and teacher informant-reports of twin RB and AGG using the 'extended univariate GxE' model (van der Sluis, Posthuma, & Dolan, 2012), an extension of the univariate GxE model (Purcell, 2002). In this model (see Figure 1), the variance decompositions of RB and AGG, respectively, were each modeled as a function of child sex (dummy-coded so that 0 = girl and 1 = boy). The sexes of both twins were first entered in a means model of each twin's

AGG (or RB). Moderation was then modeled on the residual AGG/RB variance (i.e., that which does not overlap with sex). Twins are not required to be concordant on the value of the moderator (although they can be). The first and least restrictive of these models allows for both linear moderation of A, C, and E contributions. We then fitted the more restrictive no moderator models, constraining the linear moderators to be zero and evaluating the reduction in model fit. Purcell (2002) recommends that unstandardized estimates be presented for all GxE models, as standardized or proportional estimates can obscure absolute changes with the moderator. We thus standardized our log-transformed RB and AGG scores to have a mean of zero and a standard deviation of one prior to analyses to facilitate interpretation of the unstandardized values.

To evaluate whether sex differences in etiology were enhanced in disadvantaged contexts, we then fitted the two-moderator extension of the univariate GxE model. This model allows two variables to simultaneously moderate a given outcome and to interact with one another when doing so. We first examined moderation by disadvantage (sex could also be examined, but those analyses are done above), constraining the moderation terms for disadvantage to zero and evaluating reductions in model fit. We also evaluated whether the synergistic (sex-by-disadvantage) moderation terms could be constrained to zero. The final terms are the critical ones in our case, as they directly evaluate the possibility of synergistic interactions between moderators. Put differently, we were able to statistically evaluate whether the moderating effects of sex on etiology were accentuated (or dampened) in advantaged versus disadvantaged contexts.

Mx, a structural-equation modeling program (Neale, Boker, Xie, & Maes, 2003), was used to fit models to the transformed raw data using Full-Information Maximum-Likelihood raw data techniques. When fitting models to raw data, variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; -2lnL). Model fit for the more restrictive biometric GxE models was then evaluated using four information theoretic indices that balance overall fit (via -2lnL) with model parsimony: the Akaike's Information Criterion (AIC; Akaike, 1987), the Bayesian Information Criteria (BIC; Raftery, 1995), the sample-size adjusted Bayesian Information Criterion (SABIC; Sclove, 1987), and the Deviance Information Criterion (DIC; Spiegelhalter, Best, Carlin, & Van Der Linde, 2002). The lowest or most negative AIC, BIC, SABIC, and DIC among a series of nested models is considered best. Because fit indices do not always agree (they place different values on parsimony, among other things), we reasoned that the best fitting model should yield lower or more negative values for at least 3 of the 4 fit indices (as done in prior work; see Burt et al., 2016).

Confirmatory analyses of informant effects

No matter how compelling the findings, the results of any one empirical study cannot overturn meta-analytic conclusions. We thus sought to meta-analytically validate the results of our informant-report analyses, reanalyzing the sex differences data from the Burt (2009a) meta-analysis separately for maternal and teacher informant-reports, an important extension since the sex differences analyses in Burt (2009a) collapsed across informants. Note that we were not able to perform similar analyses for disadvantage, as these data were rarely

available (nearly all twin and adoption studies examine largely middle-class samples, and are thus not well-suited for examinations of disadvantage).

Extensive details on the literature search strategy and the operationalization of AGG and RB are detailed in Burt (2009a). When examining biological sex as a potential moderator in Burt (2009a), analyses were restricted to the 8 RB studies and 9 AGG studies in which intraclass correlations were presented separately by sex (i.e., male-male pairs versus female-female pairs). This approach allowed us to directly compare etiology across boys and girls. Note that there were no teacher informant-reports of child RB that were presented separately across sex, and thus our teacher analyses were restricted to AGG. We made use of Mx to fit models to the observed correlation matrices (as done in Burt, 2009a) using maximum-likelihood model-fitting techniques. The chi-square test statistic provides a goodness-of-fit index of the model to the observed correlation matrices. These chi-square values are then converted to the AIC (in Mx, analyses conducted using correlation matrices provide fewer fit indices than raw data analyses).

