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Etiology and Measurement of Relational Aggression: A Multi-Informant Behavior Genetic Investigation

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Abstract

Although the study of relational aggression is gaining attention in the literature, little is known about the underlying causes of this behavior and the relative validity of various informants. These issues were addressed in a sample of 1981 6- to 18-year-old twin pairs (36% female, 34% male, 30% opposite-sex). Relational aggression was assessed via maternal- and self-report using a structured interview. Univariate models estimated genetic and environmental influences by informant and examined evidence for gender differences. A psychometric model combined data from both informants to estimate etiologic influences that were both common to the informants and informant-specific. In both sexes, the latent variable reflecting the mother's and child's shared perception of the child's relational aggression was substantially influenced by both additive genetic (63%) and shared environmental (37%) influences, although this latent variable accounted for much greater variance in maternal report (66%) than it did in youth report (9%). In addition, informant specific additive genetic and shared environmental influences were found only for youth report, with all remaining variance in mother's report attributed to nonshared environmental influences. Results are discussed in the context of measuring relational aggression and the importance of multiple informants.

Layperson conceptualizations of childhood aggression often include images of a callous young child labeled the "class bully", who picks physical fights with others and bullies his classmates during recess. Recent attention to "mean girls" has highlighted another sort of common bully - this time, one who uses her social status and the relational sensitivity of others to aggress against her victims in distressing, but non-physical ways. Relational aggression (RAgg) is a psychological construct that has been studied for several decades, although different terms have at times been used by different researchers (Archer & Coyne, 2005; Bjorkqvist, Lagerspetz, & Kaukiainen, 1992; Cairns, Cairns, Neckerman, Ferguson, & Gariépy, 1989; Crick & Grotpeter, 1995; Feshbach, 1969; Galen & Underwood, 1997; Lagerspetz, Björkqvist, & Peltonen, 1988). RAgg has been defined as behaviors, typically covert, that are intended to hurt others via damaging their relationships or social standing (Archer & Coyne, 2005; Crick & Grotpeter, 1995). Other terms that have been commonly used to refer to similar behaviors include social aggression (e.g., Galen & Underwood, 1997) and indirect aggression (e.g., Bjorkqvist et al., 1992; Feshbach, 1969). We use RAgg in the present manuscript because it is more specifically descriptive of indirect aggressive

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acts while also acknowledging that the behavioral content examined by researchers adhering to these three labels overlaps substantially (Archer & Coyne, 2005).

The construct of RAgg was developed partly in response to concerns that existing conceptualizations of aggression primarily reflected physically aggressive behaviors that are more common in boys (e.g., Bjorkqvist, 2001; Crick, 1996). Indeed, some researchers have theorized that RAgg may be a female-typical form of aggression because the costs of physical aggression are much higher for females than for males (Archer & Coyne, 2005; Campbell, 1999). Nonetheless, it appears that sex differences in overall levels of RAgg have been exaggerated, as boys frequently engage in RAgg as well (Archer & Coyne, 2005). A recent meta-analysis found evidence for small but significant gender differences in RAgg with presence of an informant effect such that parent and teacher reports were more likely to yield gender differences favoring higher levels of RAgg in girls than were self-reports, which yielded slightly higher levels in boys (Card, Stucky, Sawalani, & Little, 2008).

A pattern of behavior only can be considered “abnormal” if it is maladaptive—associated with distress and/or functional impairment (Bandura, 1969; Wakefield, 1992; Ullmann & Krasner, 1975). Much remains to be learned, but there is growing evidence that RAgg has harmful psychological effects on victims (Crick et al., 2001; Marini, Dane, Bosacki, & YLC-CURA, 2006; Prinstein, Boergers, & Vernberg, 2001). It is not clear that RAgg is associated with distress or functional impairment in the aggressors, however. On the one hand, there is evidence that RAgg predicts later social maladjustment in the aggressor (Card et al., 2008; Crick, 1996; Crick et al., 2001; Tomada & Schneider, 1997; Vaillancourt, Brendgen, Boivin, & Tremblay, 2003) and increased risk for later RAgg victimization (Ostrov, 2008). On the other hand, there is evidence that RAgg is not associated with distress or impairment in aggressors after their other symptoms of psychopathology are controlled (Keenan, Coyne, & Lahey, 2008). Although definitions of abnormal behavior in psychology tend to include behaviors that only harm others (Bandura, 1969; Ullmann & Krasner, 1975), recent editions of the DSM have focused only on the distress or impairment of the person engaging in the behaviors. Thus, RAgg is almost certainly maladaptive by most definitions, but it is not yet clear that it can be viewed as maladaptive according to definitions that only consider distress or impairment in the aggressor.

A second unresolved question about RAgg as a potential form of psychopathology is whether it is distinct from other forms of psychopathology. Research has found high correlations between RAgg and overt/physical aggression (r=.76; Card et al., 2008), similar in magnitude to subtypes within conduct disorder itself (Tackett, Krueger, Sawyer & Graetz, 2005). In addition, studies examining the covariation among specific externalizing behaviors including RAgg behaviors have found RAgg to hang tightly with other externalizing behaviors in both adults (Krueger, Markon, Patrick, Benning, & Kramer, 2007) and children (Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007). When the RAgg items measured in the present sample were included in an exploratory factor analysis of a broad range of psychopathology items in another large representative sample of children and adolescents, some parent-rated RAgg items loaded on a factor with symptoms of oppositional defiant disorder, some loaded on a factor with conduct disorder symptoms, and some did not load on any factor (Lahey, Applegate, Waldman, Loft, Hankin & Rick, 2004). Thus, although there is good reason to believe that RAgg is harmful, at least to its victims, the question of whether it should be viewed as a distinct form of psychopathology or as a symptom of forms of psychopathology already recognized in current taxonomies is unresolved.

As research on the topic of RAgg has accumulated, questions have arisen regarding its measurement (e.g., which informants are most valid), the underlying etiology of such behaviors, and the similarities and differences between causal factors for RAgg and other

types of childhood antisocial behavior. In this paper, we begin by discussing some of these measurement issues, reviewing genetically-informative studies of childhood antisocial behavior, and describing the genetically-informative psychometric model, which permits the investigation of measurement and etiology together in an overarching analytic framework.

The Measurement of RAgg

Various approaches to measuring the construct of RAgg and the related dimension of indirect aggression were recently reviewed by Archer and Coyne (2005). In this review, the authors noted the use of observational methods for children from toddlerhood to middle childhood (e.g., Galen & Underwood, 1997). A common method for measuring both indirect and relational aggression is via peer ratings, by having children rate multiple peers on Likert-type scales (e.g., Bjorkqvist et al., 1992) or using peer nomination methods (e.g., Crick & Grotpeter, 1995). Peer ratings appear to be the most popular method of assessment in the study of RAgg and perhaps for that reason are often relied on as a basis of comparison for information from other sources, despite a general lack of information regarding the relative utility of different sources for RAgg characterizations.

