Genetic and Environmental Stability Differs in Reactive and Proactive Aggression

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The aim of this study was to examine stability and change in genetic and environmental influences on reactive (impulsive and affective) and proactive (planned and instrumental) aggression from childhood to early adolescence. The sample was drawn from an ongoing longitudinal twin study of risk factors for antisocial behavior at the University of Southern California (USC). The twins were measured on two occasions: ages 9–10 years (N = 1,241) and 11–14 years (N = 874). Reactive and proactive aggressive behaviors were rated by parents. The stability in reactive aggression was due to genetic and nonshared environmental influences, whereas the continuity in proactive aggression was primarily genetically mediated. Change in both reactive and proactive aggression between the two occasions was mainly explained by nonshared environmental influences, although some evidence for new genetic variance at the second occasion was found for both forms of aggression. These results suggest that proactive and reactive aggression differ in their genetic and environmental stability, and provide further evidence for some distinction between reactive and proactive forms of aggression. Aggr. Behav. 34:1–16, 2009. © 2009 Wiley-Liss, Inc.

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The function of aggressive behavior is related to the motive of the perpetrator, and it has been argued that a distinction can be drawn between reactive and proactive forms of aggression in both children and adults. Reactive aggression refers to angry or frustrated responses to a real or perceived threat. This specific type of aggression has been characterized as involving high emotional arousal, impulsivity, and an inability to regulate or control affect. On the contrary, proactive aggression is conceptualized as a more regulated, instrumental form of aggression, with more positive expectancies about the outcomes of aggression [Crick and Grotpeter, 1996; Dodge, 1991; Dodge and Coie, 1987; Schwartz et al., 1998]. Behavioral genetic studies have provided evidence that heritable factors are important for both reactive and proactive aggression [Baker et al., 2008; Brendgen et al., 2006]. However, the influence of genetic and environmental factors on continuity and change in the development of reactive and proactive aggression remains poorly understood.

Theoretical explanations for reactive aggression are derived from the frustration–aggression model, which considers aggression as a hostile, angry reaction to perceived frustration [Berkowitz, 1993]. An alternative theoretical perspective for understanding the etiology of proactive aggression is the social-cognitive learning theory by Bandura [1973, 1986], which posits that aggression is an acquired instrumental behavior that is controlled by an anticipated reward. The instigating factor is the expected success of the behavior rather than the punishment [Bandura, 1973, 1986]. Crick and Dodge [1996] have shown that children engaging in proactive aggression attach a positive value to the use of aggressive behavior when dealing with conflict resolution and peer group entry, thus providing some support for this theoretical perspective [Crick and Dodge, 1996].

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In contrast to those who argue for a distinction between reactive and proactive aggression, Bushman and Anderson [2001] have provided theoretical as well as conceptual arguments against this distinction. One of their arguments is that reactive and proactive aggression co-occurs, or in other words, there may be mixed motives behind an aggressive act. A distinction between the two is therefore not meaningful [Bushman and Anderson, 2001]. Although individual differences in use of reactive and proactive aggression have been found to be moderately to highly correlated [Card and Little, 2006], there is evidence that a distinction between them is valid [Fontaine, 2006; Kempes et al., 2005; Polman et al., 2007; Vitaro et al., 2006]. Both exploratory and confirmatory factor analyses have shown that reactive and proactive aggression are factorially distinct, based on item analyses of peer-, self-, parent-, and teacher-ratings as well as direct observation [Crick and Dodge, 1996; Poulain and Boivin, 2000a; Raine et al., 2006; Salmivalli and Nieminen, 2002]. Further evidence supporting a distinction between reactive and proactive aggression has been provided by studies showing different correlates of the two forms of aggression. Reactive aggression is related to earlier physical abuse, early onset of problems (i.e., attention problems and impulsiveness) [Barry et al., 2007; Dodge et al., 1997], peer rejection, victimization [Lamarche et al., 2003; Haberstick et al., 2006; McGue et al., 1994], leadership qualities, sense of humor [Dodge and Coie, 1987], and later delinquent behavior [Brendgen et al., 2001; Pulkkinen, 1996; Vitaro et al., 1998]. Hence, there are both theoretical grounds and empirical evidence suggesting that a distinction between reactive and proactive forms of aggression is meaningful.

The few longitudinal studies that have investigated the stability in reactive and proactive aggressive behavior have reported that both types of aggression are rather stable across time [McAuliffe et al., 2006; Salmivalli and Helteenvuori, 2007]. This has also been found for general aggressive behavior. Numerous studies have shown that aggressive behavior is a fairly common and stable trait that persists from childhood into adulthood [Loeber and Hay, 1997; Olweus, 1979; van Beijsterveldt et al., 2003]. There is also evidence of some changes in reactive and proactive aggression with age. For instance, a recent study found an increase in proactive aggression from late childhood to mid-adolescence in males. On the contrary, reactive aggression showed only a marginal increase with age in both males and females [Fung Lai-chu and Raine, submitted]. The prevalence of general aggressive behavior has been found to be highest in preschool years and decrease subsequently during adolescence [Loeber and Hay, 1997; Stanger et al., 1997; Tremblay, 2002a,b]. Related to this is that when children enter into adolescence both physical and emotional developmental changes occur and there is a shift from parents to peers as the primary social group [Berk, 1994]. Hence, it is therefore interesting to investigate whether the same genetic and environmental influences are important in reactive (proactive) aggressive behavior from childhood to adolescence, or whether any new genetic and environmental influences become important during teenage years. New genetic influences during adolescence could be expected as a result of physical maturation. That is, “new genes” could become expressed at a specific age, for example genes related to pubertal development [Jacobson et al., 2002]. Further, new environmental influences could be expected to arise as a function of new peer and school experiences as children move into adolescence.

Compared to a cross-sectional twin design, which gives absolute estimates of genetic and environmental influences on aggressive behavior at a specific age or a specific time point, a longitudinal twin design makes it possible to investigate how both genetic and environmental factors contribute to the process of stability and change across development. Stability/continuity in this context refers to genetic and/or environmental influences that remain stable across time, whereas change refers to “new” genetic and/or environmental influences that become important at a later time point. Several longitudinal twin studies have also been conducted to examine the stability and change of genetic and environmental influences in aggressive behavior. These studies have generally reported that the stability in aggressive behavior is largely due to genetic factors and that change is due to nonshared or unique environmental factors (i.e., experiences that make siblings dissimilar) [Eley et al., 2003; Haberstick et al., 2006; McGue et al., 1993; van Beijsterveldt et al., 2003]. However, none of the aforementioned studies distinguished between reactive and proactive aggressive behavior.