RESULTS

Descriptive statistics for AGG and RB are presented in Table 1. Independent samples t-tests indicated that boys engaged in significantly higher levels of antisocial behavior than girls, regardless of informant or type of antisocial behavior (all $p < .001$). Mean levels of AGG and RB were also higher in mothers than in teachers, although these differences are difficult to interpret given the different number of items on the teacher and parent versions of the questionnaire. AGG and RB were modestly and positively correlated with our index of disadvantage regardless of informant (rs ranged from .06 to .14, all p<.03). Both AGG and RB decreased slightly with age, and did so regardless of informant (rs ranged from −.06 to −.09, all p<.01). When assessed via teacher informant-reports, twin RB and AGG also varied by ethnicity, such that they were less common in White participants than in non-White participants (point biserial rs were .16 and .13, respectively). There was less evidence of ethnic differences when examining maternal informant-reports (both rs were .04). Ethnicity and age were thus regressed out of the twin data prior to analysis (McGue & Bouchard, 1984).

Are sex differences in the etiology of AGG or RB specific to a particular informant?

We examined possible sex differences in etiology by informant via formal tests of etiologic moderation (Purcell, 2002; van der Sluis et al., 2012). Model fit statistics are reported in Table 2. As seen there, the linear moderation model provided the better fit to teacher-reports of twin AGG and RB, and did so decisively across all fit indices. Such results indicate that the etiologies of teacher-reported AGG and RB do indeed vary by child sex. Parameter estimates from the best-fitting models are presented in Table 3. Because girls were dummy-coded as 0, the genetic and environmental path estimates represent the etiology of that phenotype in girls. The moderator values directly index sex differences in those parameter estimates. As seen in Table 3, genetic influences were equivalent across boys and girls (i.e., the genetic moderators values were small and non-significant). Environmental influences, by contrast, were more pronounced in boys as compared to girls, with significantly higher

shared environmental influences on AGG, and significantly (or nearly significantly) higher non-shared environmental influences on both AGG and RB.

The evidence of etiologic moderation by sex was murkier for maternal-reports of child AGG and RB. The no moderation model provided a better fit to the maternal-reported child AGG data, arguing against the presence of significant etiologic differences across sex. For maternal-reported RB, there was some evidence of etiologic moderation, but only for 2 of the 4 fit indices. We thus made use of the change in χ^2 as a tie-breaker, and selected the linear moderation model as the better-fitting model. Parameter estimates from the better-fitting models are presented in Table 3. For maternal reports of child RB, the rather tepid endorsement of the linear moderation model by our fit indices was underscored by the finding that none of the specific moderators were significantly larger than zero, although the E moderator was marginally significant. Such results suggest that non-shared environmental influences on maternal-reports of RB may be slightly larger in boys. Genetic and shared environmental influences, by contrast, were equivalent across boys and girls.

Are sex differences in the etiology of child AGG and RB enhanced by disadvantage?

We next evaluated whether sex differences in the etiology of child antisocial behavior were altered by the presence of disadvantage (i.e., does sex synergistically interact with disadvantage in the etiology of child AGG or RB?) via a series of two-moderator models. Model-fitting results are presented in Table 4. The synergistic moderators (model 2) could uniformly be fixed to zero, regardless of informant or type of antisocial behavior, without a significant decrement in model fit relative to model 1. Such findings indicate that the moderating effects of sex on the etiology of antisocial behavior are not accentuated (or dampened) in disadvantaged contexts relative to advantaged contexts. This absence of disadvantage-by-sex moderation is particularly noteworthy in light of the strong evidence of etiologic moderation by disadvantage for teacher- and mother-reports of RB and for teacher-reports of AGG, such that model 3 provided a worse fit to those data than did model 2.[1]

Confirmatory analyses

Results thus far indicate the presence of clear sex differences in the etiology of teacher-reported AGG and RB, but small-to-nonexistent sex differences in the etiology of maternal-reported AGG and RB. We thus examined whether a similar pattern emerged in previously published meta-analytic data (see Supplemental Table 1 for the specific studies; note that all studies analyzed were independent of the MSUTR). Results (presented in Table 5) again pointed to differential effects of sex across informants. Genetic influences on teacher reports of AGG appeared notably larger in girls as compared to boys (60% for girls versus 43% for boys), while shared environmental influences appeared larger in boys versus girls (36% for