It has been suggested that teachers, parents, and self-ratings are less valid in comparison to peer ratings, although a number of studies of both indirect and relational aggression have used information obtained from teachers and self-report (Archer & Coyne, 2005). Studies have also used parental reports (Vaillancourt et al., 2003), although this is frequently described as a limitation (Archer & Coyne, 2005). Consistent with much of the literature on cross-informant correlations for childhood behavior, Crick et al. (1999) showed that self-reports of RAgg are poorly correlated with information obtained via other methods, leading Archer and Coyne (2005) to question the validity of self-report information for this construct. Inter-informant agreement on RAgg additionally tends to be low between teacher-report, peer-report, and observational ratings (McNeilly-Choque, Hart, Robinson, Nelson, & Olsen, 1996). From a measurement perspective, however, the use of informants providing non-overlapping information may be highly desirable, yet still consistent with low cross-informant correlations (Kraemer et al., 2003). In a recent investigation of parent- and self-report for RAgg, evidence suggested good test-retest and inter-informant reliability of parent- and youth-reported RAgg and predictive validity for reports of impairment across informants (Keenan et al., 2008). These questions and concerns over the validity of various approaches to measurement must be directly addressed in order to better identify potential sources of meaningful information about RAgg. This study specifically focuses on this issue in mother and self-reports, both of which have been highlighted as potentially questionable sources of RAgg information.

Behavior genetic studies of antisocial behavior in childhood and adolescence

An extensive body of literature has accumulated investigating genetic and environmental influences on antisocial behavior in childhood. A recent review highlighted substantial variation in results across behavior genetic studies on antisocial behavior in adults and children (Rhee & Waldman, 2002). Numerous explanations have been proposed for this variability in results, including heterogeneity of constructs within the domain of childhood antisocial behavior (e.g., Jacobson, Baker, & Raine, 2007; Tackett, Krueger, Iacono, & McGue, 2005). In general, evidence for significant genetic influences is commonly reported across informants and type of antisocial behavior (Burt, 2009; Rhee & Waldman, 2002). Some studies have shown greater additive genetic influence (A) on aggressive antisocial behaviors, while rule-breaking antisocial behaviors often show smaller, but still significant, heritability in addition to significant shared environmental influences (C; e.g., Edelbrock,

Rende, Plomin, & Thompson, 1995; Eley, Lichtenstein, & Moffitt, 2003; Tackett et al., 2005) and both types show substantial non-shared environmental influences (E). This recent work has emphasized the importance of conducting behavior genetic investigations with clearly defined behavioral constructs, as heterogeneous and/or poorly-defined constructs may result in inconsistent results across studies.

One research team recently provided the first genetically-informative investigation of RAgg (Brendgen et al., 2005). In a sample of 6-year-old twins (*N*=234 pairs), RAgg was assessed via teacher and peer-ratings. Specifically, teachers completed three items from the Preschool Social Behavior Scale (PSBS-T; Crick et al., 1997) while peers nominations were gathered for two behavioral criteria related to RAgg. The researchers interpreted results of both teacher and peer data separately in terms of a constrained ACE model, wherein A and C were constrained to be equal. Specifically, these results apportioned 20% of the variability in RAgg to each of the additive genetic and shared environmental components, with the remaining 60% attributable to non-shared environmental influences. The fit of the constrained ACE model was virtually identical to the fit of the CE model for both informants. A model with both informants was not tested.

This work provided an important first step toward a greater understanding of etiologic factors related to RAgg but was limited by a small sample size for a twin study, a focus on a younger age group which may not be evidencing substantial variability in RAgg, and limited measurement of RAgg to only 2–3 items per informant. Further, the results of the study could not converge on either an ACE or a CE model as best fitting the data, calling into question the role of additive genetic influences on RAgg. A recent study reported results from the same sample of twins (*N*=203) followed up at age 7 (Brendgen et al., 2008). In these analyses, teacher and peer-report (on the same measures as described above) were averaged to produce the measure of RAgg. For this teacher-peer measurement of RAgg, an AE model was determined to best fit the data. In summary, these two investigations of RAgg in early childhood with the same twin sample show a completely different pattern of results only a year later, posing more questions than answers regarding the etiology of RAgg. Further, to date the role of informants has not been explicitly integrated into genetically-informative analyses of RAgg.

Indeed, the use of different informants is a plausible explanation for the variability of results that have emerged from genetically-informative studies of childhood and adolescent ASB (Baker et al., 2007). Different informants of childhood and adolescent problem behaviors tend to provide relatively distinct reports about a given child's manifestation of specific behaviors; low cross-informant correlations have become standard in such research with children (Achenbach, McCounaughy & Howell, 1987; Kraemer et al., 2003). In particular, cross-informant correlations tend to be lowest for parent-child report as compared to parent-parent or parent-teacher reports (Achenbach, McConaughy, & Howell, 1987; Baker et al., 2007). Two meta-analyses have noted that other-report tends to result in higher evidence for familial influences (i.e., A and C) than self-report, although this could potentially be confounded with age as self-report is more likely to be obtained at older ages (Burt, 2009; Rhee & Waldman, 2002). In order to explicitly investigate this issue, researchers have recently turned to rigorous multi-informant behavior genetic analyses that provide the opportunity to disentangle rater-specific variance from shared variance across raters (e.g., Arseneault et al., 2003; Baker et al, 2007; Simonoff et al., 1995), which is a primary goal of the present study.

Researchers investigating any domain of childhood problem behaviors are faced with the task of deciding which informants to use and, if multiple reports are available, how best to combine them (Kraemer et al., 2003). It is also reasonable to assume that the answer to these

questions may differ for different domains of psychopathology. For example, research has shown that parent-child agreement tends to be lower for internalizing than externalizing behaviors, as externalizing problems are more likely to be directly observed by outside informants (Grills & Ollendick, 2002). The validity of self-reports from children has long been questioned for reasons such as the limitations on responding due to various stages of cognitive development or the influence of social desirability (e.g., Baker et al., 2007; Burt, McGue, Krueger, & Iacono, 2005). However, in applied clinical settings the data most readily available are often child- and parent-report. Taken together, the overall inconsistency of information obtained from various informants highlights the need to include multiple informants in future research on RAgg, but also to systematically examine common and specific contributions from such informants. In particular, given pragmatic limitations of applied assessment, the validity of information regularly obtained by clinicians from parents and children must be better understood.