In this study, we investigated the genetic and environmental sources of reactive and proactive aggressive behavior in a longitudinal study at ages 9–10 and 11–14, with data from a large twin sample. Reactive and proactive aggressive behavior was
rated by parents. As previous twin studies examining the stability of aggressive behavior in children and adolescents have not, to the best of our knowledge, distinguished between reactive and proactive forms of aggression, it is unclear to what extent genetic or environmental influences account for the stability and change in these specific forms of aggressive behavior. We aimed to clarify this by examining the developmental changes in genetic and environmental factors in reactive and proactive aggression from middle childhood to early adolescence.

METHOD

Participants

The sample was drawn from participants in the University of Southern California (USC) Twin Study of Risk Factors for Antisocial Behavior. Detailed information regarding the design and recruitment procedures of the project have been provided elsewhere [Baker et al., 2006, 2007]. In brief, the Twin Study of Risk Factors for Antisocial Behavior is an ongoing prospective longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial and aggressive behavior from childhood to adolescence. The twins are evaluated using an extensive protocol, including cognitive, behavioral, psychosocial, and psychophysiological measures. The twins and their parents were recruited from the Los Angeles community and the sample is representative of the ethnic and socio-economic diversity of the greater Los Angeles area [Baker et al., 2007]. The sample consists of 616 families (607 twin pairs and 9 sets of triplets; total N = 1,241 children and N = 616 caregivers).

In the first wave of assessment during 2000–2004, the twins and their primary caregiver participated in a 6–8 hr laboratory assessment at USC. The twins were 9–10 years old (mean age = 9.6, SD = .58) and caregiver participation was primarily (92%) the biological mothers. In Wave-2 during 2003–2006, approximately two to three years following their Wave-1 assessment (mean = 2.15 years between Waves 1 and 2), the twins were 11–14 years old (mean age = 11.8, SD = .90). The response rate for youth self-report at Wave 2 was 74% [n = 837 children]. The response rate for parent-reports at Wave 2 was 78% (n = 874 children). In the follow-up assessment, 43% of the responding families came back to USC for testing and interviews and 57% responded to mailed questionnaires. No mean differences were found between those who came to USC for interviews and those who responded to mail survey (reactive aggression: t872 = –0.29, P = .77; proactive aggression: t872 = –1.87, P = .06).

This study used parent-ratings of child aggression in Waves 1 and 2. Only twin pairs with known zygosity and with data from parent ratings on aggressive behavior from at least one time point were included. The sample is comprised of the following zygosity groups: 283 MZ male twins, 174 DZ male twins, 284 MZ female twins, 197 DZ female twins, and 303 opposite-sex DZOS twins.

Zygosity was determined through DNA microsatellite analysis (>7 concordant and zero discordant markers = MZ; one or more discordant markers = DZ) for 87% of the same-sex twin pairs. For the remaining same-sex twin pairs, zygosity was established by questionnaire items about the twins’ physical similarity and the frequency with which people confuse them. The questionnaire was used only when DNA samples were insufficient for one or both twins. When both questionnaire and DNA results were available, there was a 90% agreement between the two [Baker et al., 2006, 2007].

Measures

To measure aggressive behavior in the twins, the Reactive and Proactive aggression Questionnaire (RPQ) completed by the parents was used. The RPQ is a validated 23-item questionnaire designed to measure reactive and proactive aggression in children and adolescents from the age of eight [Raine et al., 2006]. The RPQ includes 11 reactive items (e.g., “He/she damages things when he/she is mad”; “He/she gets mad or hit others when they tease him/her”) and 12 proactive items (e.g., “He/she threatens and bullies other kids”; “He/she damages or breaks things for fun”). The items in the RPQ have a three-point response format: 0 = never, 1 = sometimes, 2 = often. The mean item response was computed within groups of items to form reactive and proactive aggression subscales.

Confirmatory factor analysis using the RPQ in the Pittsburgh Youth Study [Loeber et al., 1998] has shown an acceptable fit for a two-factor reactive–proactive model that is superior to a one-factor model [Raine et al., 2006]. This has also been replicated using the current sample, with a two-factor reactive–proactive model providing a better fit than a one-factor model across informants at Wave 1 (parent-, teacher-, self-reported data) [Baker et al., 2008].

A factor analysis conducted on the current sample at Wave 2 using Mplus 5.0 [Muthén and Muthén,
1998–2007) also confirmed that the two-factor model was superior to the one-factor model. The \( \chi^2 \) difference test indicated a highly significant improvement in fit for the two-factor model over the one-factor model (\( \Delta \chi^2 = 460, df = 1, P < .001 \)). The overall goodness of fit indices suggested that the two-factor model fit was acceptable, with an RMSEA of .067 (90% CI: 0.063–0.071) and a CFI of .82, both were better than the one-factor model (RMSEA = .082, CFI = .73).

Further, at Wave 1, when the twins were 9–10 years old, 30 families (30 boys and 30 girls) were retested after six months to evaluate test–retest reliability for the reactive and proactive aggression measures used in this study. The test–retest reliability was adequate for both measures: reactive aggression \( r = .81 \), proactive aggression \( r = .79 \). The internal consistencies (Cronbach’s \( \alpha \)) of the two measures were also sufficient at both waves: reactive aggression: Wave-1: \( \alpha = .83 \), Wave-2: \( \alpha = .85 \); proactive aggression: Wave-1: \( \alpha = .77 \), Wave-2: \( \alpha = .76 \). Reactive and proactive aggression were moderately correlated as expected [Card and Little, 2006]: Wave-1: phenotypic correlation \( r = .59; P < .001 \); Wave-2: \( r = .58; P < .001 \). Prior to analysis, the subscales were ranked and normalized to reduce the positive skew in their distributions within each wave.