[1]Although we used a more comprehensive measure of disadvantage in the current study, parameter estimates for teacher- and maternal-reports of child RB clearly replicate those Burt et al. (2016), which focused only on neighborhood disadvantage per se. In particular, C influences on child RB in the current study increased significantly with increasing levels of broader disadvantage (the estimated C moderation terms for disadvantage in model 2 were 0.59 (95% CI's 0.27, 0.94) for teacher-reports and 0.81 (95% CI's 0.19, 1.10) for maternal-reports). Genetic moderation terms were negatively signed and not statistically significant (−.06, and −.16, respectively). Teacher-reports of child AGG did not demonstrate significant shared environmental moderation (again replicating the findings of Burt, et al., 2016; the C moderator was estimated at −.12, ns). Such findings further increase confidence in the results of Burt et al., (2016).

boys and 20% for girls). Formal constraint analyses indicated that these differences were indeed statistically significant. By contrast, parameter estimates for maternal informant reports could be uniformly be constrained to be equal across sex without a decrement in fit.

These findings persisted when we further restricted the analyses to maternal and teacher informant-reports of AGG in the same samples (N=3 samples). Parameter estimates for maternal informant reports of AGG could still be constrained to be equal without a decrement in fit (AIC values: unconstrained model = 281.24; genetic constraint model = 279.90; shared environmental constraint model = 279.63), while parameter estimates for teacher informant reports of AGG could not (AIC values: unconstrained model = 281.24; genetic constraint model = 282.91; shared environmental constraint model = 282.20). In short, re-analyses of previously analyzed meta-analytic data (Burt, 2009a) indicate that there are in fact sex differences in the etiology of antisocial behavior, but they are restricted to teacher informant-reports of AGG.

DISCUSSION

Although sex differences in the etiology of youth antisocial behavior are an intuitively appealing hypothesis given the consistently higher prevalence of antisocial behavior in boys versus girls, prior meta-analytic work has uniformly come down against sex differences in etiology. The goal of the current study was to examine whether sex differences in etiology are instead confined to specific sub-populations or contexts, examining two key variables that might moderate the presence of sex differences: disadvantage and informant source. Results were not consistent with differential effects of sex on etiology in disadvantaged versus advantaged contexts, with null findings that persisted across both informants and operationalizations of antisocial behavior. The specific informant under study did matter, however. In both the MSUTR sample and the re-analyzed meta-analytic data from Burt (2009a), sex differences in etiology emerged when youth AGG was assessed using teacher informant-reports, but not when AGG was assessed using maternal informant-reports. Moreover, both sets of analyses pointed to a specific pattern of etiologic moderation by sex, such that shared environmental influences on AGG were stronger in boys than in girls. The meta-analytic data further suggested that genetic influences on teacher-reports of AGG were stronger in girls than in boys. For RB, MSUTR data indicated that non-shared environmental influences were stronger in boys than in girls.

Limitations

There are several limitations to the current study. First, we were unable to examine RB in the meta-analytic teacher data, as those data were not available. It thus remains unclear whether the informant effects detected for RB in the MSUTR will replicate in other studies. Second, we examined only a circumscribed set of potential moderators (i.e., disadvantage and specific informant-reports), even though there are there are several other possibilities (child self-reports, gender identity, race/ethnicity, even society – e.g., it could be that sex differences in etiology vary across societies with their level of gender equality). Our data were not well-suited to examinations of those other moderators, however (e.g., we were not sufficiently powered to also examine race/ethnicity, we did not have child self-reports of

AGG and RB, etc.). We hope that subsequent studies with data better suited to examine these possibilities will take up the call. Third, twins in the MSUTR sample were recruited specifically during middle childhood (i.e., ages 6–10 years-old). Although it is thus unclear whether these results will persist to adolescence, we would note that the meta-analytic data was composed primarily of adolescent samples (adolescent twin samples far outnumber those collected during childhood), tentatively suggesting that our results may persist to adolescence as well. Even so, it would be essential for future studies to examine this question in a longitudinal sample given the dramatic changes in frequency and manifestation of antisocial behavior from childhood to adolescence (antisocial behavior is far more common in adolescence than in childhood, but nearly all of this increase is attributable to increases in RB in particular (Burt, Donnellan, McGue, & Iacono, 2011; Tremblay, 2010)).