The psychometric model

The psychometric model is a common pathways model that includes information from multiple raters (see Figure 1). The common pathways model allows estimation of genetic and environmental influences on multiple measures of a phenotype via their influence on an underlying shared latent factor. This model allows estimation of additive genetic (A), nonadditive genetic (D), shared environmental (C), and nonshared environmental (E) influences acting on the variance representing shared mother and child perceptions of the child's RAgg. In addition, this model estimates additive genetic, nonadditive genetic, shared environmental, and nonshared environmental influences that are specific to each informant. In other words, the psychometric model allows us to ask whether there are additional specific genetic or environmental influences contributing to the child's self-report and the mother's report of RAgg over and above those genetic or environmental influences contributing to the shared perceptions of the child's RAgg. Notably, the common RAgg construct is unaffected by informant-specific bias and error, which will have implications for nonshared environmental influences on this component. Specifically, the nonshared environmental pathway on the common RAgg factor will not contain measurement error but will only reflect environmental influences that act to make the twins different from one another, while the nonshared environmental pathways on the mother- and child-specific RAgg factors will include measurement error as well as such environmental influences. In addition, it is important also to note that systematic rater effects and shared environmental effects on the mother-specific RAgg construct cannot be differentiated in this model. Thus, a finding of significant mother-specific shared environmental influences could reflect environmental influences that make the twins similar in levels of RAgg, maternal bias, or both. Individual rater bias in self-reports, however, will be captured as measurement error and contribute to the magnitude of nonshared environmental influences on twin report.

The psychometric model partitions the variance in a phenotype of interest within the context of classic twin study methodology. Twin study designs rely on the comparison of monozygotic (MZ) versus dizygotic (DZ) twin similarity on a construct of interest. Specifically, because MZ twins share 100% of their genes identical by descent, whereas DZ twins share 50% of their genes on average, differences in MZ and DZ twin similarity can result in inferences regarding additive genetic and shared and nonshared environmental influences on the construct.

The psychometric model offers a powerful opportunity to shed light on the ongoing controversy surrounding cross-informant discrepancy in reports of childhood behavior by examining whether these differences are meaningful and systematic. Such evidence has supported the use of multiple informants for various forms of antisocial behavior by demonstrating better fit of the psychometric model over a more restrictive rater bias model,

which assumes that differences between informants is due entirely to rater bias (Arseneault et al., 2003; Baker et al., 2007; Bartels et al., 2003a; Bartels et al., 2004). Strong convergence is seen across studies for substantial genetic influences (sometimes accounting for 80–90% of the variance) on shared reports of externalizing behaviors in early childhood (5-year-old twins; Arseneault et al., 2003), middle childhood (aged 9–12 years; Baker et al., 2007; Bartels et al., 2003a; Bartels et al., 2003b; Bartels et al., 2004) and a study utilizing a broader age range (8–16 year-old twins; Simonoff et al., 1995). In addition, these studies tend to find no significant shared environmental influences on the latent phenotype reflecting information shared by multiple informants (Arseneault et al., 2003; Baker et al., 2007; Simonoff et al., 1995). While a measure of RAgg was included in the recent study by Baker et al. (2007), it was not investigated as an independent construct. Rather, the researchers extracted an “externalizing” factor from a variety of measures of antisocial behavior to use in their behavior genetic analyses.

Taken together, these studies provide convergent support for use of a psychometric model over a rater bias model on childhood externalizing data collected from multiple informants and demonstrate how the psychometric model can provide support for the validity of the use of multiple informants for childhood externalizing behavior. In general, previous studies applying the psychometric model to childhood antisocial behavior have found strong evidence for substantial heritability on shared perceptions among informants, with no influences from shared environmental factors.

The Present Study

We had two related goals in the present study. First, using a large, population-based study of twins, we conducted the first comprehensive behavior genetic investigation of RAgg across middle childhood and adolescence. Given the sparse information on RAgg-specific etiologic influences, we attempted to provide comprehensive results of univariate model-fitting to advance our understanding of the structure, causes and measurement of RAgg. Past research has found moderate to high correlations between RAgg and physical aggression (Crick, 1996; Vaillancourt, Brendgen, Boivin, & Tremblay, 2003), which led us to hypothesize significant genetic influences on RAgg. We further expected to find small but significant shared environmental influences on RAgg, consistent with findings for other types of antisocial behavior. Second, we capitalized on the multi-informant design in the sample to address questions regarding both etiology and measurement by fitting a psychometric model to these data. Specifically, we estimated both common genetic and environmental influences on the shared variance in mother and youth report, as well as specific genetic and environmental influences unique to each informant. Using this model, we hoped to answer some persisting questions regarding the validity of mother and youth report in RAgg studies.

Method

Participants

Participants in the Tennessee Twin Study (TTS) comprised a representative sample of twins between the ages of 6 and 18 who were born in Tennessee. Participants lived in one of Tennessee’s five Metropolitan Statistical Areas (MSA; Bristol, Chattanooga, Knoxville, Memphis, and Nashville) during the time of the study. The Tennessee Department of Health provided addresses for eligible families and an age- and geographically-stratified random sample of these addresses was extracted. Informed consent was obtained from caregivers and oral assent from twins old enough to be interviewed (≥9 years of age). Interviewed caregivers were primarily biological mothers (90.8%, 7.5% biological fathers, 0.5% stepmothers, and 1.2% grandmothers), thus the parent informants for the remaining analyses

were limited to reports from biological mothers only. For more information about the TTS, see Lahey et al. (2008).

A total of 1981 twin pairs were included in the present analyses with roughly equivalent numbers of female twin pairs (MZF *N* = 376, 19.0% of overall sample; DZF *N* = 332, 16.8% of overall sample), male twin pairs (MZM *N* = 356, 18.0% of overall sample; DZM *N* = 328, 16.6% of overall sample), and opposite-sex dizygotic twin pairs (DZOS*N* = 589, 29.7% of overall sample). Information on RAgg was obtained from 1802 biological mothers of twin pairs and 1583 twin pairs via self-report (self-report was only obtained for twins ages 9 and older). Twin pairs were distributed approximately equally across age. Ethnic background of the twin pairs was designated by the caregiver (71.4% Non-Hispanic white, 23.3% African American, 1.8% Hispanic, and 3.5% multiracial or other ethnic groups). Missing data were handled using a full information maximum likelihood (FIML) approach as implemented in Mplus. Zygosity of the twins was primarily determined through a questionnaire about physical similarities between the twins (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). Ambiguous cases were resolved with 12 polymorphic DNA markers obtained from cheek swabs. Twins were randomly designated Twin 1 or Twin 2 following data collection.