The majority of caregivers completed the original English version of the RPQ (81.3%). However, a Spanish version was developed specifically for this study and completed by caregivers who were not fluent in English (18.7%). At Wave-1, we found mean differences in reactive aggression (\( t_{494} = 3.56, P < .01 \)), with Spanish speaking caregivers reporting slightly less aggressive behavior in their children compared with ratings of children from English speaking (Hispanic) caregivers. As we found mean differences for the Spanish and English survey versions among Hispanic families, we also compared teacher ratings of reactive aggression in this group. Consistent with the caregivers, teachers also reported less reactive aggressive behavior in children of Spanish-interviewed caregivers (\( t_{277} = 2.16, P = .03 \)). This indicates that children in Hispanic families where the caregiver is not fluent in English may actually show less reactive aggressive behavior; hence the difference in aggressive behavior among these children is probably not related to the questionnaire being administered in different languages. Further, no mean differences were found in proactive aggression (\( t_{494} = 1.17, P = .24 \)) for those participants who completed the English and Spanish versions. At Wave-2, there were no mean differences in either reactive (\( t_{345} = 1.42, P = .16 \)) or proactive aggression (\( t_{345} = -0.18, P = .86 \)). The Spanish RPQ is available upon request from the fourth author.

**Attrition**

Selective attrition may bias estimates in longitudinal analyses [Heath et al., 1998]. We therefore tested whether twins and their families who did not respond to Wave-2 differed from responders on baseline measures at Wave-1. Logistic regression analyses showed nonsignificant odds-ratios (OR) for family socioeconomic status, based on the Hollingshead Four-Factor Index of Social Status [Hollingshead, 1979] (OR = 0.99, 95% Confidence Interval (CI): 0.98–1.01), and several characteristics of the children including twin’s sex (OR = 0.85, 95% CI: 0.66–1.08), general intelligence (verbal and spatial abilities) (OR = 0.93, 95% CI: 0.81–1.07), teacher reported general school performance (OR = 0.98, 95% CI: 0.94–1.02), reactive aggression (OR = 0.96, 95% CI: 0.60–1.52) and proactive aggression (OR = 1.14, 95% CI: 0.98–1.33).

However, comparisons of responders and non-responders revealed significant differences in ethnicity (OR = 1.35, 95% CI: 1.03–1.76), indicating that non-Caucasian and Hispanic families were slightly more likely to cease to participate.

**Statistical Analyses**

**Descriptive statistics and correlations.** Saturated models, which estimate the variances, covariances, and means of reactive and proactive aggression scores, were fit to the transformed RPQ subscales. As these summary statistics are obtained for each zygosity group, mean and variance differences between Twin 1 and Twin 2, between males and females, and between zygosity groups can be formally assessed through the comparison of various submodels. For example, mean differences between Twin 1 and Twin 2 were ascertained by comparing a model that estimated separate means for Twin 1 and Twin 2 with one that constrains means to be the same.

To get a first indication of the underlying sources of variance and the stability of reactive and proactive aggression, comparisons were made among within-person longitudinal correlations (phenotypic stability of reactive and proactive aggression at Waves 1 and 2), intraclass twin correlations (Twin-1–Twin-2 correlations within each wave) and cross-twin cross-age correlations (e.g., Twin-1 at Wave-1 with Twin-2 at Wave-2). For example, a dizygotic (DZ) intraclass correlation approximately half the value of the monozygotic (MZ) intraclass
correlation would indicate the presence of genetic effects within a given wave, whereas a DZ intraclass correlation more than half a MZ intraclass correlation indicates the presence of both genetic and shared environmental effects. The cross-twin cross-age correlations give information about the longitudinal stability of genetic and environmental effects in a trait across time. Cross-twin cross-age correlations are interpreted the same way as intraclass correlations—that is, greater values for MZ compared with DZ pairs suggest genetic stability for a particular aggression scale across the two waves. However, this is a descriptive approach, and formal modeling is necessary to test the significance of the observational inferences made from these correlations.

**Model-fitting analyses within waves.** The classical twin design is a natural experiment that relies on the different levels of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twins to estimate the relative contribution of genetic and environmental factors to individual differences in a phenotype of interest, in this case reactive and proactive aggression. The total phenotypic variance of a measured trait can be divided into additive genetic factors (A), shared environmental factors (C), and nonshared environmental factors (E). Since MZ twins are genetically identical, additive genetic factors are correlated 1.0. For DZ twins the genetic factors are correlated .5 as they on average share 50% of their segregating genes. Shared environmental factors refer to nongenetic influences that contribute to similarity within pairs of twins. Shared environmental influences are assumed to contribute equally to similarity in MZ and DZ twins, and thus shared environmental factors correlate 1.0 in both MZ and DZ twins. Nonshared environmental factors are those experiences that make siblings dissimilar. There is no correlation for the unique environment by definition, and this parameter also includes measurement error. Heritability is the proportion of total phenotypic variance due to genetic variation.

To assess the relative influence of genetic (A), shared environmental (C), and nonshared environmental (E) effects on proactive and reactive aggression at Wave-2, when the twins were 11–14 years old, a set of univariate five-group (MZ male, DZ male, MZ female, DZ female, DZ opposite sex) models were fit. Specifically, a model where the magnitude of genetic and environmental effects were allowed to differ, and a model in which the components of variance were constrained to be equal across the two sexes were fit [Neale and Cardon, 1992]. As base line comparison, a saturated model was used. These Wave-2 results were compared with model-fitting results on proactive and reactive aggression at ages 9–10 (Wave 1), which were previously reported for this sample [Baker et al., 2008].

**Longitudinal model fitting analyses.** In addition to the univariate Wave-2 genetic analyses, we conducted longitudinal analyses of both waves simultaneously. A five-group (MZ male, DZ male, MZ female, DZ female, DZ opposite sex) bivariate Cholesky decomposition model was applied to longitudinal data separately for reactive and proactive subscales to identify genetic, shared environmental and nonshared environmental effects that are stable across age, and new effects that arise Wave-2 [Neale and Cardon, 1992].

Finally, a five-group (MZ male, DZ male, MZ female, DZ female, DZ opposite sex) two-factor common pathway model was fit simultaneously to reactive and proactive aggression subscales at both waves. With this model it is possible to analyze reactive (Wave 1 and Wave 2) and proactive (Wave 1 and Wave 2) aggression simultaneously, to investigate the extent to which genetic and environmental effects may be shared or specific to each form of aggression (reactive and proactive), and time of assessment (Wave 1 and Wave 2). A two-factor common pathway model includes common genetic and environmental effects (A_C, C_C, and E_C) that influence two latent (unobserved) factors, which represent the two forms of aggression, i.e., reactive and proactive, which in turn influence all four observed measures in the model, i.e., two aggression subscales measured at each of two time points. The model also includes time- and form-specific genetic and environmental factors (A_S, C_S, and E_S) [McArdle and Goldsmith, 1990]. Simultaneous analysis of reactive and proactive aggression also allows further investigation into their shared relationship, and the extent to which they may have distinct or overlapping etiologies during childhood and adolescence.