Finally, we examined only two, rather broad dimensions of antisocial behavior: overt aggression and covert rule-breaking. This selective focus is notable for two reasons. First, we did not consider other forms of antisocial behavior, including proactive aggression, reactive aggression, and social or relational aggression. Social aggression is especially interesting in this regard, as it varies only minimally across sex (but may be slightly more common in girls; Card, Stucky, Sawalani, & Little, 2008). On-going work in the MSUTR sample has also found no evidence of sex differences in the etiology of social aggression, for either maternal or teacher informants (Slawinski, Klump, & Burt, in progress). Although these findings require replication, they suggest that the current findings for teacher informant-reports do not extend to all antisocial phenotypes.

Second, the dimensions of AGG and RB can themselves be partitioned into four sub-dimensions (RB into status and property violations, AGG into physical aggression and oppositionality/defiance). And although differences among these sub-dimensions have emerged, available work has suggested that they may be more similar than they are different (Simonoff, Pickles, Meyer, Silberg, & Maes, 1998; Tremblay, 2010). For example, anger and emotional dysregulation appear to be core psychological features of both oppositional-defiant and physically aggressive behaviors (Burt & Donnellan, 2008; Tremblay, 2010). Moreover, oppositionality is often considered a precursor to more serious forms of aggressive conduct problems (Loeber, Burke, Lahey, Winters, & Zera, 2000). For their part, status and property violations evidence very similar developmental trajectories (Tremblay, 2010; van Lier et al., 2009) and etiological patterns (Simonoff et al., 1998). In short, the distinction between AGG and RB appears far more significant than do the respective distinctions within RB and AGG. Given these results, the current study focused on the broader dimensions of AGG and RB as defined above. Future studies should ensure that our results extend to other measures of AGG and RB as well.

Confidence Statement

Despite these limitations, we have a very high degree of confidence in our informant results. Because multiple informant-reports are necessarily collected within-person (i.e., we have more than one informant-report on each twin), our informant analyses were able to compare heritability estimates within the same set of children, a statistically and inferentially powerful approach. We then constructively replicated our informant results using previously

published meta-analytic data. The consistency of our results across these independent sources of data not only decreases the chance that we have identified a spurious association, but also serves the important function of illuminating discrepancies between our results and those of prior meta-analytic work.

Next, and although we believe they meaningfully contribute to the literature, we do have somewhat less confidence in our disadvantage results (we would describe our level of confidence as modest-to-moderate). On the plus side, our sample was unusually well-suited for the analyses in that it is both large and oversampled for disadvantage. Our null findings were also consistent across informants and operationalizations of antisocial behavior, further increasing confidence in our results. On the flip side, our disadvantage analyses necessarily took the form of a between-family comparison (i.e., families residing in advantaged contexts versus those residing in disadvantaged contexts). The between-family nature of the design required us to fit a dual moderator model (i.e., sex, disadvantage, sex-by-disadvantage), a far less powerful model than the single moderator model (Purcell, 2002) used for our (within-person) informant analyses. Between-family approaches are also typically less conclusive than within-family/within-person approaches, since families residing in one context differ in many ways from those residing in the other, rendering the specific moderator somewhat ambiguous (that said, it could be argued that this same ambiguity paradoxically augments confidence in our null disadvantage-by-sex findings).

Conclusions and implications

The results of the current study yield a number of important conclusions and implications. First, the current results were not consistent with the hypothesis that familial and contextual disadvantage enhance (or suppress) sex differences in the etiology of antisocial behavior, despite the fact that the current study was well-suited for detecting those effects. Our sample was both over-sampled for disadvantage and consisted of more than 1,000 twin pairs. We were thus sufficiently powered to detect etiologic differences in a two-moderator GxE model (see Purcell, 2002 for power analyses of simulated data). The current findings thus suggest that, although disadvantage appears to have potent effects on the etiology of youth antisocial behavior (Burt et al., 2016; Cleveland, 2003; Tuvblad et al., 2006), these effects do not vary across sex. Future studies should examine other contextual influences, including level of gender equality across societies, as contextual moderators of sex differences.