Measures

Child and Adolescent Psychopathology Scale (CAPS)—RAgg was assessed via the CAPS (Lahey et al., 2004), a structured interview assessing DSM-IV symptoms of common childhood disorders and additional relevant behavioral domains. Participants were asked about a series of behaviors and indicated how often the behavior occurred on a 4 point response scale (not at all, just a little, pretty much, very much). Items on the CAPS were randomized and counterbalanced in order of administration with a randomly-selected half of the participants completing the CAPS in reverse order. Seven items (presented in Table 1) were written to measure RAgg based on conceptualizations of the construct in the extant literature.

Results

Operationalizing the RAgg construct in the present study

In response to questions about measuring RAgg, we first investigated the structure of the 7 items for each informant to determine whether they could be conceptualized as a unitary construct. Confirmatory factor analyses were conducted using Mplus (Muthen & Muthen, 1998–2006) with a weighted least squares estimator (WLSMV; Flora & Curran, 2004), treating items as ordinal for both mother (*N*=3674 individual mother reports) and twin (*N*=3158) report. Data treated as ordinal in Mplus will utilize polychoric correlations which are more statistically appropriate when analyzing individual items measured on a Likert scale. In addition, data were treated as clustered at the family level to account for dependence of the data within families by using a robust standard error estimator. Results of the CFAs provided strong support for a one-factor structure with all items showing substantial loadings (all >.40) on the factor for both mother (CFI=.97, RMSEA=.03) and youth (CFI=.99, RMSEA=.02) report. See Table 1 for results of the mother and twin CFAs. These factor loading patterns provide support for all items representing strong markers of the underlying RAgg factor. Scale reliabilities were computed for both mother (α=.66) and youth (α=.62) report.

Based on these results, we created indicators of RAgg by summing the scores on all 7 items for each informant (resulting in 2 variables for each twin: RAgg-mother, RAgg-twin). Specifically, as the tests for measurement invariance did not show evidence for improved fit

with constrained factor loadings across groups, we utilized a unit-weighting approach to constructing the scales which has been shown to result in scale scores that are more stable than regression-based factor scores (e.g., Wainer, 1976). Descriptive statistics and correlations are presented in Table 2 across gender, zygosity, informant, and age. The correlations between mother- and self-report are consistent with correlation estimates for other types of child behavioral problems (e.g., Achenbach et al., 1987). The correlations between RAgg and age reveal no clear systematic pattern of covariation, such that RAgg scores do not appear to systematically vary across age in this sample. Thus, age was not specifically investigated as a covariate but rather was controlled for in the genetically-informative analyses that follow.

Univariate model fitting

After constructing the RAgg scores, we fit univariate models separately by informant to examine genetic and environmental influences on the RAgg construct. The full ACE model estimates the amount of variance in the RAgg construct attributable to additive genetic influences (A), shared environmental influences (C), and non-shared environmental influences (E). We also fit a series of reduced models containing additive genetic and non-shared environmental influences (AE), shared environmental and non-shared environmental influences (CE), and non-shared environmental influences (E) models for each informant group. The correlations presented in Table 2 support the likelihood of both additive genetic influences, such that the MZ correlations are always larger than the DZ correlations, as well as shared environmental influences, such that the DZ correlations are also larger than half the MZ correlations. These correlations also suggest different patterns across gender and informant, particularly when comparing the mother report for female and male twins. As this is the first large-scale twin study of RAgg, we took a comprehensive approach to univariate model-fitting in order to present broad descriptive information regarding the genetic and environmental influences on RAgg. All model fitting was conducted with Mplus.

To investigate potential sex differences, we fit a common effects model which allows examinations of differences in model fit when all biometric parameters are free to vary across sex versus when all are held constant across sex for an ACE, AE, CE, and E model (Prescott, 2004; Saudino, Carter, Purper-Ouakil & Gorwood, 2008). In the full ACE model, the best model fit for mother report was found when parameter estimates were allowed to vary across sex (χ^2=44.69, *df*=17) while the best fit for youth report was a fully constrained model with all biometric parameters held constant across sex (χ^2=50.52, *df*=20). The overall best-fitting model for youth report was a fully-constrained AE model (χ^2=52.02, *df*=21). Comprehensive results of these models are presented in Table 3. We additionally fit an ADE model (where D represents dominance genetic variance) for each informant group. The D parameter was not significant in any of the models, consistent with the pattern of twin correlations presented in Table 2, so these results are not presented here.

Multivariate model fitting: The psychometric model

We next fit the psychometric model to mother and youth reports simultaneously in all twin pairs to estimate genetic and environmental influences on the mother's and child's shared perception of the child's RAgg. The overall model estimated A, C, and E contributions for the shared variance among mother and youth reports (referred to here as "common" parameters) as well as specific A, C, and E contributions for each informant (see Figure 1). Several variations of the psychometric model were estimated, based on successful models applied to other domains of childhood antisocial behavior (Arseneault et al., 2003) as well as investigation of nonsignificant parameter estimates in the overarching model (see Table 4). Additionally, consistent with previous work (Arseneault et al., 2003), we standardized all RAgg scores within sex. To further examine potential sex differences, all variations of the

psychometric model were additionally tested separately for male and female twins. Investigations in both these samples converged on the same model indicating best overall fit as presented in Table 4 and further described below.

The best fitting model specified no mother-specific A or C contributions as well as no common E contributions (see Figure 2). Specifically, in the best-fitting model, the latent RAgg variable was defined by both mother and youth reports as indicated by the significant factor loadings, although a much greater amount of variance in the mother report (66%) was accounted for by the latent variable than that in the youth report (9%). The latent variable was substantially influenced by both additive genetic influences (accounting for approximately 62% of the variance in the latent RAgg construct) and shared environmental influences (accounting for approximately 38%). Only the youth report showed significant informant-specific additive genetic influences (accounting for approximately 37% of the variance) and shared environmental influences (accounting for approximately 4% of the variance), as well as contributions from the non-shared environmental component (accounting for approximately 49% of the variance). Non-shared environmental influences additionally accounted for 34% of the variance in the measurement of mother report.

Discussion

RAgg is a relatively new focus of scientific investigation within the broader domain of childhood antisocial behavior. Many questions about the causes and measurement of this construct have emerged from recent studies. The results from this genetically-informative, multi-informant study provide evidence of substantial meaningful variance obtained from both maternal reports and youth self-reports of RAgg behaviors. Further, in addition to substantial genetic influences on RAgg, this study indicates a prominent role of shared environmental influences on RAgg, marking an etiologic point of departure from many other childhood and adolescent antisocial behaviors. Although previous research on childhood and adolescent ASB has suggested a small role of shared environmental influences, findings have been largely inconsistent across studies (Burt, 2009; Rhee & Waldman, 2002).

