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Examining Genetic and Environmental Effects on Reactive Versus Proactive Aggression

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This study compared the contribution of genes and environment to teacher-rated reactive and proactive aggression in 6-year-old twin pairs (172 pairs: 55 monozygotic girls, 48 monozygotic boys, 33 dizygotic girls, 36 dizygotic boys). Genetic effects accounted for 39% of the variance of reactive aggression and for 41% of the variance of proactive aggression. The remainder of the variance was explained by unique environmental effects. Genetic as well as unique environmental effects were significantly correlated across reactive and proactive aggression (genetic correlation = .87, environmental correlation = .34), but this overlap was largely due to a common underlying form of aggression (i.e., teacher-rated physical aggression). Once common etiological factors due to physical aggression were accounted for, reactive and proactive aggression shared no other genes and only a few environmental influences, although additional specific genetic and environmental effects were observed for both reactive and proactive aggression. These specific effects indicate that both reactive and proactive aggression may be influenced mostly by socialization experiences that are specific to each type of aggression and only to a very small degree by specific genes.

Keywords: twins, proactive aggression, reactive aggression, behavioral genetics

For almost 2 decades, researchers have emphasized the distinction between two types of aggressive behavior on the basis of their underlying function or motivation: proactive and reactive aggression (e.g., Dodge & Coie, 1987). Proactive aggression, which has been described as instrumental, offensive, and cold-blooded, requires neither provocation nor anger. In contrast, reactive aggression has been described as affective, defensive, and hot-blooded, involving angry outbursts in response to actual or perceived provocations or threats. Proactive and reactive aggression often cooccur, and continuous proactive and reactive aggression scores correlate on average at $r = .70$ ($\pm .15$) in variable-centered studies (for a review, see Vitaro & Brendgen, 2005). Similarly, person-centered studies show that around one half of the children who engage in some form of aggressive behavior are both proactively and reactively aggressive, whereas around one third are only reactively but not proactively aggressive, and few children, around 15%, seem to engage in only proactive but not in reactive aggression (Dodge, Lochman, Harnish, Bates, & Pettit, 1997; Pulkkinen, 1996; Vitaro, Gendreau, Tremblay, & Oligny, 1998). Despite the high overlap between the two types of aggression, exploratory and confirmatory factor analyses show that reactive and proactive aggression are factorially distinct (Brown, Atkins, Osborne, & Milmanow, 1996; Crick & Dodge, 1996; Day, Bream, & Paul, 1992; Dodge & Coie, 1987; Pellegrini, Bartini, & Brooks, 1999; Poulin & Boivin, 2000a; Salmivalli & Nieminen, 2002).

To account for the conceptual and factorial distinctiveness of reactive and proactive aggression, Dodge (1991) proposed a theoretical model according to which reactive and proactive aggression originate from different socialization experiences. Specifically, this model postulates that reactive aggression develops in reaction to a harsh, threatening, and unpredictable environment such as, for example, abusive and cold parenting. Conversely, proactive aggression thrives in "supportive" environments that foster the use of aggression as a means to achieve one's goals. In line with the theoretical model proposed by Dodge, parents of reactively aggressive children have been found to be more controlling and punitive than parents of proactively aggressive or nonaggressive children (Bowen & Vitaro, 1998). Moreover, reactively aggressive children show histories of physical abuse,

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whereas their proactively-only and proactively–reactively aggressive counterparts do not (Dodge et al., 1997). In contrast, proactively aggressive youth seem to enjoy rather positive family relations compared with their reactively aggressive counterparts, although at the same time they report less parental monitoring and fewer household rules than do either the reactively aggressive group or the nonaggressive group (Poulin & Boivin, 2000b). When one investigates the effects of environmental factors on the etiology of a behavior, however, the findings are difficult to interpret when only one child per family is assessed. For example, the links between a putative environmental variable such as hostile parenting behavior and reactive aggression in the child may in fact be due to the genetic transmission of problem behaviors (DiLalla, 2002; Rhee & Waldman, 2002).