Models were fit with the structural equation program Mx [Neale et al., 2003], using a maximum likelihood estimation procedure for raw data. This estimation technique can handle incomplete data, e.g., retain twins with data from just one time point, or one twin in a pair. Raw maximum likelihood yields a goodness of fit index called log-likelihood, which cannot be evaluated itself. Instead, the adequacy of fit is assessed by computing twice the difference between the log-likelihood of a full model and the log-likelihood of a submodel, in which parameters are fixed to be zero or constrained to be

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equal. This difference follows a $\chi^2$ distribution with the difference in the number of estimated parameters in the two models as the degrees of freedom. A significant $\chi^2$ indicates that the model with fewer parameters to be estimated fits the data worse. Second, the suitability of the models was also determined by comparing the model’s Akaike Information Criterion. The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values (i.e., larger negative) of AIC indicating the most suitable model [Akaike, 1987]. Finally, a third model-selection statistic was the Bayesian Information Criterion, where increasingly negative values correspond to increasingly better fitting models [Raftery, 1995].

RESULTS

Descriptive Statistics

Table I presents mean, standard deviation, and number of participants at Wave-1 and Wave-2. No significant mean or variance differences were found between Twin-1 and Twin-2. Nor were there any mean or variance differences between zygosity groups for proactive or reactive aggression at either of the two time points. Significant mean differences were found between males and females, for reactive aggression at both time points (Wave-1: $\chi^2 = 14.88$, df = 3, $P < .001$; Wave-2: $\chi^2 = 9.90$, df = 3, $P = .02$), with males showing higher scores than females.

Twin Correlations

Intraclass and cross-twin cross-age correlations for the reactive and proactive aggression are presented in Table II. All MZ intraclass correlations were higher than DZ intraclass correlations, suggesting genetic influences for both types of aggression. For example, the intraclass correlations for reactive aggression, Wave-1, were .48 for MZ males and .60 for MZ females. The corresponding numbers for DZ twins were lower, .35 and .46. There was no evidence for genetic nonadditivity (i.e., dominance or epistasis), as none of the MZ intraclass correlations exceeded twice the values of the DZ intraclass correlations for the same sex-twin pairs. All the DZ intraclass correlations were more than half the MZ intraclass correlations suggesting

| TABLE I. Number of Participants (N), Mean, and Standard Deviation (SD) for Reactive and Proactive Aggression, Ages 9–10 and 11–14, by Zygosity and Sex |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | Wave-1    |      | Wave-2    |      |      | Wave-1    |      | Wave-2    |      |
|                 | Reactive aggression | Mean | SD  | N   | Proactive aggression | Mean | SD  | N   | Reactive aggression | Mean | SD  | N   | Proactive aggression | Mean | SD  | N   |
| MZ male        | .70       | .33  | 283  | .09  | .15  | 283  | .60       | .32  | 205  | .07  | .12  | 205  |
| DZ male        | .75       | .36  | 174  | .14  | .22  | 174  | .68       | .39  | 110  | .13  | .21  | 110  |
| MZ female      | .61       | .30  | 284  | .08  | .13  | 284  | .49       | .31  | 197  | .07  | .12  | 197  |
| DZ female      | .65       | .36  | 197  | .09  | .16  | 197  | .63       | .39  | 155  | .09  | .14  | 155  |
| OS DZ          | .67       | .32  | 303  | .09  | .14  | 303  | .62       | .37  | 207  | .09  | .19  | 207  |

SD, standard deviation; N, number of participants; MZ, monozygotic; DZ, dizygotic; OS, opposite sex.

| TABLE II. Intraclass and Cross-Twin Cross-Age Correlations, by Sex and Zygosity |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | Males      |      | Females     |      |      | MZ |      | DZ |      | MZ |      | DZ |      | DZ OS |
|                 | Wave-1    |      | Wave-2    |      |      | Wave-1    |      | Wave-2    |      |
| Intraclass twin correlations$^a$ | .48* | .35* | .60* | .46* | .50* |
| Wave-2          | .49* | .33* | .58* | .38* | .42* |
| Cross-twin cross-age correlations | .20* | .32* | .31* | .22* | .20* |
| Wave-1–Wave-2   | Intraclass twin correlations$^a$ | .61* | .34* | .57* | .48* | .55* |
| Wave-2          | Intraclass twin correlations | .55* | .35* | .46* | .27* | .40* |
| Cross-twin cross-age correlations | .34* | .25* | .18* | .10  | .26* |

$^aP < .05$.

$^a$Wave-1 intraclass correlations for reactive and proactive aggression have previously been reported on these data (Baker et al., 2008).
shared environmental effects, apart from proactive aggression Wave-1 in males. MZ intraclass correlations were all less than one, which suggest influence of nonshared environment.

Cross-twin cross-age correlations, as shown in Table II, give a first indication of stability and change in genetic and environmental influences for reactive and proactive aggression across time. In most cases (except for MZ/DZ males’ reactive aggression), DZ correlations were greater than half MZ correlations, indicating that both genetic and shared environmental effects contribute to the stability in both forms of aggressive behavior. However, etiological patterns suggested by these twin correlations can be tested more formally using structural equations models.

Model-Fitting Analyses Within Each Wave

Model-fitting analyses for reactive and proactive aggression at age 9–10 have previously been carried out on these data [Baker et al., 2008]. These results are summarized in Table III, in order to facilitate comparisons to the analyses of Wave-2 and the longitudinal stability between the two waves. In brief, an ACE model constraining parameters to be equal across males and females fit the data best compared with the saturated model for both reactive and proactive aggression (reactive aggression: \(\chi^2 = 16.76, df = 12, P = .16\); proactive aggression \(\chi^2 = 14.84, df = 11, P = .20\)). For reactive (proactive) aggression 26% (in males) and 32% (in females) of the variance was due to genetic influences, 27% (in males) and 21% (in females) was explained by shared environmental influences, and the remaining 46% (in males) and 47% (in females) was due to nonshared environment.

