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Differential Genetic and Environmental Influences on Reactive and Proactive Aggression in Children

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Abstract

While significant heritability for childhood aggression has been claimed, it is not known whether there are differential genetic and environmental contributions to proactive and reactive forms of aggression in children. This study quantifies genetic and environmental contributions to these two forms of aggression in an ethnically diverse urban sample of 9–10 year old twins (N=1219), and compares results across different informants (child self-report, mother, and teacher ratings) using the Reactive–Proactive Aggression Questionnaire (RPQ). Confirmatory factor analysis of RPQ items indicated a significant and strong fit for a two-factor proactive–reactive model which was significantly superior to a one-factor model and which replicated across gender as well as the three informant sources. Males scored significantly higher than females on both self-report reactive and proactive aggression, findings that replicated on mother and teacher versions of the RPQ. Asian–Americans scored lower than most ethnic groups on reactive aggression yet were equivalent to Caucasians on proactive aggression. African–Americans scored higher than other ethnic groups on all measures of aggression except caregiver reports. Heritable influences were found for both forms of aggression across informants, but while boys' self-reports revealed genetic influences on proactive (50%) and reactive (38%) aggression, shared and non-shared environmental influences almost entirely accounted for girls' self-report reactive and proactive aggression. Although genetic correlations between reactive and proactive aggression were significant across informants, there was evidence that the genetic correlation was less than unity in boys self reported aggression, indicating that genetic factors differ for proactive and reactive aggression. These findings provide the first evidence for varying genetic and environmental etiologies for reactive and proactive aggression across gender, and provide additional support for distinction between these two forms of aggression.

Keywords

Aggression; Reactive; Proactive; Genetic; Gender

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There is increasing evidence from behavioral genetic research on child, adolescent, and adult personality for the role of heritable influences in shaping aggressive behavior. An initial review by Plomin et al. (1990) of 11 twin studies of aggression found only modest heritability for aggression, with average concordance rates of 0.32 and 0.14 for monozygotic (MZ) and dizygotic (DZ) twins respectively. In contrast, a later meta-analysis (Miles and Carey 1997) argued that genetic processes explain up to 50% of the variability in aggression. A major gap in this literature is that to date we know very little, if anything, on the differential heritable and environmental influences on two specific forms of aggression: reactive (impulsive, affective, "hot-blooded") and proactive (planned, instrumental, "cold-blooded"). The present study aims to understand how genes and environment may influence these conceptually different forms of aggressive behavior in children, and the extent to which their etiologies may be overlapping or distinct.

Establishing the heritability of subtypes of childhood aggression is important for several reasons. First, antisocial and aggressive behavior is very heterogeneous; by establishing a genetic knowledge-base of different forms of aggression, a more refined and sophisticated understanding of aggression will ultimately ensue. Second, quantifying heritability for reactive and proactive aggression can inform theories of aggressive behavior, some of which have postulated different etiologies for various forms of aggression. Third, establishing heritability for differential forms of childhood aggression will help guide future molecular genetic approaches; for example, if there is substantial heritability for proactive but not reactive aggression, molecular studies would be advised to focus on the fundamental trait features that are thought to underlie this form of aggression, such as planning and regulation.

Differential Heritability of Proactive and Reactive Aggression

Different sources of evidence give rise to differential predictions of the heritability of aggression in children. Several reviews and studies have suggested that heritability for aggression increases significantly with age, and genetic influences are smaller in children than in adults (Miles and Carey 1997; Cadoret et al. 1997; Rhee and Waldman 2002). This perspective would predict modest or little heritability for proactive and reactive aggression in children as young as 9 years old. In contrast, some twin studies have suggested that in childhood, aggressive behavior has higher heritability than delinquency (Simonoff et al. 1998; Edelbrock et al. 1995), suggesting a more substantive role for genetic influences.

To date, few studies have examined different forms of aggressive behaviors. However, different perspectives on the etiology of reactive and proactive aggression may give rise to different predictions of the heritability for these two forms of aggression. On the one hand, theoretical models of reactive, defensive aggression have predominantly followed the frustration-aggression model of Berkowitz (1963), whereby aggressive behavior is viewed as a learned response to frustration. Similarly, Dodge (1991) has argued that chronic, life-threatening danger could predispose to reactive aggression and the hypervigilance associated with this form of aggressive behavior. Reactively aggressive children are also unpopular with their peers and are socially isolated (Dodge and Coie 1987; Price and Dodge 1989), and are also thought to come from harsher family environments (Dodge et al. 1997b; Dodge 1991). In particular, reactive (but not proactive) aggression has been linked to childhood abuse (Dodge et al. 1997a). These perspectives would lead to an initial prediction that environmental influences predominate for this reactive form of aggression.

Alternatively, it has been argued that affective, reactive aggression is the most common form of aggressive behavior in animals and underlies most human violence (Meloy 1988) and at intermediate levels can be viewed as normative, representing a critical self-defense mechanism common to non-primates, primates, and humans alike. The normative neural circuitry

underlying reactive, defensive aggression in animals has been laid out in detail (Gregg and Siegel 2001). Given natural individual differences and strong heritability for brain circuitry (Thompson et al. 2001), it could be argued that there would in fact be significant heritability for reactive aggression.

There are also reasons to anticipate that proactive aggression could be more influenced by environmental factors. A main theoretical perspective for understanding the etiology of proactive (instrumental) aggression is social leaning theory which argues that aggressive behavior is a learned response which is reinforced by its positive consequences and significantly influenced by coercive family processes (Bandura 1983; Patterson 2002). Crick and Dodge (1996) have also shown that proactive aggression is associated with the child's anticipation of positive outcomes of aggression, indicating some support for this social learning perspective on proactive aggression. Thus, environmental influences could be predicted to be paramount in explaining proactive aggression.