Specifically, this study utilized a psychometric model to test genetic and environmental influences on both the variance common to mother and youth reports of RAgg as well as on the informant-specific variance. The latent RAgg variable was significantly informed by both mother and youth report, but more of the variance in the mother's report was captured by the latent RAgg construct, with much less of the variance in the child's report reflecting the latent construct. In other words, a larger amount of variance in the child's report was left unaccounted for after estimating the shared RAgg component. The latent RAgg factor was substantially influenced by both genetic and shared environmental influences, however. After accounting for the shared variance with the youth report, the mother-specific information was attributable entirely to non-shared environmental influences (and measurement error). In contrast, the youth-specific information showed substantial unique genetic, shared environmental, and non-shared environmental influences, even after accounting for the variance shared with the maternal report.

Relative to other studies of youth antisocial behavior that employed a psychometric model, there are several novel findings. The most salient finding that appears to be somewhat unique to the RAgg construct is the substantial influence of shared environmental influences on both the latent RAgg construct and the variance specific to the youth report. In contrast, other studies utilizing a psychometric model have not supported substantial shared environmental influences on the shared latent factor (Arseneault et al., 2003; Baker et al., 2007; Simonoff et al., 1995). The lack of significant nonshared environmental influences is also different from previous results, although nonshared environmental influences on the

shared factor in previous studies have consistently been quite small. This overall pattern of findings for common nonshared environmental influences is consistent with the potential importance of measurement error, such that informant-specific measurement error that would be reflected in nonshared environmental influences in univariate genetic analyses would not contribute to the shared factor in the psychometric model. In addition, the lack of mother-specific genetic and/or shared environmental influences is a difference from previous studies. In other words, while the non-overlapping variance in the youth report is still substantially influenced by genetic and shared environmental factors, the analogous non-overlapping variance in the mother report is not.

Turning specifically to measurement questions regarding the use of various informants, while the maternal report is clearly providing important information (such that the latent factor is influenced by genetic and shared environmental factors), this variance is primarily overlapping with information provided in the youth report. The smaller contribution of child report than maternal report to the shared variance is consistent with other studies (e.g., Arseneault et al., 2003; Baker et al., 2007; Simonoff et al., 1995), suggesting that parents may generally provide information about externalizing behaviors that would be consistent with a consensus approach focusing on shared information between reporters. In the present study, we cannot determine whether the mother-specific or child-specific information regarding RAgg will hold greater clinical utility, we can only determine the extent to which this informant-specific information is influenced by systematic shared etiologic factors.

These findings are consistent with an emphasis on the importance of both overlapping and non-overlapping information provided by multi-informant studies (Kraemer et al., 2003). Results from the univariate analyses suggest that mothers may be relying somewhat on gender biases in their conceptualizations of their children's RAgg, such that the best-fitting model for mother report allowed quantitative differences between genders but the best-fitting model for the youth report specified no such gender differences. Such a rater bias in terms of the child's gender is somewhat consistent with a recent quantitative review (Card et al., 2008) and must be considered in future studies using mother report. It is important to note that rater bias in youth self-report would be accounted for in non-shared environmental influences that are informant specific, while rater bias in mother-report would be accounted for in (or rather, indistinguishable from) informant-specific shared environmental influences, which are not significant in these results. In addition, the potential for biases related to social desirability on parent- and self-report should be directly examined in studies utilizing a range of informants. Future investigations must work toward a better understanding of the ability of common and specific sources of variance to predict important behavioral outcomes in order to fully understand the potential relevance of child-specific RAgg information.

Following from these results, this study suggests that aspects of the environment acting similarly on two twins growing up in the same household substantially influence the twins' levels of RAgg. Acts of RAgg may be more susceptible to social influence via one's family members or shared peer groups, similar to some of the presumed influences on rule-breaking deviant behavior in adolescence. In addition, this study provides substantial information about the role of different informants in measuring RAgg. Specifically, both mothers and children as assessed by structured interview are providing meaningful information about the child's behavior, such that it is substantially influenced by measurable, systematic causal factors.

Further, the significant genetic and shared environmental influences on the shared variance between mother and youth reports indicate that the extent to which they agree is offering meaningful information about the child's behavior, supporting the use of multiple informants in future RAgg studies. The results of this study suggest that information

gathered from the youth's self-report after accounting for that shared with the mother's report is also measuring some systematic, meaningful variation in the child's behavior. That is, despite the tendency to minimize the potential validity of child and adolescent self-reports in the RAgg literature, these results indicate that such reports hold important information that can be traced to systematic causal factors. As pointed out by other researchers (e.g., Baker et al., 2007), it will be important in future investigations to determine whether such child-specific genetic influences are contributing directly to the construct of interest or in fact reflect genetic influences on constructs that could present a rater bias, such as personality traits. An important remaining question is whether this non-overlapping variance contributes incrementally to predictions of external validity indicators of interest.

Limitations

A number of assumptions specific to twin study methodology deserve mention (see e.g., Plomin, DeFries, McClearn, & McGuffin, 2001, for a more comprehensive discussion of twin study methodology and associated assumptions). The validity of twin studies rests on the equal environments assumption, which asserts that although monozygotic twins are genetically identical, this greater genetic similarity (relative to dizygotic genetic similarity) does not result in more similar environmental influences on the target phenotype. Violations of this assumption could result in inflated estimates of genetic influence. Another important assumption of twin designs is that results from twin studies are generalizable to the larger, non-twin population. Complementary genetically-informative research using designs other than twin studies may be particularly helpful in addressing a number of potential limitations of the standard twin design (Turkheimer, D'Onofrio, Maes, & Eaves, 2005; Van Hulle, Rodgers, D'Onofrio, Waldman, & Lahey, 2007). Related work has recently focused on potential protective effects of positive sibling relationships, which will need to be further understood in the context of the twin relationship but may have important implications in understanding the way twins respond to environmental influences (Gass, Jenkins, & Dunn, 2007). In addition, basic twin study methodology assumes that genetic and environmental influences are additive rather than interactive, although important areas of future study including identification of potential underlying mechanisms such as gene-environment interactions and gene-environment correlations.

Although the combination of mother- and self-report was a strength of the present study, the lack of information from teachers and peers limited our ability to fully investigate the psychometric model across informants. In particular, teacher and peer reports are a common source of information in studies of RAgg (Archer & Coyne, 2005) that predict important information about negative outcomes (Crick, 1996). These results suggest that mothers and youth are providing systematic and meaningful information about RAgg, but how this information overlaps with and complements information from other informants, as well as the relative utility of information from different sources in predicting important behavioral outcomes are important future research questions. The large age range in the current sample restricted our ability to examine developmentally-specific questions about the etiology of RAgg. Larger within-age genetically-informative samples may be helpful in addressing potential influences of age on the causes of RAgg.