Second, the current findings provided additional evidence that, although rarely considered in the results of etiologic studies, informants appear to matter a great deal. By far the most common informant in studies of children, including in twin studies of children, is the children's mother. Teachers' perceptions of the children are also common, but are collected in fewer studies and on fewer children in those studies (as they are frequently harder to get). In the meta-analytic data re-analyzed here, for example, maternal informant-reports were available on 8,056 twin pairs (i.e., 16,112 twins), while teacher informant-reports were available on 3,018 twin pairs (i.e., 6,036 twins; or 37.4% the size of the maternal informant sample). Youth self- reports are rarer still in studies of children (this is not true in studies of adolescents), primarily due to concerns about reliability. As a consequence, meta-analytic results and conclusions are heavily weighted towards maternal perceptions of her children.

To the extent that findings differ across informants (De Los Reyes & Kazdin, 2005), this heavy weighting of maternal informant-reports could alter meta-analytic (and individual study) conclusions. Indeed, that appears to be the case here. Prior meta-analyses had concluded that there were no sex differences in the etiology of aggressive and non-aggressive antisocial behavior. When the Burt (2009a) data were disambiguated by informant, however, the conclusion changed: rather than no sex differences at all, the data indicate that there are no sex differences in etiology when examining maternal informant-reports of the child. When examining teacher informant-reports of the child, however, the meta-analytic data suggest that AGG is more environmental in origin in boys and more genetic in origin in girls – a conclusion that persisted to the independent MSUTR sample examined here.

How do we understand etiologic differences across maternal and teacher informant-reports?

There are several possible explanations for the robust differences in etiologic moderation by sex across informants observed here. One possibility relates to the attribution bias context model, whereby mothers and teachers are exposed to different slices of the child's behavior and thus develop different opinions/attributions regarding the same child (De Los Reyes & Kazdin, 2005). Mothers of school-aged children typically observe their children in less structured settings, and are privy to only some of what happens during the school day. Teachers, on the other hand, observe children in a more rigid classroom setting (indeed, meta-analytic work has reported mother-teacher correlations of only .27; Achenbach et al., 1987). In short, it may be the case the antisocial behavior on the part of any one child is simply easier to observe in scholastic settings because it is so disruptive to the functioning of the classroom. Alternately, it is possible that antisocial behavior is more gendered in its presentation at school (perhaps due to child-enforced social norms that constrain physical aggression in girls). In this case, we might expect, for example, aggression in school to be more heritable in girls, as only those genetically predisposed to aggression might be willing to act on those impulses in the face of social pressure not to do so. In short, sex differences in the origins of antisocial behavior may in fact be specific to school settings.

Differential validity of the two informant-reports is another possible explanation for our results, such that one informant may be providing more valid information on the child's antisocial behavior than is the other, with downstream consequences for estimates of etiology. We might assume that mothers are the better informant, given that they have typically known their child since s/he was born. Interestingly, however, there is at least some data suggesting that teachers may be the stronger of the two informants (Clark, Durbin, Donnellan, & Neppl, 2016; Clark, Durbin, Hicks, Iacono, & McGue, 2017), likely because they have a clearer, more developmentally refined sense of typical child behavior as a consequence of their exposure to many children of the same age. In this light, it is possible that the current findings for teachers reflect the 'true' etiology of child antisocial behavior. Critically, however, multiple studies have indicated that multi-informant assessments including a parent and the teacher (and the child when possible) are the most valid indicators of child behavior in terms of predicting outcome (Achenbach et al., 1987). Such findings argue against the possibility that etiological estimates obtained from teacher-reports are the

'right' ones and towards a more nuanced view of different etiologies in different settings as observed by different informants.

What do these findings mean for our understanding of the origins of youth antisocial behavior?

The current findings suggest that, at least when assessed from teachers' perspectives, antisocial behavior is more heritable in girls and more environmental in boys. There are several, non-mutually exclusive explanations for this pattern of results. The first is that boys are more responsive to environmental influences on antisocial behavior while at school. Consistent with this possibility, recent work has indicated that 'resistance to peer influence' or resistance to peer pressure can be understood as a trait that varies across individuals and increases with age (Sumter, Bokhorst, Steinberg, & Westenberg, 2009). It also varies consistently across sex, such that boys are less resistant (i.e., more susceptible) to peer influence than are girls (Monahan, Steinberg, & Cauffman, 2009; Sumter et al., 2009). The stronger shared environmental influences on antisocial behavior in boys may thus reflect this increased susceptibility to peer influences. Alternately, it may be that boys are exposed more often to the relevant environmental influences on antisocial behavior. Consistent with this possibility, Rowe and colleagues (1995) found that although the same variables predicted antisocial behavior in boys and girls, boys were exposed to those influences more frequently.