Informant Variation

A second important gap in the literature on the heritability of aggression is that very few studies have employed multiple informants. Parent, teacher, and child reports of antisocial behavior tend to correlate at low or sometimes very low levels (Hudziak et al. 2003; Tomada and Schneider 1997), with correlations between teachers and parents frequently in the 0.2 to 0.3 range (Achenbach et al. 1987). In consequence, a significant source of the heterogeneity in past findings (Rhee and Waldman 2002) may be due to report source, since studies of children tend to rely on parent and teacher ratings, while self-report measures are more common in studies of adolescents and adults. Even within a given age band, the possibility of different etiologies across informants exists, since ratings of aggression are based on different information—teachers observe children for a limited time in school settings and have a wider comparison group, while parents are most familiar with children outside of school but for longer and more intimately. Children are aware of their own behaviors across all settings, both home and school, and are also most intimately familiar with their own motivations (a key factor in discriminating reactive from proactive aggression). Individual differences in personal characteristics of each rater can influence their ratings of aggression (e.g., cognitive ability, social desirability), leading to rater bias unique to each informant. Each rater has its strengths and weaknesses, and no one rater is ideal, making it important to examine heritable influences across informants to have a clearer understanding of the etiology of aggression in childhood.

In the first review of twin studies of aggression, Plomin et al. (1990) commented: “Because aggressive behavior is likely to be heterogeneous and influenced by the context of assessment, we need multivariate measures that differentiate types and levels of aggressive behavior and multi-method approaches that consider and compare interviews and questionnaires for self-report, parent ratings, teacher ratings, and peer ratings” (p. 128). Despite this exhortation nearly three decades ago, there has been relatively little progress on this issue, with some notable exceptions (e.g. Hudziak et al. 2003). While it has been suggested that laboratory measures of aggression produce lower heritability estimates than self-report and parent measures (Miles and Carey 1997; Cadoret et al. 1997), it is not known whether child, parent, and teacher measures of proactive and reactive aggression systematically differ in heritability estimates. If different sources produce different results, this has implications for research on both biological and environmental correlates of these two forms of aggression.

Dimensionality of Aggression

A third, and conceptually important issue concerns whether proactive and reactive aggression are really separate forms of aggression. It has been argued that the distinction between the two is not longer useful, that most forms of aggression stem from mixed motives, and that schemas

and scripts learnt by direct experience or observation learning provide a better conceptual framework for understanding aggression (Bushman and Anderson 2001). This perspective makes several predictions: (1) a two-factor reactive-proactive model of aggression should not provide a significantly better fit to reactive and proactive questionnaire items than a one-factor, general aggression model; (2) the extent of environmental influences on the two purported aggression subcomponents should not differ; (3) given the highlighted role of social learning theory on schemas and scripts, genetic influences on aggression should be minimal. While some initial evidence disconfirms the first prediction (Poulin and Boivin 2000; Kempes et al. 2006; Raine et al. 2006), other predictions are largely untested.

We are aware of only one other modestly sized twin study which recently reported genetic influences in both reactive (h^2=0.39) and proactive aggression (h^2=0.41) in 6-year olds (Brendgen et al. 2006). Although interesting, these results are based on a modest sample size (N=172 twin pairs) and are based on only one rater (teachers). Moreover, opposite-sex twin pairs were not included in that study, thereby limiting the power to detect sex differences in genetic and environmental influence on aggression. It remains to be seen how the etiology of reactive and proactive aggression may appear as a function of informant source, gender, and at a later age.

Thus, the key goal of this study was to establish initial data on heritable influences on these two different forms of aggression, and to examine the relative influence of shared and non-shared environmental factors. Gender differences in the etiologies of reactive and proactive aggression are also investigated. To address the informant issue, we assessed reactive and proactive aggression from three different but commonly-utilized informants: the child, the mother, and the teacher. Because it could be argued that proactive and reactive aggression are not truly separate forms of aggression, we used confirmatory factor analysis to test whether this two-factor model produces a significantly better fit to the item data than the one-factor model. We employed a large sample of 605 pairs of twins with a narrow age range (9–10 years) to help establish reliable findings on a specifically child, pre-adolescent population. The fact that this is a relatively representative sample from a diverse urban community also allowed for the analysis of both gender and ethnicity differences in levels of proactive and reactive aggression. Although ethnic group differences in broader constructs of aggression have been well studied (see McLaughlin et al. 2007), there is little understanding of how such differences may vary across different forms of aggression.

Method

Study Design

The University of Southern California Twin Study of Risk Factors for Antisocial Behavior employs a longitudinal twin design in which MZ and DZ twin pairs and their primary caregivers are studied. The first wave of assessment occurred when the twins were 9–10 years old, with follow-up assessments approximately every 2 years. These include measures obtained through child and caregiver interviews, as well as laboratory observations, teacher reports, and school records. Additional measures of social and biological risk factors are also obtained but described elsewhere (Baker et al. 2002, 2006, 2007).

Subject Recruitment and Sample Characteristics

The participants included 605 families of twins (n=596 pairs) or triplets (n=9 sets) and their primary caregivers. The sample was comprised of both male and female MZ and DZ pairs, including both same sex and opposite DZ twins. Among the 1,219 child participants, there was an approximately equal gender distribution, with 48.7% boys (n=594) and 51.3% girls (n=625), while the caregivers were primarily female (94.2%). Median family income was $45,500,

which is comparable to the median income in California at the time of testing ($47,493; http://quickfacts.census.gov/qfd/states/06000.html). Mean ages at assessment were 9.60 years (SD=0.60) for the total sample of children and 40.14 years (SD=6.61) for their caregivers.

Twins and their families were ascertained primarily through local schools, both public and private, in Los Angeles and the surrounding communities (see Baker et al. 2002 for a detailed description). Qualifications for the study were based on age of the twins (9 or 10 years old at the time of initial assessment), their English proficiency (a stanine score of at least 3.0 on a standardized test of English proficiency), and availability to participate in a 6–8 h laboratory assessment on any day of the week. In addition, the twins' primary caregiver was required to speak either English or Spanish fluently. Child interviews were conducted in English only, while caregiver interviews were conducted in either English or Spanish.

Children's ethnicity was determined by the ethnicity of their two biological parents, as reported by the primary caregiver. As such, the twin/triplet sample was comprised of 26.6% Caucasian (n=161 pairs), 14.3% Black (n=86 pairs), 37.5% Hispanic (n=227 pairs), 4.5% Asian (n=27 pairs), 16.7% Mixed (n=101 pairs) and 0.3% other ethnicities (n=2 pairs). Among the Mixed group, most children (57.4%; n=58 pairs) have one Hispanic parent, and thus nearly half of the sample (47.1%; n=281 twin or triplet sets) is of at least partial Hispanic descent. This ethnic distribution is comparable to that in the general Los Angeles population (www.census.gov/main/www/cen2000.html), and therefore provides a diverse community sample representative of a large urban area.