Future Directions

One important future direction is to build on this initial evidence for etiologic factors involved in the development of RAgg. The prominence of shared environmental influences prompts important questions regarding the role of the environment in shaping RAgg behaviors. Deviant peer groups have been highlighted as a potential influential mechanism in the development of antisocial behaviors, possibly playing a role in rule-breaking

behaviors in particular (e.g., Moffitt, 1993; Moffitt 2003). Nonetheless, some of the same genetic influences that contribute to an individual's antisocial behavior may also underlie the levels of antisocial behavior in his or her peers (Rowe & Osgood, 1984), thus calling into question the interpretation of peer characteristics as a strictly environmental influence. Research has found that relationally aggressive girls report more relational RAgg in their friends (Grotpeter & Crick, 1996; Werner & Crick, 2004), positioning peer influences as a particularly important candidate for environmental facilitation of RAgg.

It is also possible that the peer group exerts influences on RAgg through multiple pathways. In addition to social mimicry, studies have suggested that peer influences may play a role via the influence of peer rejection on later RAgg behavior (Werner & Crick, 2004; Yeung & Leadbeater, 2007). Beyond influences from the peer group, another environmental mechanism potentially involved in the development of antisocial behavior is parenting style, such as lack of monitoring or harsh discipline (Rhee & Waldman, 2002). For example, one recent study found that hostile/inconsistent parenting at age 2 was a significant predictor of increasing RAgg across early-middle childhood (Vaillancourt, Miller, Fagbemi, Côté, & Tremblay, 2007). This has primarily been studied in relation to other types of antisocial behavior (e.g., overt aggression) so it will be important for future RAgg research to incorporate parenting variables. In addition, these issues raise the important questions regarding the development of RAgg across time and potential changes in normative patterns and interaction with environmental stressors that may occur at different developmental periods.

Finally, it will also be important for future researchers to work toward placing RAgg in a more comprehensive context of the taxonomy of psychopathology. In particular, while RAgg has been conceptualized as a variant of externalizing pathology, more explicit attempts to tease apart the nature of RAgg relative to other domains of childhood psychopathology are needed. The extent to which social influences might play a role in the development of these behaviors raises important questions regarding whether RAgg should be considered a mental disorder, similar to taxonomic questions that have been raised regarding "adolescence-limited" antisocial behavior (Keenan et al., 2008; Moffitt, 1993; Underwood, Galen, & Paquette, 2001). Despite these questions, recent studies of the domain of externalizing pathology have included RAgg behaviors as one such component in both children (Baker et al., 2007) and adults (Krueger, Markon, Patrick, Benning, & Kramer, 2007). In addition, RAgg has been directly linked to borderline personality disorder symptomatology (e.g., Ostrov & Houston, 2008) and increased comorbidity with internalizing disorders relative to overt aggression (Card et al., 2008). As reviewed previously there is substantial evidence for the impairment caused by RAgg behaviors, for both aggressor and victim (Crick et al., 2001). In order to begin explicating the relationship between RAgg and pathological externalizing behaviors, researchers must make concerted efforts to integrate and directly compare RAgg findings with those in the broader antisocial behavior literature.

Work with adult populations has highlighted the utility of underlying etiological factors in elucidating common and specific factors in hierarchical models of psychopathology (e.g., Krueger et al., 2002). Researchers have established that RAgg and overt aggression are significantly correlated (Card et al., 2008; Crick, Casa, & Mosher, 1997; Vaillancourt et al., 2003) with some evidence that shared genetic factors may play a large role in this covariance (Brendgen et al., 2008), so future work should examine whether and to what degree shared etiological influences are responsible for this relationship. Nonetheless, questions regarding the best factorial structure of different types of aggression persist (Underwood et al., 2001), and will require comprehensive, structural examinations that replicate across samples and age groups (e.g., Vaillancourt et al., 2003). Similarly,

replication of results across different measures of RAgg remains important as well as increased use of common RAgg measures. Such work will propel RAgg research forward while also rooting it in the broader externalizing pathology literature.

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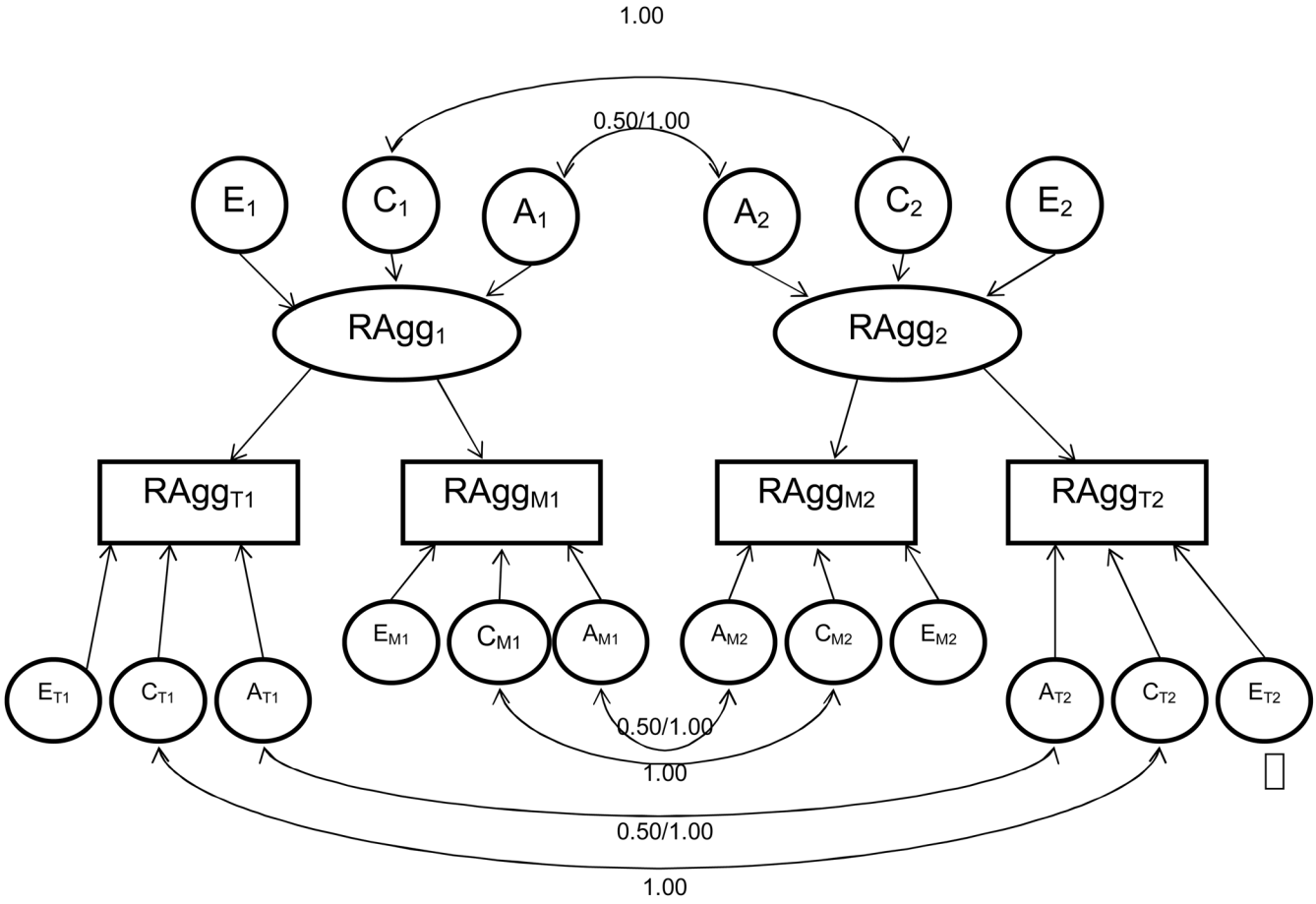

Figure 1.