As argued by several researchers (e.g., Jaffee et al., 2005; Partridge, 2005), to analyze genetic effects on aggressive behavior with real precision one would have to measure the genotype. Unfortunately, functional genes for aggressive and antisocial behavior have yet to be found, although some promising candidate genes have been identified (e.g., Caspi et al., 2002). Moreover, genetic risk for a complex social behavior such as aggression likely involves multiple genes (Plomin & Crabbe, 2000). If the actual multiple genes are unknown, data from monozygotic (MZ) and dizygotic (DZ) twin pairs can be used to estimate the heritability of the behavior of interest (DiLalla, 2002; Jaffee et al., 2005). Specifically, the comparison of the phenotypic similarity of MZ twins, who are genetically identical, with the phenotypic similarity of same-sex DZ twins, who on average share only half of each other's genes, makes it possible to estimate the relative contribution of genetic factors, shared environmental factors (e.g., the neighborhood or family environment), and nonshared environmental factors (e.g., different friendship networks of the two twins in a pair) to the phenotypic similarity between twins (Neale & Cardon, 1992). On the basis of a genetically informed approach using a twin design, the first goal of the present study was to examine the relative contribution of genetic and (shared and nonshared) environmental factors to proactive aggression and reactive aggression, respectively. The second goal was to investigate an etiological mechanism that might explain the positive correlation between the two types of aggression.

Etiology of Proactive and Reactive Aggression: Genetic and Environmental Influences

Several twin studies have examined the heritability of aggression in children, particularly in regard to physical aggression (for reviews, see DiLalla, 2002; Miles & Carey, 1997; Rhee & Waldman, 2002). Although the estimates vary depending on the particular measure used and the age range of the sample, the results suggest that about 50% of the variance of physical aggression is determined by genes (approximate range between 40% and 80%). The remainder of the variance of physical aggression seems to be influenced mainly by unique or nonshared environmental factors, whereas only a relatively small and often nonsignificant contribution of environmental sources shared between twins has been reported in the literature. Most twin studies are based on mothers' or averaged parental ratings of child aggressive behavior, but similar results have been obtained with the juxtaposition of different reporting sources such as mothers and fathers (Van den Oord, Boomsma, & Verhulst, 2000), teachers and peers (Brendgen et al., 2005), or parents, teachers, independent observers, and child self-reports (Arseneault et al., 2003). Existing evidence also suggests that males and females do not seem to differ in terms of the relative magnitude of genetic and environmental effects on their aggressive and antisocial behavior (Rhee & Waldman, 2002; but see Miles & Carey, 1997, for contradictory findings).

Despite the wealth of twin studies on childhood aggressive behavior, no twin study so far has distinguished between proactive and reactive aggression, although scholars have criticized the lack of knowledge in this context (e.g., DiLalla, 2002). At least theoretically, it is conceivable that especially reactive aggression is determined not only by environmental factors but also to a considerable extent by genetic factors. Support for this notion comes from findings that reactive aggression shows significant and specific links to temperamental characteristics (Vitaro, Brendgen, & Tremblay, 2002), which are themselves highly heritable (Cyphers, Phillips, Fulker, & Mrazek, 1990). For example, reactive but not proactive aggression is associated with a temperamental disposition toward anxiety, angry reactivity, emotional disregulation, and inattention (Dodge & Coie, 1987; Dodge et al., 1997; Price & Dodge, 1989; Shields & Cicchetti, 1998; Vitaro et al., 2002). In addition, specific physiological correlates have been found for reactive aggression, thus providing further support for the possibility of a biological—and potential heritable—basis of reactive aggression. Thus, only reactively, but not proactively, aggressive children show elevated levels of skin conductance during stress (Hubbard et al., 2002). Together, these findings are in line with the view of reactive aggression as an intense temperament-based response to an aversive stimulus.

Similar to reactive aggression, proactive aggression may show not only strong environmental influences but also some heritability. This view is in line with the conceptualization of proactive aggression as an expression of underlying psychopathic characteristics, especially callous–unemotional traits. Callous–unemotional traits refer to a specific affective (e.g., absence of guilt, constricted display of emotion) and interpersonal (e.g., failure to show empathy, use of others for one's own gain) style that is related specifically to proactive but not reactive aggression in both adults (Cornell et al., 1996) and children (Frick, Cornell, Barry, Bodin, & Dane, 2003). Temperamentally, aggressive children with callous–unemotional traits are less reactive than are other aggressive children to threatening and emotionally distressing stimuli (Blair, 1999; Loney, Frick, Clements, Ellis, & Kerlin, 2003) and are less sensitive to cues of punishment when a reward-oriented response set is primed (Barry et al., 2000). This temperamental style is characterized physiologically by underreactivity in the sympathetic nervous system (Kagan & Snidman, 1991), which is markedly distinct from the previously mentioned physiological correlates observed for reactive aggression.