Reactive–Proactive Aggression Questionnaire (RPQ)

The RPQ is a brief, 23-item self-report questionnaire, with each item coded and scored on a three-point scale (0=never, 1=sometimes, 2=often). The instrument provides three scores: proactive aggression (11 items), reactive aggression (12 items), and total aggression (see Raine et al. 2006 for full details). The RPQ assesses both physically and verbally aggressive behaviors, and in the case of reactive aggression, assesses anger generated in response to external stimuli. It takes approximately 3 min for the child to complete, has a reading age of 8 years, and is appropriate for use for both children and adults. Parallel versions were used for caregiver and teacher ratings, with wording changes to reflect the rating of another person rather than oneself. Caregivers and teachers rated each twin separately, with the RPQ being included in a larger set of surveys about each child. Raters are not requested to use any particular timeframe, but rather to indicate how often each item is generally true of the child at the time of assessment. Data were complete for 1,207 children, 1,210 caregivers, and 724 teachers.

Consistent with past studies which have observed significant proactive-reactive intercorrelations of 0.76 (Dodge and Coie 1987), 0.67 (Grafman et al. 1996), 0.47 (Poulin et al. 1997), 0.41 (Kagan 1992), and 0.67 (Raine et al. 2006), the proactive and reactive scales (computed as item means) were significantly ($p<0.001$) correlated for self-report (r=0.48 for boys; r=0.46 for girls), teacher (r=0.80 for boys, r=0.73 for girls), and caregiver (r=0.63 for boys; r=0.56 for girls) versions of the RPQ in the present sample.

Statistical Methods

Confirmatory Factor Analysis The 23 items (12 reactive and 11 proactive) were subjected to confirmatory factor analysis (CFA). Separate analyses were conducted for child, mother, and teacher versions to assess for invariance of factor structure across raters. Confirmatory factor analysis was conducted using EQS 6 (Bentler 1995, Bentler and Wu 2002). Due to significant kurtosis for many of the items (Mardia's normalized multivariate kurtosis was greater than 3) as well as variability in kurtosis across items (ranging from •0.24 to 0.34), the heterogeneous kurtosis method was used to estimate the distribution of covariances (Bentler et al. 1991; Kano

et al 1990). Listwise deletion procedures were used to remove any cases with missing data on one or more RPQ items.

Two models based on prior research were evaluated: a one-factor model (general aggression) and a two-factor model (proactive and reactive aggression). To provide a baseline comparison for these two models, the null model was also fitted. Under this model, each of the items is assumed to represent completely independent and uncorrelated dimensions of aggression. Six commonly used goodness-of-fit indices were reported to assess the fit of the models: chi-square ($\bullet^2$), chi-square/df ($\bullet^{2/df}$; Bentler and Dudgeon 1996), the normed fit index (NFI; Bentler and Bonnet 1980a, b), the non-normed fit index (NNFI; Bentler and Bonett 1980a,b), the comparative fit index (CFI; Bentler 1990), and the root mean squared error of approximation (RMSEA) index (McDonald 1989). The Satorra–Bentler scaled difference chi-square (S–B $\bullet^2$) was used for assessing model fit instead of unadjusted $\bullet^2$ values (Satorra and Bentler 2001). Direct comparisons were made between the one and two-factor models that are in hierarchical relationship (i.e. nested) using the difference chi-square ($\bullet \bullet^2$) test (Loehlin 1992), with the one-factor model nested within the two-factor model. After the CFA model was fit to the full sample, we conducted a two-group analysis to verify that the model was similar across gender. Covariances along with the measurement model were constrained to be equal or invariant across the two groups. The same six goodness-of-fit indices were used to assess the fit of these models.

Genetic Analyses Bivariate genetic models were fitted to proactive and reactive aggression within each rater, using a full Cholesky parameterization to estimate the magnitude and significance of additive genetic (A), shared twin environment (C), and non-shared environment (E) variance for each scale, and the equality of effects between males and females. In addition to providing estimates of relative importance of genetic, shared and non-shared environment to each form of aggression, these analyses yield estimates of the genetic correlation (r_A), shared twin environment correlation (r_C) and nonshared environment correlation (r_E) between reactive and proactive aggression; i.e., the extent to which genetic and/or environmental factors correlate between the two types of aggression. We also considered common-factor models to estimate the genetic and environmental effects both common and specific across raters, but deemed these inappropriate given the relatively low phenotypic correlations between the three raters.

Given the highly skewed distributions for proactive aggression within each rater, all scales were transformed using a ranking and normalization procedure prior to genetic analyses. Models were fit to the transformed scores using raw maximum likelihood estimation procedures in order to make use of data for all participants, including those for which there were incomplete data for pairs of twins. Saturated covariance models were also fit to estimate the phenotypic means and variance-covariance matrices within each of the five zygosity groups, and these served as a baseline comparison model for the bivariate Cholesky models. Significance tests for differences among means across zygosity group as well as for Twin A and Twin B were performed in these covariance models. For both proactive and reactive scales, no mean differences were found across co-twins in same-sex pairs or across zygosity groups. The saturated model with equal means across twin and zygosity group was thus used as a basis of comparison for other more highly constrained (bivariate) genetic models. Models were compared using both chi-square statistics and Akaike's Information Criterion (AIC) (Akaike 1987)

In order to confirm the two-factor structure of the items across twins, zygosity group and gender, additional genetic models using one or two latent common factors for each twin were fit to the item data within each rater. A bivariate ACE Cholesky decomposition with sex differences was used for the latent factors underlying the 23 items in the two-factor model,

while the second set of ACE factors were dropped for both boys and girls in the one-factor model.

Results

Confirmatory Factor Analysis

The correlated two factor model (reactive–proactive) was compared with a one-factor model (general aggression) and the null model. Factor loadings and goodness-of-fit indices are presented for the total sample in Tables 1 and 2. Both the two-factor and one-factor models fitted better than the null model. In addition, the chi-square difference test indicated a highly significant improvement in fit for the two-factor model (reactive-proactive) over the one-factor model for children (•• ^{2}=383, df=2, p<0.0001), mother (•• ^{2}=397, df=2, p<0.0001), and teacher (•• ^{2}=971, df=2, p<0.0001) report versions. For all three versions, all fit indices were without exception superior for the two-factor model than the one-factor model.