Full psychometric model for RAgg utilizing mother and self-report. Latent variables are represented by circles and observed variables are represented by rectangles. The upper part of the figure reflects influences on the common variance in RAgg from both mother and self-report. Common additive genetic influence is represented by A (numerical subscripts represent twins 1 and 2), shared environmental influences by C, and non-shared environmental influences by E. The lower part of the figure reflects influences on variance in RAgg that is specific to each informant. Additive genetic (A_M), shared environmental (C_M) and non-shared environmental (E_M) represent mother-specific influences. Similarly, additive genetic (A_T), shared environmental (C_T), and non-shared environmental (E_T) represent influences specific to twins' self-report. Single-headed arrows indicate influences of latent variables on observed variables, while double-headed arrows indicate correlations among variables. Correlations between genetic influences for MZ twins are fixed to 1.0, reflecting completely shared genetic material, while genetic correlations between DZ twins are fixed to 0.5, reflecting 50% shared genetic material between DZ twins on average. Correlations between shared environmental influences for all twins are fixed to 1.0, and correlations for non-shared environmental influences are fixed to 0.

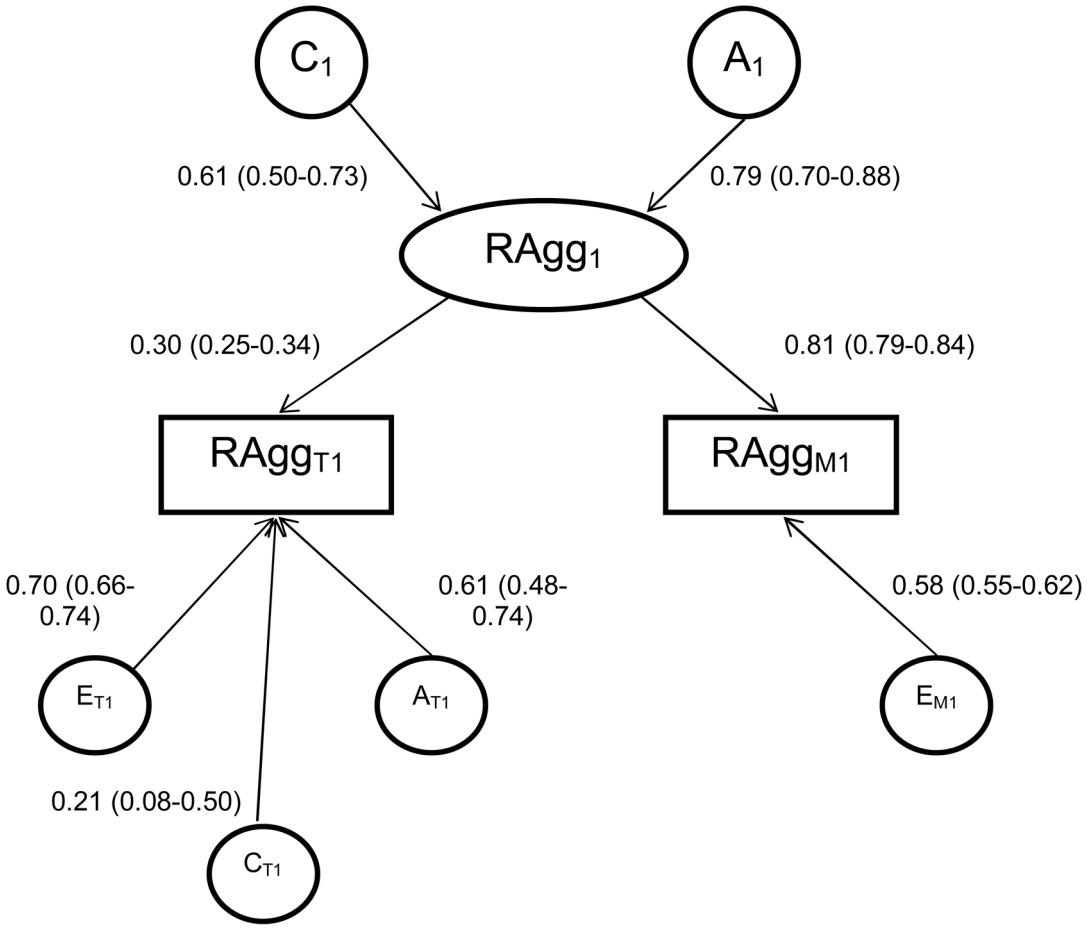

Figure 2.
Best-fitting psychometric model with standardized parameter estimates and 95% confidence intervals for Twin 1. All parameters constrained to be equal across Twin 1 and 2. Standardized parameter estimates for etiologic influences can be squared to represent the proportion of variance accounted for in RAgg.

Table 1

Item Loadings From Confirmatory Factor Analyses of RAgg Items

Item	Mother report	Youth report
Item stem: Has your child ever…	Factor loading	Factor loading
…tried to keep kids that he/she didn't like out of his/her group of friends?	0.65 *(.60–.69)*	0.48 *(.43–.53)*
…told his/her friends that he/she would stop liking them unless they did what he/she wanted?	0.72 *(.67–.76)*	0.64 *(.58–.71)*
…written notes to other children his/her age criticizing someone he/she was angry with?	0.66 *(.61–.71)*	0.62 *(.57–.67)*
…made prank telephone calls to children his/her age he/she didn't like?	0.63 *(.55–.71)*	0.65 *(.59–.71)*
…teased other people in a mean way besides his/her brother/sister?	0.71 *(.67–.75)*	0.68 *(.64–.72)*
…spread rumors about someone he/she didn't like to make others not like that person, too?	0.83 *(.79–.88)*	0.81 *(.76–.85)*
…stopped talking to people because he/she was mad at them?	0.59 *(.55–.63)*	0.51 *(.47–.56)*

Table 2

Descriptive statistics and correlations for RAgg scale across gender, zygosity, informant, and age.