Taken together, there is thus considerable evidence for the notion that both reactive aggression and proactive aggression may be determined not only by environmental influences but also to a significant extent by genetic factors. By the same token, the findings from previous studies show that the physiological, temperamental, and environmental correlates of reactive and proactive aggression seem to differ considerably. The existing evidence thus suggests that the genetic and environmental factors contributing to reactive and proactive aggression, respectively, should be rather

specific to each type of aggression and not show too much overlap. The question therefore arises as to which etiological mechanism explains the positive correlation between the two behaviors.

Etiology of Proactive Versus Reactive Aggression: Explaining the Overlap Between the Two Types of Aggression

In light of the distinct individual and environmental correlates of reactive and proactive aggression, some researchers have suggested that the strong correlation between proactive and reactive aggression might be because the commonly used measures of reactive and proactive aggression such as the items developed by Dodge and Coie (1987) tap into the same form of aggressive behavior, specifically physical aggression (Little, Jones, Henrich, & Hawley, 2003). For example, the proactive aggression scale of the Dodge and Coie instrument includes items such as, "This child uses (or threatens to use) physical force to dominate other children," and the reactive aggression scale includes items such as, "When this child is teased or threatened, he/she gets angry easily and strikes back." Confirmatory factor analysis based on self-reports of aggressive behavior in children in Grades 5–10 supported Little et al.'s (2003) proposition that a common underlying form explains the comorbidity of proactive and reactive aggression. Specifically, these authors showed that when taking into account any confounds between proactive and reactive aggression that are due to a similar form of aggression, proactive and reactive aggression are basically uncorrelated.

On the basis of findings reported by Little et al. (2003), it is thus conceivable that reactive and proactive aggression are indeed to a significant extent determined by the same (i.e., correlated) genetic and environmental factors. The overlap between the genetic and environmental factors influencing proactive and reactive aggression, however, should be because proactive aggression and reactive aggression share a common underlying form, notably physical aggression. In other words, the genetic and environmental factors that are shared by proactive and reactive aggression should be the same ones that also influence physical aggression. Hence, common etiological factors due to a common underlying form such as physical aggression should completely explain any overlap that can be observed between the genetic and environmental factors influencing reactive and proactive aggression.

Apart from overlapping etiological features due to the same underlying form of aggression, however, both reactive and proactive aggression should also be determined by additional genetic and environmental factors that are not related to a common underlying form. Moreover, to the extent that a common underlying form can entirely explain the overlap between proactive and reactive aggression, these additional genetic and environmental factors should be uncorrelated and thus be specific to proactive aggression and reactive aggression, respectively. This notion is based on the previously mentioned findings that, despite their strong correlation, reactive and proactive aggression have different temperamental and physiological correlates and are predicted by different familial contexts. It is these distinct biological and social influences on proactive and reactive aggression that should be reflected by the expected additional, uncorrelated genetic and environmental factors. The notion of additional, uncorrelated genetic and environmental factors influencing proactive and reactive aggression is further supported by the fact that the correlation between reactive and proactive aggression usually varies between $r = .60$ and $r = .85$ (Vitaro & Brendgen, 2005). In other words, at least a quarter, if not more, of the interindividual variability in reactive and proactive aggression should be explained by genetic and/or environmental factors that are not associated with any common underlying form of aggression and that are specific to proactive and reactive aggression.

Objectives of the Present Study

In summary, our goal in the present study was to examine the following questions: (a) To what extent is the variance of reactive aggression and proactive aggression, respectively, explained by genetic, shared environmental, or nonshared environmental factors? (b) Can the covariance between reactive aggression and proactive aggression be explained by underlying genetic, shared environmental, or nonshared environmental factors that are related to a common underlying form such as physical aggression? (c) What proportion of variance of proactive and reactive aggression is explained by genetic or environmental factors that are not associated with any common underlying form of aggression and that are specific to proactive and reactive aggression? These questions were addressed with the most frequently used reporting source of reactive and proactive aggression, namely teacher ratings. In contrast to peer ratings, teacher ratings have been shown to produce a clear factorial distinction between reactive and proactive aggression (Poulin & Boivin, 2000a). In addition, teacher ratings of reactive and proactive aggression show good stability as well as good construct and predictive validity based on concurrent and longitudinal links with related antisocial behaviors and peer difficulties (Vitaro & Brendgen, 2005). Measures were assessed in children of kindergarten age, the youngest age when the distinction between reactive and proactive aggression has been clearly identified in previous research (Lansford, Dodge, Pettit, & Bates, 2002), thus providing information about the genetic and environmental influences on the two types of aggression at the earliest developmental period possible.