The child version gave a better fit for the two-factor model than either the mother or teacher versions on all indices without exception, with an RMSEA of 0.037 indicating an excellent fit and with all other fit indices at 0.91 or above for the child version. Nevertheless, the two-factor model fits were good or at least acceptable even for mother and teacher versions, with fit indices of 0.89 or above, RMSEA below 0.09, and the CFI indicating that at least 90% of the covariation in the data is reproduced by the two-factor model. Although fit indices for the teacher version were less favorable than child or caregiver versions, in all three informants, the two-factor model fit significantly better than the one-factor model.

Correlations between the two latent factors based on the CFA were moderate to high for all three informants: r=0.57 in boys and r=0.62 in girls for child self-report; r=0.63 in boys and r=0.73 in girls for caregiver reports; and r=0.78 in boys and r=0.89 in girls for teacher reports. Although the two-factor model of aggression appears to hold across different raters, the separation of reactive and proactive forms appears most evident for self-report.

Additional CFA using a multiple group approach (results available upon request) suggested that invariance of the two-factor structure between the two sexes within each rater could be obtained under certain constraints, including allowing correlated error variances for some of the proactive items (e.g. “Fights to be cool” and “Carries weapon to use in a fight”), in self report measure with S–B •• ^{2}=29.78 df=19, p>0.05 suggesting that metric invariance although not complete does partially hold across gender (with the exception of two items). Practical fit indices also supported the model with factor loadings equal (except two items) across groups as a reasonable well-fitting representation of the data (CFI=0.90, NNFI=0.98, RMSEA=0.014; these results available upon request).

Finally, the two-factor structure was confirmed further in genetic models of the item data for twin pairs. The one-factor general aggression model with unitary ACE effects influencing both reactive and proactive items was compared to a two-factor model with separate but correlated genetic and environmental influences for the reactive and proactive factors. The one-factor model fit significantly worse than the two-factor model for both boys and girls in each rater (Child self report: •• ^{2}=221.85, df=6, p<0.001 in boys; •• ^{2}=157.14, df=6, p<0.001; Caregiver report: •• ^{2}=370.34, df=6, p<0.001 in boys; •• ^{2}=169.97, df=6, p<0.001; Teacher report: •• ^{2}=344.37, df=6, p<0.001 in boys; •• ^{2}=57.16, df=6, p<0.001).

Test–Retest Reliability

Test–retest reliability was assessed for 60 randomly selected participants for self-report and caregiver versions of the RPQ over a 6-month retest period. This is a much longer retest period than usually employed and can confound temporal reliability with genuine change on

aggression; it does however provide a stringent test of reliability. Retest reliabilities were as follows: caregiver proactive (r=.79, p<0.0001), caregiver reactive (r=0.81, p<0.0001), caregiver total aggression (r=0.84, p<0.0001), self-report proactive (r=0.67, p<0.0001), self-report reactive (r=0.64, p<0.0001), self-report total aggression (r=0.71, p<0.0001).

Internal Reliability

xAll three versions of the RPQ showed acceptable internal reliability, with Cronbach's alpha ranging from 0.74 to 0.94 (see Table 3). As would be expected, relatively higher reliabilities were obtained for the longer, total score scales than for shorter subscales, while somewhat higher internal reliabilities were obtained for the teacher version.

Gender and Ethnic Group Differences in Mean Levels

Males consistently showed higher scores than females on reactive, proactive and total aggression scores on all three report sources (see Table 3). Effect sizes ranged from 0.15 to 0.43, with somewhat larger gender differences for the teacher version than for caregiver and self-report versions.

Ethnic differences were pronounced on self-report and teacher versions but were almost absent on the caregiver version (see Table 4). On the self-report RPQ, African–American children scored higher on proactive, reactive, and total aggression scales than most other ethnic groups. These differences were even stronger on the teacher RPQ, but absent on the caregiver RPQ.

Asian–American children scored significantly lower on reactive aggression compared to almost all other groups on both self-report and caregiver versions, but not the teacher version. Interestingly, Asian-Americans were comparable on proactive aggression to Caucasians and Hispanics on all versions of the RPQ.

Caucasians had equivalent self-report reactive aggression scores to African–Americans and significantly outscored both Hispanic, Asian–American, and Mixed groups. Nevertheless, these differences were not observed on either teacher or caregiver versions.

Correlations between Self-Report, Teacher, and Caregiver Reports

Consistent with other reports of low relationships between self-report, teacher, and caregiver measures of antisocial behavior (e.g. Achenbach et al. 1987), correlations between the three RPQ versions across raters were significant but small in magnitude, with subscale and total scale inter-correlations ranging from 0.18 to 0.26, (mean=0.22, median=0.23, all p<0.0001).

Genetic and Environmental Influences

Twin Correlations Twin correlations for reactive and proactive aggression within each of the five zygosity groups are presented in Table 5. The twin correlations are all moderate, except for child report in DZ boy–girl pairs. MZ correlations were higher than DZ correlations except for girls' self-report. This pattern suggests the presence of a genetic influence (A) in both reactive and proactive aggression for both mother and teacher reports of boys and girls, as well as boys' own self-reports. However, the DZ correlations are generally greater than one-half the MZ correlations, suggesting the presence of significant shared twin environmental influences for both proactive and reactive aggression across all raters.

Genetic Modeling Estimates of genetic and environmental influences on proactive and reactive aggression were obtained through bivariate genetic models. Compared to saturated covariance models, full ACE models (with separate means and ACE effects in males and females) fit extremely well within each rater: Child self-report •^{2}=41.32, df=48, p=.74; AIC=•54.68; Caregiver report •^{2}=46.32, df=48, p=0.54, AIC=•49.68; teacher report •^{2}=44.80, df=48,

p=0.61, AIC=•51.20. Sub-models with various constraints were thus fit to each scale, including (a) equating ACE effects in boys and girls; and (b) dropping A, C, or both effects. In all models, means for reactive and proactive aggression were allowed to differ for boys and girls, given strong evidence of significant sex differences in this sample. Models with dominance effects were not fit to the data since there was no strong evidence for genetic non-additivity (i.e., in no case did the MZ correlation exceed twice the value of the DZ correlation for same-sex pairs).