The final explanation focuses more heavily on the larger genetic influences in girls. Early work by Cloninger and colleagues indicated that, although the genetic and environmental factors influencing antisocial behavior do not differ across the sexes, "*female probands have more ill relatives than do male probands*" (pg. 946, Cloninger et al., 1978). These findings have been interpreted in the context of a liability threshold model, such that females must be more severely affected to manifest the trait (i.e., because the threshold in liability for antisocial behavior is higher in females than in males). Put another way, females need to a higher genetic loading to express the trait. Subsequent work has at least partially supported this possibility (Bohman, 1996; Monuteaux et al., 2004). Unfortunately, the current analyses cannot directly disambiguate between resistance to peer influence, increased exposure, and/or threshold differences across sex. Future work should seek to do just this, directly contrasting the various explanations for etiologic sex differences in school settings.

What do these findings mean for meta-analytic work in the future?

The final broad conclusion from the current study relates to the utility of meta-analyses for crystallizing our understanding of the literature. By synthesizing the results of all available studies, meta-analyses serve an incredibly useful function in research. The current results provide an obvious but important caveat to this general conclusion: results (and thus, conclusions) are dependent on the analyses undertaken. Prior work contrasting the respective etiologies of AGG and RB (Burt 2009a) examined each potential moderator of etiology separately, and did not consider the possibility of joint moderation. In large part, this reflected the goals of that meta-analysis, which was specifically designed to clarify whether AGG and RB were equally heritable, and whether the observed differences in heritability persisted across various moderators. Sex differences themselves were not a direct focus of the project. Even so, the seemingly clear absence of differences in etiology across sex in

Burt (2009a) has since been cited to support the notion that the etiologies of AGG and RB do not vary across sex, a problematic decision in retrospect given that available evidence of sex differences in teacher informant-reports of AGG was embedded in those data. In this light, the current results provide a cautionary tale, and suggest, among other things, that future meta-analyses of twin and adoption study results should consider joint moderation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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The meta-analytic data reexamined here were previously published in Burt (2009a). The etiologic moderation of antisocial behavior by disadvantage in these data was previously published in Burt et al., (2016).

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General Scientific Summary

Our study suggests that genetic influences on youth aggression are stronger in girls, while environmental influences are stronger in boys. However, this holds true only in scholastic contexts. Outside of school, aggression is equally heritable in boys and girls.

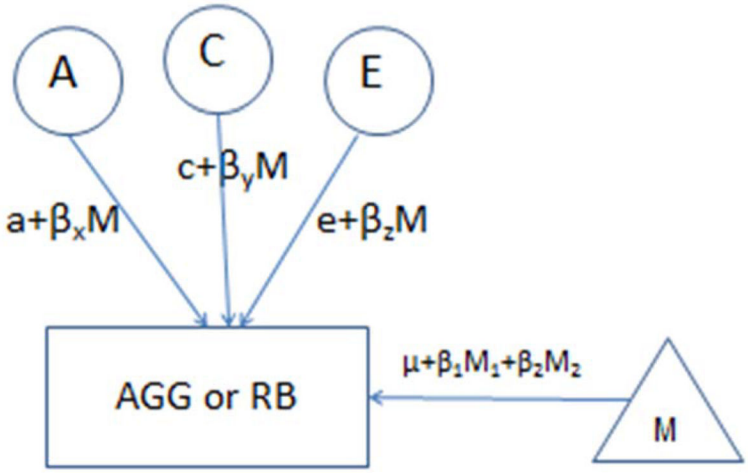

Figure 1. Path diagram of the full linear moderator model

Note. A, C, and E represent genetic, shared environmental, and non-shared environmental influences, respectively. Interactions with the moderator (i.e., M; the child's biological sex) are added to these genetic and environmental influences, and are estimated separately for each component of variance (i.e., $\beta_X M$, $\beta_Y M$, and $\beta_Z M$ for a, c, and e paths, respectively). For ease of presentation, the co-twin variables and paths are omitted here, though they are estimated in the model.

Table 1

Descriptive Statistics.