Model-fitting results for reactive and proactive aggression at age 11–14 (Wave-2) are also displayed in Table III. For both reactive and proactive aggression a model constraining the relative influence of genetic and environmental factors to be equal in males and females fit the data best compared to the saturated model (reactive aggression: \(\chi^2 = 17.65, df = 12, P = .13\); proactive aggression \(\chi^2 = 11.61, df = 11, P = .40\)). The model constraining variance components to be equal for males and females also had a smaller AIC, indicating that it is a more parsimonious model, and a smaller BIC, indicating a better fit.

Genetic effects accounted for 43% of the variance in reactive aggression at age 11–14 (\(\chi^2<.05\)); 15% of the variance was due to shared environmental effects (nonsignificant), and the nonshared environment

### TABLE III. Model-Fitting Analyses Within Each Wave for Reactive and Proactive Aggression, at Ages 9–10 and 11–14 Years

<table>
<thead>
<tr>
<th>Wave</th>
<th>Overall fit</th>
<th>Model difference test</th>
<th>Parameter estimates (95% CI)</th>
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<tbody>
<tr>
<td></td>
<td>–2LL</td>
<td>df</td>
<td>AIC</td>
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<tr>
<td>Wave-1</td>
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<tr>
<td>Reactive aggression</td>
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<td>Proactive aggression</td>
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<tr>
<td>Males#Females</td>
<td>2,384.56</td>
<td>866</td>
<td>652.56</td>
</tr>
<tr>
<td>Males = Females</td>
<td>2,386.42</td>
<td>869</td>
<td>648.42</td>
</tr>
</tbody>
</table>

–2LL, \(-2\)log-likelihood); AIC, Akaike’s Information Criterion; BIC, Bayesian Information Criterion; \(\Delta\chi^2\), difference in log-likelihoods between nested models; \(\Delta df\), change in degrees of freedom.

Univariate results on reactive and proactive aggression, Wave-1 have previously been reported on these data (Baker et al., 2008).
accounted for the remaining 42% of variance in reactive aggression ($P < .05$). For proactive aggression, 48% of the variance was due to genetic effects ($P < .05$), 8% of the variance was due to shared environmental effects (nonsignificant), and the remaining 44% was due to the nonshared environment ($P < .05$). At a glance, genetic factors appeared slightly higher at the second time point compared with the first time point, whereas the shared environment effect slightly declined across age.

**Longitudinal Model-Fitting Analyses**

First, bivariate Cholesky decomposition models were used to assess the effects of stable and new genetic and environmental influences in reactive and proactive aggression from childhood to early adolescence. For both reactive and proactive aggression, a saturated model was used as a baseline to which the Cholesky decomposition was compared (see Table IV). For reactive aggression, a Cholesky decomposition equating genetic and environmental estimates to be equal in males and females provided a better fit of the data based on AIC and BIC criteria ($AIC = 1,245.84; BIC = -4,036.72$), and did not significantly differ from the saturated model ($\Delta \chi^2 = 73.01; \Delta df = 57; P = .08$). Similarly, for proactive aggression, a Cholesky decomposition equating genetic and environmental estimates to be equal in males and females fit the data better based on BIC ($AIC = 1,396.74; BIC = -3,961.27$), and did not significantly differ from the saturated model ($\Delta \chi^2 = 72.96; \Delta df = 57; P = .08$). This model estimates fewer parameters and is therefore more parsimonious, although the AIC was slightly smaller for the model estimating different parameters in the two sexes ($AIC = 1,394.52$). Further, mean differences between reactive aggression scores across the two time points were found ($\chi^2 = 54.80, df = 18, P < .001$), with mean values being lower at Wave 2.

No mean differences between proactive aggression scores across the two time points were found ($\chi^2 = 14.64, df = 18, P = .69$).

The total estimated genetic and environmental effects for reactive and proactive aggression at each time point can be obtained by summing the contributions of common and unique components (see Fig. 1a and b for reactive aggression and proactive aggression, respectively). For example, the estimated heritability in reactive aggression at Wave-1 is $(a_{11})^2 = .54^2 = .29$, and at Wave-2 $(a_{22})^2 + (a_{22})^2 = .48^2 + .49^2 = .47$. In general, the total genetic and environmental effects estimated for each measure in the bivariate model are consistent with those derived in each univariate genetic models. Slight variation in the parameter estimates is a result of additional information available in cross-twin cross-age covariance.

For reactive aggression, the phenotypic stability correlation was derived to be $r = .54$ [i.e., $(a_{11}a_{22}) + (e_{11}e_{22}) + (e_{11}e_{22}) = (.54*.48) + (.50*.12) + (.68*.32)$]. For proactive aggression this correlation was $r = .50 = (.58*.73) + (.45*.00) + (.68*.11)$. From the estimates presented in Figure 1a and b, it is also possible to calculate how much the phenotypic stability correlation is due to genetic influences, shared environmental and nonshared environmental influences.

For reactive aggression, 48% of the phenotypic stability correlation was explained by genetic factors: $(a_{11}a_{22})/r = (.54*.48)/.54 = .48$. The shared environment explained 11% of the stability, though nonsignificant. The remaining 41% of the phenotypic stability was due to the nonshared environ-

### TABLE IV. Bivariate Longitudinal Model-Fitting Results for Reactive and Proactive Aggression, at Ages 9–10 and 11–14 Years

<table>
<thead>
<tr>
<th>Model no.</th>
<th>Overall fit</th>
<th>Compared to model no.</th>
<th>Model difference test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$-2LL$</td>
<td>DF</td>
<td>AIC</td>
</tr>
<tr>
<td><strong>Reactive aggression</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1a Saturated model</td>
<td>5,372.83</td>
<td>2,043</td>
<td>1,286.83</td>
</tr>
<tr>
<td>1b Cholesky &amp; $e_{11}$</td>
<td>5,437.94</td>
<td>2,091</td>
<td>1,255.94</td>
</tr>
<tr>
<td>1c Cholesky: equate parameters in males and females &amp; $e_{11}$</td>
<td>5,445.84</td>
<td>2,100</td>
<td>1,245.84</td>
</tr>
<tr>
<td><strong>Proactive aggression</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2a Saturated model</td>
<td>5,523.78</td>
<td>2,043</td>
<td>1,437.78</td>
</tr>
<tr>
<td>2b Cholesky &amp; $e_{11}$</td>
<td>5,576.52</td>
<td>2,091</td>
<td>1,394.52</td>
</tr>
<tr>
<td>2c Cholesky: equate parameters in males and females &amp; $e_{11}$</td>
<td>5,596.74</td>
<td>2,100</td>
<td>1,396.74</td>
</tr>
</tbody>
</table>