Compared to the saturated models, ACE components could be constrained to be equal for boys and girls in both mother (•^{2}=64.90, df=57, p=0.22, AIC=•49.10) and teacher reports (•^{2}=62.00, df=57, p=0.30, AIC=•52.00), but not child reports (•^{2}=82.39, df=57, p=0.01, AIC=•31.61). In child reports the best fitting model included only A and E effects in boys, but C and E effects in girls (•^{2}=43.77, df=54, p=0.84, AIC=•64.22). For caregiver and teacher reports, neither A nor C could be dropped from the model without a significant reduction in fit. Thus, additive genetic (A) effects were important for all measures of aggression except girls' self reports, and shared environmental (C) effects were important for all measures of aggression except boys' self-reports.

ACE Estimates Standardized final model estimates of relative effects of A, C, and E are presented in Table 6, along with their 95% confidence intervals (CI). Regarding genetic effects, for boys, significant heritability (A) was observed for both forms of aggression across all three informants, with highest heritabilities for self-report proactive (0.50) and reactive (0.38) aggression. In striking contrast, there was zero heritability for girls' self-reported reactive and proactive aggression. Genetic influences for proactive aggression tended to be higher than for reactive aggression. Although heritability estimates were not significantly different from one another based on chi-square tests (Boys' self report •^{2}=2.24, df=1, p=0.13; Caregiver report •^{2}=.19, df=1, p=0.99; Teacher report •^{2}=2.62, df=1, p=0.10), models with heritability constrained to be equal for proactive and reactive aggression did show less favorable AIC values compared to those with unconstrained heritabilities (AIC=•63.99, •50.91, and •51.38 for child, caregiver, and teacher reports, respectively). There is thus a trend toward significantly greater heritability for proactive aggression, which should be explored in other larger samples.

Common environmental influences were significant in all cases except in boys' self-reported aggression, and were most notable for reactive aggression across all three raters. C effects were significantly higher for reactive compared to proactive aggression for teacher reports (•^{2}=5.15, df=1, p=0.02) as well as girls' self-reports (•^{2}=9.15, df=1, p=0.002). There is substantial evidence for non-shared environmental influences on both forms of aggression in both genders (E ranging from 0.37 to 0.86), although it should be noted that E effects include measurement error for each scale.

Rater Bias Models Given the significant estimates of shared environmental influence, particularly for teacher reports of reactive aggression, post hoc analyses also explored the possibility of rater bias. Because the same teacher completed the RPQ for both twins in only 31% of the twin pairs, we were able assess the effects of a shared rater for teacher reports. Thus, in order to evaluate the extent to which rater bias might have occurred in RPQ ratings, and especially how this might have affected our ACE estimates, additional genetic analyses were performed using classroom status (same vs. different) for the two twins. This effectively estimates a separate variance component (T) reflecting increased similarity for twins in the same classroom and who were rated by the same teacher (see Baker et al. 2007 for details). Similar to what we have previously reported for the broader construct of externalizing behavior problems (Baker et al. 2007), significant rater bias effects were found for both Reactive (T=0.35; 95% CI=0.24, 0.49) and Proactive (T=0.34; 95% CI=0.25, 0.46) aggression. Nonetheless, both genetic and shared environmental effects remained significant for both aggression scales even after accounting for rater bias (Reactive: A=0.19, 95% CI=0.05, 0.38;

C=0.30, 95% CI=0.11, 0.46; Proactive A=0.31, 95%CI=0.13, 0.49; C=0.14, 95%CI=0.02, 0.30). Moreover, classroom status did not affect ACE estimates in either caregiver or child self reported aggression, suggesting that being in the same classroom does not generally account fort greater twin similarity across the board.

Finally, to address further the possibility of sample bias in the teacher reports (given the return rate of 62.5%), we compared the caregiver and self-report RPQ scores for those children whose teachers did and did not participate. There were no significant differences in either proactive (self-report: t=1.63, df=1031; caregiver report: t=0.69, df=1035) or reactive aggression (self-report: t=0.84, df=1031; caregiver report: t=0.85, df=1035) for children with and without teacher RPQ data (p>0.10 in all cases), suggesting that teacher responses were not associated with the child's aggression per se.

Genetic and Environmental Correlations Given the high phenotypic correlation between reactive and proactive aggression (ranging from r=0.46 to 0.80, depending on sex of child and rater), it is important to consider the extent to which genetic and environmental influences are common to these two forms of aggression. The extent to which these two forms of aggression share the same etiology can be understood through the genetic (r_A) and environmental correlations (r_C and r_E) between reactive and proactive aggression, which are also produced from the bivariate Cholesky models. These estimates from the best fitting models above are presented in Table 7, separately for each rater. As shown, the genetic correlation (r_A) is substantial (0.57 to 1.00) and significant ($p<0.05$) in all cases where A effects are evident, including boys' self-report and caregivers' and teachers' reports (boys and girls combined). Shared twin environmental influences are also significantly correlated for the two forms of aggression in both mothers' and teachers' reports, as well as in girls' self-reports. Most importantly, the 95% confidence intervals for r_A and r_C in both caregiver and teacher reports include the value of 1.0, suggesting complete overlap in the genetic and shared environmental influences for reactive and proactive aggression when the child is rated by others. For child reports, however, neither r_A (in boys) nor r_C (in girls) includes 1.0 in the 95% confidence intervals, indicating the importance of unique genetic and shared environmental influences for children's own ratings of their reactive and proactive aggression. These results highlight the importance of children's self-report as more refined measures of differential forms of aggressive behavior, as compared to ratings by others who may not clearly distinguish the motives and circumstances surrounding the child's aggressive behavior.

Discussion

The present study used a combination of phenotypic and genetic analyses to determine whether aggression can be meaningfully separated into proactive versus reactive behaviors. To our knowledge, this is the first multi-informant twin study of reactive and proactive aggression in early adolescence. In phenotypic analyses, strong replication was obtained for the superiority of a two factor proactive-reactive aggression model over a one-factor general aggression model across all three report sources, although the self-report source gave the best fit and the fit for the teacher source the least strong fit. Consistent with other studies, however (Dodge and Coie 1987; Grafman et al. 1996; Kagan 1992; Poulin et al. 1997; Raine et al. 2006), in our study proactive aggression was significantly correlated with reactive aggression for all reporters and genders (r=0.46 to 0.80). Males scored significantly higher than females on both reactive and proactive aggression, findings that replicate across all three informants. Ethnic differences generally confirmed prior findings on broadly-measured aggression (e.g., Laird et al. 2005; McLaughlin et al. 2007), with the exception that Asians scored lower than most ethnic groups on reactive aggression but were equivalent to Caucasians on proactive aggression.