	Females				Males				Cohen's *d* effect size
	Mean (SD)	**n**	**Min**	**Max**	**Mean (SD)**	**n**	**Min**	**Max**	
Teacher-reported AGG	1.24 (3.48)	754	0	39	2.20 (4.26)	797	0	36	.25**
Teacher-reported RB	0.47 (1.23)	754	0	12	0.82 (1.64)	797	0	13	.24**
Mother-reported AGG	4.02 (4.43)	992	0	28	5.02 (5.30)	1042	0	31	.20**
Mother-reported RB	1.19 (1.57)	992	0	14	1.82 (2.15)	1042	0	14	.34**

Note. AGG and RB represent Aggressive Behavior and Rule-Breaking Behavior, respectively. Raw AGG and RB scores are reported here. AGG could conceivably range from 0 to 40 for teacher-report and 0 to 36 for mother-report. RB could conceivably range from 0 to 24 for teacher-report and 0 to 34 for mother-report. Means were compared across boys and girls using independent samples t-tests,

** p<.01.

Table 2

Fit Indices for GxE models examining child sex as an etiological moderator of child antisocial behavior, separately by informant

Model	-2lnL	df	AIC	BIC	SABIC	DIC
Teacher-reported AGG						
Linear ACE moderation	**4141.35**	**1539**	**1063.35**	**−3106.08**	**−662.41**	**−1691.84**
No moderation	4188.25	1542	1104.25	−3092.73	−644.29	−1675.72
Teacher-reported RB						
Linear ACE moderation	**4162.45**	**1539**	**1084.45**	**−3095.54**	**−651.86**	**−1681.29**
No moderation	4208.76	1542	1124.76	−3082.47	−634.04	−1665.47
Mother-reported AGG						
Linear ACE moderation	5432.86	2022	1388.86	−4285.35	−1074.32	−2427.26
No moderation	**5439.08**	**2025**	**1389.08**	**−4292.63**	**−1076.84**	**−2431.78**
Mother-reported RB *						
Linear ACE moderation	5273.58	2022	1231.58	−4363.99	−1152.96	−2505.89
No moderation	5290.84	2025	1240.84	−4366.75	−1150.95	−2505.90

Note. The best fitting model for a given set of analyses is highlighted in bond font, and is indicated by the lowest AIC (Akaike's Information Criterion), BIC (Bayesian Information Criterion), SABIC (sample size adjusted Bayesian Information Criterion), and DIC (Deviance Information Criterion) values for at least 3 of the 4 fit indices.

* for mother-reported RB, neither model provided a clear improvement in fit. In this case, we made use of the significant change in χ^2 (computed by comparing the -2lnL and change in df) as a tie-breaker. We thus conclude that the linear ACE moderation model provides a marginally better fit to the data.

Table 3

Unstandardized path and moderator estimates for best-fitting ACE moderation models.

PATHS			LINEAR MODERATORS		
a	**c**	**e**	**A_1**	**C_1**	**E_1**
Teacher-reported AGG					
0.59* (0.36, 0.71)	−.28 (−0.51, 0.06)	**0.56*** (0.50, 0.63)	0.16 (−0.19, 0.52)	**0.73*** (0.37, 0.96)	0.09~ (−0.004, 0.20)
Teacher-reported RB					
0.66* (0.44, 0.78)	−0.27 (−0.53, 0.53)	**0.52*** (0.46, 0.59)	0.16 (−0.20, 0.41)	0.41 (−0.74, 0.74)	**0.19*** (0.09, 0.30)
Mother-reported AGG					
0.76* (0.65, 0.84)	0.21 (−0.43, 0.43)	**0.60*** (0.56, 0.64)	---	---	---
Mother-reported RB					
0.65* (0.47, 0.79)	**0.44*** (0.13, 0.61)	**0.50*** (0.45, 0.55)	0.14 (−0.13, 0.32)	-.06 (−0.39, 0.36)	0.06~ (-.01, 0.14)

Note. A, C, and E (upper and lower case) respectively represent genetic, shared, and non-shared environmental parameters on rule-breaking (RB). Bold font and an asterisk indicate that the estimate is significant at p<.05. Primary analytic results are presented in the top half of the table. Because girls were dummy coded as 0, the genetic and environmental contributions to child antisocial behavior reports for girls can be obtained by squaring the path estimates (i.e., a, c, and e). For boys, linear moderators (i.e., A_1, C_1, E_1) were added to the paths using the following equation: $Unstandardized\ Variance_{Total} = (a + A_1(sex))^2 + (c + C_1(sex))^2 + (e + E_1(sex))^2$. The variance component estimates calculated this way are presented in the text.