	RAgg scores Means and standard deviations				Informant correlations				Age correlations			
	T1	**T2**	**M1**	**M2**	**T1-T2**	**M1-M2**	**T1-M1**	**T2-M2**	**T1**	**T2**	**M1**	**M2**
MZF	2.04 (2.19)	1.85 (2.05)	1.70 (1.98)	1.36 (1.97)	.41** *(.31–.50)*	.65** *(.58–.71)*	.27** *(.16–.38)*	.19** *(.08–.30)*	.13* *(.02–.24)*	.08 *(−.04–.19)*	.05 *(−.06–.16)*	.02 *(−.09–.13)*
DZF	2.14 (2.12)	2.16 (2.46)	1.72 (1.90)	1.60 (1.95)	.36** *(.25–.46)*	.35** *(.24–.44)*	.32** *(.20–.43)*	.33** *(.21–.44)*	.08 *(−.04–.20)*	.08 *(−.05–.20)*	.09 *(−.02–.20)*	.00 *(−.12–.11)*
MZM	2.33 (2.24)	2.27 (2.26)	1.27 (1.49)	1.08 (1.76)	.54** *(.45–.62)*	.66** *(.59–.72)*	.15* *(.03–.28)*	.15* *(.02–.27)*	−.01 *(−.13–.11)*	.03 *(−.09–.14)*	−.04 *(−.15–.07)*	.00 *(−.11–.11)*
DZM	2.36 (2.27)	2.50 (2.54)	1.54 (1.83)	1.30 (1.81)	.39** *(.29–.50)*	.61** *(.53–.68)*	.19** *(.05–.31)*	.31** *(.18–.43)*	.12 *(−.01–.24)*	.03 *(−.09–.15)*	−.06 *(−.18–.06)*	.02 *(−.10–.14)*
DZOS	2.16 (2.08)	2.14 (2.02)	1.67 (1.94)	1.25 (1.54)	.16** *(.07–.25)*	.48** *(.41–.54)*	.17** *(.07–.26)*	.22** *(.13–.31)*	.11* *(.02–.20)*	.11* *(.02–.20)*	.08 *(−.01–.16)*	−.04 *(−.13–.05)*

Table 3

Proportions of Variance with 95% Confidence Intervals of Genetic and Environmental Influences on RAgg for Mother and Self-Report

	Variance Components						
	Genetic	Environmental					
	a	c	e	χ^2	*df*	RMSEA	BIC
Mother report							
ACE –females	**.42 (.30–.55)**	**.22 (.13–.34)**	**.36 (.30–.42)**	**44.69**	**17**	**.07**	**−82.76**
-males	**.21 (.09–.39)**	**.46 (.34–.60)**	**.33 (.28–.38)**				
All parameters vary across sex							
ACE	.37 (.27–.49)	.29 (.20–.39)	.34 (.31–.39)	109.73	20	.11	−40.21
Fully constrained across sex							
AE –females	.67 (.62–.72)		.33 (.29–.38)	89.69	19	.10	−52.75
-males	.70 (.66–.74)		.30 (.26–.34)				
All parameters vary across sex							
AE	.68 (.65–.71)		.32 (.29–.35)	139.84	21	.13	−17.60
Fully constrained across sex							
CE –females		.47 (.44–.51)	.53 (.49–.56)	80.20	20	.09	−69.74
-males		.62 (.58–.65)	.38 (.35–.42)				
All parameters vary across sex							
CE		.54 (.51–.57)	.46 (.43–.50)	147.39	21	.13	−10.05
Fully constrained across sex							
E –females			1.00	690.44	21	.30	533.00
-males			1.00				
All parameters vary across sex							
E			1.00	726.22	22	.30	561.29
Fully constrained across sex							
Youth report							
ACE –females	.15 (−.01–.72)	.26 (.06–.61)	.59 (.49–.71)	41.63	17	.07	−83.61
-males	.54 (.44–.66)	.01 (−.16–.31)	.45 (.38–.53)				

	Variance Components						
	Genetic	Environmental					
	a	c	e	χ^2	*df*	RMSEA	BIC
All parameters vary across sex							
ACE	.39 (.25–.57)	.08 (.00–.26)	.53 (.47–.59)	50.52	20	.07	−96.82
Fully constrained across sex							
AE –females	.44 (.36–.52)		.56 (.49–.65)	48.19	19	.07	−91.78
-males	.54 (.47–.61)		.46 (.39–.53)				
All parameters vary across sex							
AE	**.49 (.44–.54)**		**.51 (.46–.56)**	**52.02**	**21**	**.07**	**−102.69**
Fully constrained across sex							
CE –females		.31 (.24–.38)	.69 (.62–.77)	69.74	19	.09	−70.23
-males		.39 (.32–.46)	.61 (.54–.68)				
All parameters vary across sex							
CE		.35 (.31–.39)	.65 (.61–.69)	72.14	21	.09	−82.57
Fully constrained across sex							
E –females			1.00	272.81	21	.20	118.10
-males			1.00				
All parameters vary across sex							
E			1.00	273.96	22	.19	111.88
Fully constrained across sex							

Note. Best fitting models are in bold.

RMSEA = Root Mean Square Error of Approximation. BIC = Bayesian Information Criterion. h^2 = heritiability, proportion of genetic variance; c^2 = proportion of shared environmental variance, e^2 = proportion of non-shared environmental variance.

Table 4

Fit Indices of Psychometric Models for Mother and Youth Reports of Youth's RAggression

	Overall model fit					Model difference test	
	X^2	*df*	AIC	RMSEA	BIC	$\Delta\chi^2$	Δdf
Full psychometric model	67.78	13	41.78	.07	−30.89		
No mother-specific A	68.09	14	40.09	.06	−38.17	0.31	1
No mother-specific C	68.34	14	40.34	.06	−37.92	0.56	1
No mother-specific A or C	68.34	15	38.34	.06	−50.91	0.56	2
No youth-specific A	70.52	14	42.52	.06	−35.74	2.74	1
No youth-specific C	67.84	14	39.84	.06	−38.42	.06	1
No common A	76.46	14	48.46	.07	−29.80	8.68	1
No common C	70.91	14	42.91	.06	−35.35	3.13	1
No common or unique C	93.86	16	61.86	.07	−27.58	26.08	3
No mother-specific A or C, no common E	**69.63**	**16**	**37.63**	**.06**	**−51.81**	**1.85**	**3**

Note. Best fitting model is in bold.

A = genetic variance component; C = shared environmental variance component; E = non-shared environmental variance component. AIC = Akaike's Information Criterion. RMSEA = Root Mean Square Error of Approximation. BIC = Bayesian Information Criterion.