A key finding from the genetic analyses is that there is significant heritability for both proactive and reactive aggression as early as 9 years of age. Significant genetic influences were generally obtained for both proactive and reactive aggression, but there were important differences across gender and rater. In particular, a striking gender difference was obtained on self-report aggression. For boys, strong genetic influences were found for proactive (50%) and to a less extent reactive (38%) aggression. In contrast, no heritability was observed for female self-report proactive or reactive aggression, with shared and non-shared environmental influences almost entirely accounting for variability in these two forms of aggression in 9–10 year old girls. One prediction that could follow from these findings is that neurobiological research on self-report aggression may show somewhat stronger effects for males than for females, particularly for biological variables with a strong genetic loading. In contrast, genetic influences in both females and males were found for teacher reports of proactive (45%) and reactive aggression (20%) as well as caregiver reports of proactive (32%) and reactive aggression (26%), and apparently equivalent for both sexes, leading to the prediction that neurobiological research on teacher or caregiver-rated aggression may find effects for both male and female children. Given the lack of sex differences in genetic and environmental etiology using caregiver and teacher ratings, these findings are thus not entirely consistent with the conclusion from the meta-analysis by Miles and Carey (1997) that genetic influences tend to be higher for male than female aggression, and suggest that this issue requires further investigation.

In terms of etiological difference between reactive and proactive aggression, a salient finding is that genetic influences appeared to contribute more to proactive than reactive aggression, (with the exception of girls self-report, where no genetic effects were found for either form of aggression). This suggests that reactive aggression in young children may arise more in response to environmental influences such as the aggravating behavior of peer group members than proactive aggression, which may instead be more influenced by genetic processes.

What was common to both forms of self-report aggression in boys is that the influence of the shared (twin) environment was virtually non-existent. In contrast, for females there were differences between proactive and reactive aggression, with non-shared environmental influences being significantly higher for proactive (86%) than reactive (64%) self-report aggression. This may indicate that for females, proactive aggression may arise more through modeling of sibling and parent behavior and parental use of aggression to achieve desirable goals. This in turn has implications for future research on the differential etiology of reactive versus proactive aggression as assessed using self-reports. These findings are not consistent with the views that environmental processes (learning of scripts and schemas) predominate for both forms of aggression, and that there is little distinction between reactive and proactive forms of aggression (Bushman and Anderson 2001).

A potentially important finding from this study is that different conclusions on the influence of genetic, shared environmental, and non-shared environmental influences are obtained from the different report sources of aggressive behavior. Significant differences were obtained in ACE estimates across informants, findings that also varied with gender. Regarding reactive aggression, for boys, self-reports showed stronger effects of genetic influences than mothers reports, while for girls, non-shared environmental influences were stronger for self-reports than mother or teacher reports. These differences across raters precluded a multi-informant modeling approach. One implication of these interesting but complex findings is that depending on the informant source, researchers could draw quite different conclusions concerning not just the heritable, shared environmental, and non-shared environmental influences of proactive and reactive aggression, but also the specific biological and psychosocial factors that impact these forms of aggression. The use of multiple informants is a methodological strength but it also raises complex questions to which there is no easy resolution. While one strategy may be

to aggregate scores from all three sources to obtain a more comprehensive assessment of proactive and reactive aggression, the low correlations between these sources (r=0.18 to 0.26) does not provide strong support for this approach (see also Baker et al. 2007 for a further discussion of this issue).

The RPQ was originally developed for self-report use because such reporting was felt to have a potentially important advantage over teacher rating or parent measures. Intrinsic motivation for the aggressive act forms a key distinction between proactive and reactive aggression (e.g. the proactive "had fights to show who was on top" and the reactive "damaged things because you felt mad"), and children as the initiator of the act likely have more insight into the driving motivation behind the act, a motivation which is often obscure to the observer. The self-report version resulted in a better two-factor model fit than the mother and teacher versions. In contrast, a questionable result from the mother version is that it was relatively insensitive to ethnic group differences, even though these differences are well-established for broadly-assessed aggression. Furthermore, common environment effects are not commonly found in psychological traits in humans, but while absent or minimal for the self-report version they were generally more substantial for mother and teacher versions. Given the fact that the same individual provides ratings of the two co-twins (for 30% of the teachers and 100% of the caregivers), it is possible that these shared environmental effects reflect some form of rater bias. The common environment effects did remain significant in teacher ratings, even when controlling for rater bias, but it is impossible to separate rater bias from true common environmental influence in caregiver ratings. The confound between rater bias and common environmental influence does not exist in child self-report versions, however, making this a particularly important tool for investigating both genetic and environmental effects in aggression. The self-report version of the RPQ could thus be particularly useful for pursuing biological/genetic hypotheses in boys and psychosocial/environmental hypotheses in girls for proactive and reactive aggression, given the striking gender differences for the ACE components of the self-report measure.

One important finding from the multivariate genetic analyses is the substantial overlap in both the genetic and environmental processes that give rise to both reactive and proactive forms of childhood aggression—in fact, there is almost complete overlap of genetic and shared environmental processes in reactive and proactive aggression as rated by either parents or teachers, based on results of genetic and environmental correlations (see Table 7). Despite evidence for shared influences, there is also evidence that different etiological processes are involved in shaping reactive and proactive forms of aggression, particularly for child self-reports, since genetic and environmental correlations are significantly less than unity. These findings indicate that while there is strong linkage between proactive and reactive aggression, there is clear evidence that different processes contribute to these two forms of aggression when reported by the children themselves. Yet again, the almost complete overlap between reactive and proactive aggression for A and C (but not E) components illustrates differences in conclusions across informant sources.