Table 4

Fit Indices for disadvantage X sex analyses

Model	-2lnL	df	AIC	BIC	SABIC	DIC
Disadvantage and child sex as etiologic moderators of child antisocial behavior						
Teacher-reported AGG						
1. Full model	4114.94	1535	1044.94	−3105.83	−668.51	−1695.26
2. No synergistic moderation	**4115.71**	**1538**	**1039.71**	**−3115.54**	**−673.46**	**−1702.21**
3. No moderation by disadvantage	4137.83	1541	1055.83	−3114.57	−667.72	−1698.49
4. No moderation	4186.49	1544	1098.49	−3100.33	−648.72	−1681.49
Teacher-reported RB						
1. Full model	4099.12	1535	1029.12	−3113.75	−676.42	−1703.17
2. No synergistic moderation	**4101.55**	**1538**	**1025.55**	**−3122.62**	**−680.54**	**−1709.29**
3. No moderation by disadvantage	4154.56	1541	1072.56	−3106.21	−659.36	−1690.12
4. No moderation	4204.03	1544	1116.03	−3091.56	−639.95	−1672.72
Mother-reported AGG						
1. Full model	5415.35	2018	1379.35	−4280.25	−1075.57	−2425.83
2. No synergistic moderation	5417.28	2021	1375.28	−4289.68	−1080.24	−2432.50
3. No moderation by disadvantage	5422.95	2024	1374.95	−4297.23	−1083.02	−2437.29
4. No moderation	**5433.76**	**2027**	**1379.76**	**−4302.21**	**−1083.25**	**−2439.53**
Mother-reported RB						
1. Full model	5234.70	2018	1198.70	−4370.57	−1165.90	−2516.16
2. No synergistic moderation	**5241.15**	**2021**	**1199.15**	**−4377.74**	**−1168.30**	**−2520.57**
3. No moderation by disadvantage	5267.42	2024	1219.42	−4374.99	−1160.79	−2515.06
4. No moderation	5282.97	2027	1228.97	−4377.61	−1158.64	−2514.92

Note. The best fitting model for a given set of analyses is highlighted in bond font, and is indicated by the lowest AIC (Akaike's Information Criterion), BIC (Bayesian Information Criterion), SABIC (sample size adjusted Bayesian Information Criterion), and DIC (Deviance Information Criterion) values for at least 3 of the 4 fit indices.

Table 5

Meta-analytic heritability estimates for child antisocial behavior, separately across sex

	Girls			Boys			AIC for constraint (or no sex differences) model		
	A	C	E	A	C	E	No A differences	No C differences	No E differences
Teacher-reported AGG	**.60** (.47, .74)	**.20** (.07, .33)	**.20** (.18, .22)	**.43** (.31, .55)	**.36** (.24, .48)	**.21** (.19, .24)	438.44	437.72	435.52
Mother-reported AGG	**.57** (.50, .64)	**.19** (.12, .26)	**.24** (.23, .26)	**.54** (.48, .61)	**.20** (.14, .27)	**.25** (.24, .27)	435.06	434.86	435.90
Mother-reported RB	**.42** (.32, .54)	**.32** (.21, .43)	**.25** (.23, .28)	**.41** (.32, .51)	**.34** (.25, .44)	**.24** (.22, .27)	434.78	434.84	435.22

Note. A, C, and E represent genetic, shared environmental, and non-shared environmental influences, respectively. AGG and RB represent aggressive and non-aggressive antisocial behavior, respectively. All ACE parameter estimates were significantly larger than zero (i.e., 95% CIs do not overlap with .00). For maternal-reports of AGG, we analyzed 10 independent samples containing 4,215 male pairs and 3,844 female pairs. For maternal-reports of RB, we analyzed 6 independent samples containing 1,690 male pairs and 1,328 female pairs. For teacher-reports of AGG, we analyzed 3 independent samples containing 994 male pairs and 1,049 female pairs. There were no teacher-reports of RB presented separately by sex in the meta-analysis. The AIC value for the fully unconstrained or 'sex differences' model was **436.76**. We then constrained each parameter across sex and evaluated the change in model fit. AIC values for each constraint model are presented on the right half of the table. Gray shading indicates parameter estimate differs significantly across sex as indicated by a poorer fitting AIC.