$-2LL$, −2(log-likelihood); AIC, Akaike’s Information Criterion; BIC, Bayesian Information Criterion.
mental influences. Conversely, one can examine the extent to which longitudinal change in aggression may be due to genetic and environmental factors, by computing the ratio of each unique Wave-2 specific effect (i.e., new effects at Wave-2) to the total of the Wave-2 specific effects. For reactive aggression, the Wave-2 specific effects were due primarily (48%) to nonshared environmental influences [i.e., \(e_{22}^2/(a_{22}^2 + c_{22}^2 + e_{22}^2) = 0.56^2/(0.49^2 + 0.30^2 + 0.56^2) = 0.48\)], whereas the genetic (38%) and shared environmental (14%) effects were nonsignificant.

For proactive aggression, 85% of the stability was due to genetic factors, with the remaining 15% due to the nonshared environment. Conversely, the Wave-2 specific effects were due primarily to nonshared environmental influences (93%). Again, the genetic (0%) and shared environmental (7%) Wave 2 specific effects were nonsignificant.

While the bivariate longitudinal analyses indicated strong genetic stability for both reactive and proactive aggression, the separate analyses of each form of aggression do not address the extent to which their etiologies may overlap. Thus, a series of multivariate models were fit simultaneously to the reactive and proactive aggression scales at both waves, to investigate this etiological overlap. Specifically, the model specified two latent factors, one for each form of aggression (reactive and proactive), with the respective subscales at both time points loading onto each factor. Thus, each latent factor represents one form of aggression as measured at different time points in the two waves of assessment. The scale-specific effects (i.e., for each form of aggression at a single time point) represent genetic and environmental effects unique to each form of aggression during a particular assessment (Wave 1 or 2). The extent to which there are genetic or environmental influences unique to a given time or form of aggression can be investigated by examining these subscale-specific parameters.

Given the lack of sex differences in all univariate and bivariate models, a multivariate Cholesky model constraining male and female estimates to be equal was used as a baseline to which the two-factor common pathway was compared. The two-factor common pathway model (with separate but correlated latent factors for reactive and proactive forms of aggression) provided a better fit to the data according to BIC and AIC criteria (\(\text{AIC} = 2,093.27; \text{BIC} = -8,270.43\)), and did not significantly differ from the Cholesky model (\(\Delta \chi^2 = 5.13; \Delta \text{df} = 7; \ P = 0.64\)).

A series of reduced models were fit in order to simplify the two-factor common pathway model, see...
Table V. A model in which the common genetic factor was dropped from the two latent aggression factors was first fit, although it failed miserably (Model 2a, $\Delta \chi^2 = 18.77; \text{df} = 3; P < .001$). Next, a model dropping the common shared environmental factor was fit (Model 2b, $\Delta \chi^2 = 1.46; \text{df} = 3; P = .69$), and a model dropping both the common genetic and shared environmental factors (Model 2c, $\Delta \chi^2 = 131.53; \text{df} = 6; P < .001$). Thus, the shared environmental factor common to both reactive and proactive aggression could be dropped without a significant reduction in fit (Model 2b). The two-factor common pathway model could be further reduced by dropping time and measurement-specific genetic influences (proactive and reactive aggression, Wave 1) and time and measurement specific shared environment influences (reactive and proactive aggression, Wave 2) (Model 2d, $\Delta \chi^2 = 4.52; \text{df} = 7; P = .72$). Figure 2 displays standardized parameter estimates from this reduced two-factor common pathway model.

Squaring the standardized parameter estimates presented in Figure 2 provides the relative

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### Table V. Multivariate Longitudinal Model-Fitting Results for Reactive and Proactive Aggression, at Ages 9–10 and 11–14 Years

<table>
<thead>
<tr>
<th>Model no.</th>
<th>Overall fit</th>
<th>Model difference test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>−2LL</td>
<td>DF</td>
</tr>
<tr>
<td>1</td>
<td>Cholesky: equate parameters in males and females</td>
<td>10,486.14</td>
</tr>
<tr>
<td>2</td>
<td>2-Factor Common Pathway: equate parameters in males and females</td>
<td>10,491.27</td>
</tr>
<tr>
<td>2a</td>
<td>Drop common genetic factor</td>
<td>10,510.03</td>
</tr>
<tr>
<td>2b</td>
<td>Drop common shared environmental factor</td>
<td>10,492.72</td>
</tr>
<tr>
<td>2c</td>
<td>Drop common genetic and shared environmental factors</td>
<td>10,622.80</td>
</tr>
<tr>
<td>2d</td>
<td>Drop common shared environmental factor and time-specific genetic effects (reactive and proactive aggression, Wave 1) and time-specific shared environmental effects (reactive and proactive aggression, Wave 2)</td>
<td>10,495.79</td>
</tr>
</tbody>
</table>

−2LL, −2(log-likelihood); AIC, Akaike’s Information Criterion; BIC, Bayesian Information Criterion.

---

![Fig. 2. Standardized path estimates from a two-factor common pathway model for reactive aggression and proactive aggression in 9–10 and 11–14-year-old twins. Common additive genetic factors (A) and nonshared environmental factors (E) are depicted in circles. Ovals denote the two latent factors (i.e., Reactive Aggression and Proactive Aggression). Measured variables are depicted in rectangles. A_s: additive genetic residual variance specific to each measure, likewise for shared environment (C_s), and nonshared environment (E_s).](image-url)
contributions to the phenotypic variance for each form of aggression as derived from both waves of assessment. A common genetic factor explained 80% \((P < .05)\) of variance in the first latent factor (labeled Reactive Aggression) and 63% \((P < .05)\) of variance in the second latent factor (labeled Proactive Aggression). A nonshared environmental factor common to both Reactive and Proactive Aggression factors explained 20% \((P < .05)\) of variance in the Reactive Aggression latent factor and 37% \((P < .05)\) of variance in the Proactive Aggression latent factor.