Regarding ethnic differences, as anticipated from prior research African-Americans had higher proactive and reactive aggression scores than most other ethnic groups on both self-report and teacher ratings, except as noted above for the mother ratings. Perhaps the most interesting (and unexpected) finding from the analysis of ethnic differences on proactive and reactive aggression is that while Asians had significantly lower scores on reactive aggression compared to most other ethnic groups including Caucasians, they did not differ significantly on proactive aggression to Hispanics, Caucasians and the mixed ethnic group. Indeed, Asians had non-significantly higher scores on teacher proactive aggression compared to Caucasians (d=0.20). Given findings from temperament research showing Asians are more inhibited than Caucasians (Kagan 1994), and given that disinhibition is believed to be a risk factor for reactive impulsive

aggressive behavior (Raine et al. 1998), it may be that Asians possess a greater ability to inhibit aggressive feelings generated by insults, and yet be equally adept at using aggression in a controlled and regulated fashion to achieve their desired goals.

Despite a six-month retest period, excellent test-retest reliability for the caregiver version was obtained, with reliabilities ranging from .79 to .84. Retest reliability for the self-report version were somewhat lower, ranging from 0.64 to 0.71. Three caveats need to be placed on these retest reliabilities, particularly for the self-report version. First, the test-retest period at six months was stringent and conservative; reliabilities may be even higher but be reduced due to genuine, reliable developmental change in aggression over time. Second, if the self-report version is more sensitive to the motivational processes underlying proactive and reactive aggression, true developmental change may occur more in this version than the caregiver and teacher versions which is less sensitive to this fundamental aspect of proactive–reactive aggression. Third, while self-report retest reliability was modest, retest coefficients of 0.64 to 0.71 approached the internal reliability of the self-report scales which ranged from 0.74 to 0.82, indicating relatively robust retest reliability.

Although the two-factor model of reactive and proactive aggression is well supported in both phenotypic (CFA) and genetic analyses, the distinction between these two forms of aggression seems particularly strong in the child self-report version. This was evident in the best overall fit in the CFA in boys and girls self-rated aggression, as well as in the significant departure of the genetic correlation from unity, both results suggesting a valid phenotypic distinction between reactive and proactive items. The less good fit of the two-factor model in CFA of caregiver's report happens to coincide with less sensitivity to mean ethnic differences in this version of the RPQ. Given that parents have limited information about their own child in relation to their larger peer group, parents may be less sensitive overall to subtle but important aspects of their children's aggressive behavior, including their relative standing when compared to a wide variety of other children, as well as the reactive-proactive distinction. Moreover, the even less good CFA fit in teacher reports compared especially to child self-ratings does also coincide with some evidence for rater bias based on genetic analyses. Teachers may be less able to distinguish perpetrators from victims, and less able than children themselves to accurately separate reactive from proactive forms of aggression. Taken together, the findings suggest that children themselves may be able to distinguish and report more clearly the different forms of aggression at this early age. Of the three sources, the self-report version may have the greatest utility in research, particularly that aiming to understand different types of aggression that stem from different motivations and other causal factors.

In conclusion, findings from both confirmatory factor analysis and multivariate genetic models provide evidence for two correlated, but essentially different, forms of aggressive behavior in children. While genetic and environmental influences are correlated across these two forms of aggression, there is also evidence for different etiological processes acting on proactive and reactive aggression when using children's self-reports. The fact that a two-factor proactive-reactive model fitted the data significantly better than a one-factor model—particularly in the child self-report version—indicates that the distinction between these two forms of aggression is a valid one. Findings further indicate that there are different gender-specific environmental and genetic etiologies for reactive and proactive aggression that have implications for future research.

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Table 1

Factor Loadings for the RPQ Items in the Two-Factor Model for Each Respondent (Self, Caregiver, Teacher)

RPQ items	Factor loadings					
	Reactive			Proactive		
	Self	Caregiver	Teacher	Self	Caregiver	Teacher
Yelled when annoyed	0.56	0.63	0.77	—	—	—
Angry when provoked	0.55	0.66	0.82	—	—	—
Angry when frustrated	0.52	0.62	0.78	—	—	—
Temper tantrums	0.41	0.52	0.58	—	—	—
Damaged things when mad	0.31	0.40	0.27	—	—	—
Angry when don't get way	0.53	0.66	0.79	—	—	—
Mad when lost a game	0.49	0.56	0.76	—	—	—
Angry when threatened	0.50	0.58	0.66	—	—	—
Felt better after hitting	0.32	0.36	0.37	—	—	—
Hit to defend self	0.44	0.52	0.38	—	—	—
Hit when teased	0.50	0.57	0.57	—	—	—
Fight for status	—	—	—	0.56	0.66	0.70
Taken things from others	—	—	—	0.42	0.54	0.55
Vandalized for fun	—	—	—	0.30	0.26	0.49
Gang fight to be cool	—	—	—	0.40	0.23	0.56
Hurt others to win game	—	—	—	0.37	0.37	0.53
Force to manipulate others	—	—	—	0.49	0.51	0.73
Force to obtain money	—	—	—	0.22	0.31	0.35
Threatens and bullies	—	—	—	0.38	0.50	0.72
Obscene phone calls for fun	—	—	—	0.30	0.10	0.29
Manipulate others to gang up	—	—	—	0.33	0.22	0.64
Carried weapon for use	—	—	—	0.06	0.05	0.21
Yelled to manipulate	—	—	—	0.49	0.62	0.64

Table 2

Model-fitting Results Comparing the Null Model with the One Factor (General Aggression) Model and the Two-Factor (Proactive–Reactive) Model for Each of The Three Respondents (Self, Caregiver, Teacher)

Summary of model fit indices							
Model	$\bullet^2$	**df**	$\bullet^{2/df}$	**NFI**	**NNFI**	**CFI**	**RMSEA**
Self-report							
Null Model	7222	253	28.5	—	—	—	—
One factor	984	229	4.3	0.86	0.88	0.89	0.052
Two factor	601	227	2.6	0.92	0.94	0.95	0.037
Mother-report							
Null Model	12266	253	48	—	—	—	—
One factor	1596	229	7.0	0.87	0.87	0.89	0.071
Two factor	1199	227	5.3	0.90	0.91	0.92	0.060
Teacher-report							
Null Model	11659	253	46	—	—	—	—
One factor	2304	229	10	0.80	0.80	0.82	0.130
Two factor	1333	227	5.8	0.89	0.89	0.90	0.094

All $\bullet^2$ values are statistically significant ($p<0.0001$). The Sattora-Bentler Scaled difference chi-square was used in assessing fit and comparing models.

NFI Normed Fit Index, NNFI Non-Normed Fit index, CFI Comparative Fit Index, RMSEA Root Mean-Squared Error of Approximation index.

Table 3

Means, SDs, Gender Difference Effect Sizes and Coefficient Alpha for RPQ Scores for Males, Females, and the Total Sample

	Total Sample	Males	Females	d	t	p	alpha
Self-report							
Proactive							
Mean	0.90	1.12	0.69	0.24	4.14	0.0001	0.74
SD	1.80	2.07	1.49				
Reactive							
Mean	6.99	7.32	6.67	0.18	3.17	0.002	0.76
SD	3.61	3.70	3.48				
Total							
Mean	7.89	8.44	7.37	0.21	3.95	0.0001	0.82
SD	4.74	5.00	4.40				
Teacher							
Proactive							
Mean	1.93	2.37	1.53	0.26	3.13	0.002	0.91
SD	3.22	3.69	2.68				
Reactive							
Mean	5.15	6.11	4.26	0.43	5.32	0.0001	0.91
SD	4.50	4.82	3.93				
Total							
Mean	6.73	8.11	5.52	0.38	4.39	0.0001	0.94
SD	7.01	7.90	5.87				
Caregiver							
Proactive							
Mean	1.13	1.27	0.99	0.15	2.56	0.011	0.77
SD	1.87	2.07	1.66				
Reactive							
Mean	7.42	7.84	7.01	0.23	3.90	0.0001	0.83
SD	3.67	3.70	3.61				
Total							
Mean	8.55	9.13	7.99	0.23	3.91	0.0001	0.86
SD	5.02	5.16	4.82				

Table 4

Ethnic Group Differences in the RPQ

	Caucasian	Hispanic	African–American	Asian	Mixed	F (df)	p	t (p<0.05)
Self-report								
Proactive								
Mean	0.63	0.98	1.16	0.56	1.05	3.78	0.005	AA>C,A
SD	1.15	1.99	2.11	1.16	2.04	(4,1199)		M>A
Reactive								
Mean	7.53	6.64	7.57	5.44	6.84	6.62	0.0001	AA>C,A,M
SD	3.29	3.59	4.09	3.44	3.53	(4,1197)		C>H,M,A A<C,H,AA,M
Total								
Mean	8.14	7.62	8.74	6.00	7.90	4.13	0.003	AA>H,A
SD	3.97	4.85	5.56	4.13	4.86	(4,1192)		A<C,H,AA,M
Teacher								
Proactive								
Mean	1.26	1.94	3.58	1.78	1.92	6.60	0.0001	AA>C,H,A,M
SD	2.41	3.14	4.67	3.62	2.91	(4,568)		H>C
Reactive								
Mean	4.74	4.82	7.33	4.72	5.10	5.67	0.0001	AA>C,H,A,M
SD	4.19	4.42	5.19	4.16	4.23	(4,568)		
Total								
Mean	5.58	6.29	11.01	7.08	6.52	7.95	0.0001	AA>C,H,A,M
SD	5.63	6.78	9.64	7.39	6.36	(4,568)		
Caregiver								
Proactive								
Mean	1.07	1.15	0.98	0.89	1.33	1.15	0.33	
SD	1.70	1.84	2.04	1.70	2.09	(4,1197)		
Reactive								
Mean	7.62	7.40	7.13	6.06	7.74	2.75	0.027	A<C,H,M
SD	3.51	3.62	3.61	3.26	4.08	(4,1153)		
Total								
Mean	8.68	8.57	8.14	6.94	9.07	2.23	0.063	
SD	4.68	4.94	5.13	4.54	5.63	(4,1150)		

t-tests are two-tailed

C Caucasian, H Hispanic, A Asian, AA African-American, M Mixed

Table 5

Twin Correlations for Reactive and Proactive Aggression

	MZ boys	DZ boys	MZ girls	DZ girls	DZ boy-girl
Reactive					
Child report	0.38*	0.28*	0.37*	0.38*	0.08
Mother report	0.48*	0.35*	0.60*	0.46*	0.50*
Teacher report	0.59*	0.49*	0.70*	0.43*	0.60*
Proactive					
Child report	0.60*	0.34*	0.12	0.28*	0.14
Mother report	0.61*	0.34*	0.57*	0.48*	0.55*
Teacher report	0.56*	0.42*	0.74*	0.38*	0.35*

Table 6

ACE Estimates (and 95% Confidence Intervals) from Best-fitting Bivariate Cholesky Models Fitted to Reactive and Proactive Aggression for Each Rater

	A	C	E
Reactive			
Child report			
Boys	0.38* (0.25, 0.49)	—[a] —[a]	0.62* (0.51, 0.75)
Girls	—[a] —[a]	0.36* (0.24, 0.48)	0.64* (0.54, 0.76)
Mother report			
Both sexes	0.26* (0.04, 0.49)	0.27* (0.08, 0.45)	0.46* (0.39, 0.55)
Teacher report			
Both sexes	0.20* (0.02, 0.46)	0.43* (0.20, 0.58)	0.37* (0.29, 0.47)
Proactive			
Child report			
Boys	0.50* (0.36, 0.60)	—[a] —[a]	0.50* (0.40, 0.64)
Girls	—[a] —[a]	0.14* (0.01, 0.26)	0.86* (0.74, 0.99)
Mother report			
Both sexes	0.32* (0.09, 0.55)	0.21* (0.02, 0.39)	0.47* (0.40, 0.56)
Teacher report			
Both sexes	0.45* (0.14, 0.62)	0.14* (0.01, 0.39)	0.41* (0.32, 0.53)

All aggression scales were transformed to rank normal scores within each rater.

A Genetic influences, C shared environmental influences, E non-shared environmental influences

[a] Parameter fixed at 0.0

* $p<0.05$

Table 7

Genetic and Environmental Correlations Between Reactive and Proactive Aggression

	r_A	r_C	r_E
Child report			
Boys	0.57*,** (0.38, 0.74)	— —	0.46*,** (0.33, 0.57)
Girls	— —	0.53*,** (0.15, 0.96)	0.53*,** (0.44, 0.61)
Mom report			
Both sexes	0.76* (0.28, 1.0)	0.76* (0.24, 1.0)	0.43*,** (0.33, 0.52)
Teacher report			
Both sexes	1.00* (0.64, 1.00)	1.00* (0.67, 1.00)	0.53*,** (0.41, 0.63)

* significantly greater than 0 ($p<0.05$)

** significantly less than 1.0 ($p<0.05$)