There was a significant amount of time and measurement-specific variance for each of the variables (i.e., not all of the variance could be explained by the two latent aggression factors). For reactive and proactive aggression, Wave 1, there were time-specific shared environmental influences (reactive aggression: \(C_s = 13\%\); proactive aggression: \(C_s = 19\%\)). There were also some time-specific genetic influences \((A_s)\) for both types of aggression at Wave 2, explaining 26% in reactive aggression and 29% in proactive aggression. The residual variances also received contributions from non-shared environmental sources, explaining 38, 30, 25, and 18% of the variance in reactive aggression, Wave 1 and Wave 2, and proactive aggression Wave 1 and Wave 2, respectively. It should be noted that nonshared environmental influences on the specific subscales also include measurement error.

**DISCUSSION**

Parental ratings of reactive and proactive aggression in a large sample of twins were used to understand the origins of individual differences in reactive and proactive aggression and to estimate the genetic and environmental contributions to stability and change between childhood (age 9–10) and early adolescence (age 11–14). Reactive aggression ratings decreased on average across time, while proactive aggression remained stable in mean levels. Individual differences in reactive and proactive aggression, however, both showed moderate stability across time. Bivariate longitudinal analyses indicated that the stable variance in reactive aggression was mainly due to genetic and nonshared environmental influences, whereas the stable individual differences in proactive aggression were largely genetically mediated. When the two forms of aggression were examined separately in these bivariate models, the remaining (unstable) variance in both reactive and proactive aggression after accounting for the stable portion was mainly due to nonshared environmental influences.

Multivariate longitudinal analyses also showed that the stable portions of reactive and proactive aggression across time share common genetic and nonshared environmental factors. Nonetheless, evidence for etiological distinction between reactive and proactive aggression was found as indicated by significant time and scale specific effects. In fact, when considered jointly in the multivariate genetic models, unique genetic variance important to both reactive and proactive aggression was revealed during the later assessment in mid-adolescence (age 11–14 years old). Thus, the genetic distinction between these two forms of aggression appears to become even more important as children develop.

**Etiology of Individual Differences in Reactive and Proactive Aggression at Different Ages**

During childhood, when the twins were 9–10 years old, heritability explained 26% of the total variance in reactive aggressive behavior, and 32% of the total variance in proactive aggressive behavior. Shared environmental influences explained approximately one quarter each in reactive and proactive aggression [Baker et al., 2008]. In pre- and early adolescence, when the twins were 11–14 years old, heritability increased for both forms of aggression and accounted for approximately half of the variance, while shared environmental influences decreased and only explained 15% in reactive aggression and 8% in proactive aggression. Our findings contrast somewhat to the findings in a recent study on reactive and proactive aggression in six-year-old twin pairs, which reported a heritability estimate of approximately 40% in both aggression dimensions, but no shared environmental influences [Brendgen et al., 2006]. Our higher estimate of shared environmental influences at age 9–10 may be due in part to rater bias, since the same caregiver provided ratings of both twins in this study, while Brendgen et al. [2006] used teacher-rated aggression and only included cases having two different teacher-provided ratings for each twin.

The overall pattern in our analyses within each wave for both reactive and proactive aggressive behavior indicated that genetic influences become increasingly more important and shared environmental effects decreased in importance as children develop from middle childhood to early adolescence. This pattern of decrease in shared environment and a concomitant increase in heritability during development is relatively common for personality traits and cognitive abilities [Bartels et al., 2002; Loehlin,
reactive aggressive children are relatively friendless whereas proactive aggressive friends, whereas Boivin (200b) found that proactive aggressive behavior at both time points was due to nonshared environmental influences. Nonshared environmental effects may include antisocial experiences unique to the individual and not shared by his or her co-twin. Differential experiences with peer environments by for example choosing antisocial peers. However, with the design of this study, such evocative and active gene–environment correlations end up as part of the heritability estimate, and may explain some of the increasing genetic influences.

It is also possible that part of the shared environment reflects parental influences. As children grow older, parental influences probably become weaker and this could explain the decline in shared environmental influences. That is, it may be easier for parents to control or influence their children’s aggressive behavior at a younger age. Many children also transit from elementary to middle school settings by the Wave-2 testing, so that common experiences for two twins may be correspondingly less by age 11–14.

We also found that approximately 40% of the total variance common to reactive and proactive aggressive behavior at both time points was due to nonshared environmental influences. Nonshared environmental effects may include antisocial experiences unique to the individual and not shared by his or her co-twin. Differential experiences with peer group thus might be a more likely source of nonshared environmental influences on reactive and proactive aggression. For example, Poulin and Boivin (200b) found that proactive aggressive children have proactive aggressive friends, whereas reactive aggressive children are relatively friendless [Poulin and Boivin, 200b].

Etiology of Stability and Change in Reactive and Proactive Aggressive Behavior Across Time

Univariate analyses at each of the two time points at first glance seem to suggest that reactive and proactive aggression are difficult to distinguish in their genetic and environmental etiology, since the patterns of relative influence are similar for both forms of aggression. On the other hand, longitudinal analysis incorporating data from both childhood and adolescence reveal important differences in the developmental courses of the two forms of aggression, in that greater genetic stability was evident for proactive than reactive aggression. Moreover, mean values decreased from ages 9–10 to age 11–14 years for reactive aggression, but no mean differences were found between proactive aggression scores across the two time points, again suggesting greater stability (phenotypically) for proactive than reactive aggression. It is possible that the average decline in reactive aggression across time is related to developmental changes. That is, as a child grows older, he or she is better able to regulate his/her emotions and gain better stability and self-control [Harpur and Hare, 1994]. In keeping with several other studies on aggressive behavior [McGue et al., 1993; Stanger et al., 1997; van Beijsterveldt et al., 2003], reactive and proactive aggressive behaviors showed a moderate phenotypic stability from ages 9–10 to 11–14 years.

Specifically, the bivariate longitudinal analyses revealed that genetic and nonshared environmental influences contributed to the stability in reactive aggression. This finding is in agreement with a previous study on general aggressive behavior [van Beijsterveldt et al., 2003]. The genetic stability may partly explain why one particular child may have lower or higher reactive aggression scores than their peers at a given point in time, and also why the same child may persist in reactive aggression from childhood to early adolescence. The continuity in reactive aggression was mediated by nonshared environmental influences. Aspects of the nonshared environment include for example being continuously exposed to harsh and threatening environments, i.e., peer victimization and rejection, poor and harsh parenting, factors that have been shown to be related to reactive aggressive behavior [Dodge et al., 1997; Lamarche et al., 2007; Salmivalli and Helteenvuori, 2007; Schwartz et al., 1998].

The bivariate longitudinal analyses results also showed that the stability of proactive aggression was accounted for almost entirely by genetic factors. This finding is in line with other longitudinal twin
studies on aggressive behavior, which have not distinguished between proactive and reactive forms [Eley et al., 2003; Haberstick et al., 2006; McGue et al., 1993]. This suggests that genetic influences on proactive aggressive behavior in childhood either creates a series of events that lead to continued proactive aggressive behavior in adolescence, or that these genes continue to influence proactive aggressive behavior directly across ages 9–10 and 11–14. It is also possible that proactive aggressive behavior is related to other stable and heritable personality characteristics and that this to some extent explains the genetic stability seen in proactive aggression. For instance, highly heritable psychopathic and callous and unemotional traits [Viding et al., 2005] are theoretically linked to proactive aggression. That is, psychopathic and callous and unemotional traits encompass a disregard for others, which could translate to aggression toward others for personal gain [Barry et al., 2007; Frick et al., 2003; Kempes et al., 2005]. Psychopathic traits have also been found to be related to earlier proactive but not reactive aggression [Raine et al., 2006].

The multivariate longitudinal analyses revealed a common genetic and nonshared environmental overlap between reactive and proactive aggressive behavior across time. However, not all genetic and environmental influences were in common between reactive and proactive aggression. This was indicated by the time- and measurement-specific effects. Time- and measurement-specific effects would suggest that these behaviors are somewhat independent in their underlying genetic and environmental liability. Consistent with the univariate analyses within each wave, shared environmental effects were important at ages 9–10 and genetic influences were important at ages 11–14 years.

The bivariate longitudinal analyses also provided evidence of “new” nonshared environmental effects at ages 11–14 years, which could account for individual changes in aggression over time. The nonshared environmental part of the change in reactive and proactive aggression scores could be the result of a socialization process influenced by peers, teachers, and other significant persons [Swaim et al., 2006]. The combination of genetic influences on continuity and nonshared environmental influences on change has previously been reported in other studies on child psychopathology [Haberstick et al., 2005; O’Connor et al., 1998b] including aggressive behavior [Eley et al., 2003]. It should also be mentioned that nonshared environmental effects are confounded with measurement error.

The multivariate longitudinal analyses showed time-specific genetic influences at ages 11–14 years. Time-specific genetic influences in this age group could in part be the result of a developmental trajectory that could be linked to the onset of puberty [Jacobson et al., 2002]. Related to this, several studies including animal models have shown a relationship between testosterone and aggression. It has also been shown that the relationship between testosterone concentrations and future aggression may be causal. A study of castrated male mice on low testosterone replacement found that those receiving a testosterone injection after a successful aggressive encounter were more aggressive in subsequent encounters compared with those that received a saline injection after a successful aggressive encounter [Trainor et al., 2004]. Studies including human subjects have also shown a relationship between testosterone levels and aggression. Recently, a study reported that testosterone concentrations after a competitive interaction predicted future reactive aggression in men [Carré et al., 2009].

Limitations of This Study

Some limitations of the study need to be highlighted. First, this study relied solely on parental reports of their children’s aggressive behavior. While child self-reports may also be informative at these ages [see Baker et al., 2008 for a comparison of child, parent, and teacher ratings during the first assessment], children’s self-rated aggressive behavior appears less stable between ages 9–10 and 11–14 years old (reactive aggression: \( r_p = .30 \); proactive aggression: \( r_p = .19 \) between the two assessments) compared with parent reports in this sample. Moreover, genetic and shared environmental influences were nonsignificant for a smaller sample for which child reports were available during the second assessment at age 11–14 for both reactive and proactive aggression and consequently the genetic and shared environmental mediation of stability were both nonsignificant. Hence, it was therefore difficult to draw any firm conclusion from the longitudinal child self-report data.

A second limitation may be attrition. From Wave-1 when the twins were 9–10 years old, to Wave-2 when the twins were 11–14 years old, several subjects refused to participate in the follow-up assessment or dropped out of the study entirely. A possible explanation to this may be that as children grow older and enter into adolescence, school work, peers, and leisure activities might take up more of their time and they might be more likely...
to refuse further participation. We performed some analyses to test for selective attrition. Even though all odds ratios were nonsignificant, apart from ethnicity, it is possible that those dropping out include a disproportionate percentage with psychopathology. Thus, it is not certain that all the results are generalizable to individuals with the most extreme externalizing behaviors.

Other limitations are related to some of the basic assumption in the twin design. For example rater bias, which occurs when parents either stress the similarities (or siblings imitating each other’s behavior) or differences (or siblings taking on competing behaviors) in their children. If there is rater bias or true sibling imitation/contrast effects, variance differences between MZ and DZ twins are expected. An imitation sibling effect is confounded with shared environmental influences, and a contrast sibling effect is confounded with dominant effects [Bartels et al., 2004; Rietveld et al., 2003a,b; Vierikko et al., 2003, 2004]. We found for both types of aggression shared environmental influences, in particular when the twins were 9–10 years old; hence it is possible that part of the shared environment is due to rater bias.

Furthermore, it is generally assumed in twin models that random mating occurs in the parent generation. Assortative mating tends to increase similarity between DZ twins, thereby bias the heritability estimates downward and the shared environmental estimates upward. Assortative mating for most personality traits has been found to be low in magnitude [Maes et al., 1998], the exception is however antisocial behavior [Krueger et al., 1998; Taylor et al., 2000]. It is possible that part of the shared environment seen in reactive and proactive aggression is due to positive assortment.

Despite these limitations, this study adds to the existing literature on aggressive behavior in several important ways. The overall pattern that emerged from our analyses at each of the two time points for both forms of aggression was that genetic influences become increasingly more important and shared environmental effects decreased in importance as children grew up from middle childhood to early adolescence. Moreover, the longitudinal data from 9–10 to 11–14 years of age showed that continuity in reactive aggressive behavior resulted from genetic and nonshared environmental influences, whereas continuity in proactive aggression was primarily influenced by genetic factors. New effects in reactive and proactive aggression were mainly due to nonshared environmental influences, although there was some evidence of new genetic effects for both reactive and proactive aggression at the second wave of assessment. The findings in this study provide further evidence for some distinction between reactive and proactive forms of aggression.

ACKNOWLEDGMENTS

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Aggr. Behav.


Aggr. Behav.