Method

Sample

Participants in the present study were part of an ongoing longitudinal study (Quebec Newborn Twin Study) of a population-based sample of twins from the greater Montreal area who were recruited at birth between November 1995 and July 1998 ($N = 648$ twin pairs). Zygosity was assessed at the age of 18 months on the basis of physical resemblance via the Zygosity Questionnaire for Young Twins (Goldsmith, 1991). For a subsample of these twin pairs ($n = 123$), a DNA sample was evaluated with respect to 8–10 highly polymorphous genetic markers (Forget-Dubois et al., 2003). The comparison of zygosity based on the similarity of these genetic markers with zygosity based on physical resemblance revealed a 94% correspondence rate, which is similar to rates obtained in older twin samples. Eighty-four percent of the families were of European descent, 3% were of African descent, 2% were of Asian descent, and 2% were Native North Americans. The remaining families (9%) did not provide ethnicity information. The demographic characteristics of the twin families were compared with those of a sample of single births that is representative of the large urban centers in the province of Quebec (SantéQuébec, Jetté, Desrosiers, & Tremblay, 1998) when the children were 5 months of age.

The results showed that the same percentage (95%) of parents in both samples lived together at the time of birth of their child(ren); 44% of the twins compared with 45% of the singletons were the firstborn children in the family; 66% of the mothers and 60% of the fathers of the twins were between 25 and 34 years of age compared with 66% of the mothers and 63% of the fathers of the singletons; 17% of the mothers and 14% of the fathers of the twins had not finished high school compared with 12% and 14% of the parents of the singletons; the same proportion of mothers (28%) and fathers (27%) in both samples held a university degree; 83% of the twin families and 79% of singleton families held employment; 10% of the twin families and 9% of the singleton families received social welfare or unemployment insurance; and finally, 30% of the twin families and 29% of the singleton families had an annual total income of less than $30,000, 44% (42%) had an annual total income between $30,000 and $59,999, and 27% (29%) had an annual total income of more than $60,000. These results indicate extremely similar sociodemographic profiles in the twin sample and the representative sample of single births.

The sample was followed longitudinally when the twins were age 5, 18, 30, 48, and 60 months, and the researchers focused on a variety of child-related and family-related characteristics. A sixth wave of data collection was completed when children were 6 years of age to assess their social adaptation in kindergarten. The present article describes findings from this latest wave of data collection. The average age at assessment was 72.7 months (3.6 *SD*). Attrition in the sample averaged at approximately 7% per year, resulting in a total of 366 twin pairs for the data collections at age 6 years (MZ males = 72, MZ females = 79, DZ males = 51, DZ females = 49, DZ mixed sex = 115). However, because estimation of genetic models rests mainly on the comparison of MZ and same-sex DZ twins (Neale & Cardon, 1992), mixed-sex DZ pairs were not included in the analyses. Twins remaining in the study at 6 years of age did not differ from those excluded at 5 months of age in regard to zygosity status or parent-rated temperament. The excluded twin families also did not differ from the remaining twin families in regard to any of the sociodemographic measures mentioned previously except that fathers in the remaining study sample had a slightly higher level of education than fathers of the twins who were excluded from the study.

Measures and Procedure

All instruments were administered in either English or French, depending on the language spoken by the kindergarten teachers (see description of measures below). Following the procedure suggested by Vallerand (1989), instruments that were administered in French but were originally written in English were first translated into French and then translated back into English. Bilingual judges verified the semantic similarity between the back-translated items and the original items in the questionnaire. The research questions and instruments were approved by the International Review Boards of the University of Quebec at Montreal and the University of Montreal, as well as the school board administrators. Prior to data collection, active written consent was obtained from the parents. When children were age 72 months, data collection took place in the spring of the kindergarten year, to ensure that the teachers had gotten to know the children in their class. In 249 (68%) out of the 366 twin pairs, the two twins did not attend the same classroom, whereas in 117 (32%) pairs, the two twins attended the same classroom.

Children's levels of reactive and proactive aggression were assessed through the most widely used instrument in this regard, namely teacher ratings using six items based on the proactive and reactive aggression items developed by Dodge and Coie (1987). A fourth reactive aggression item was added to the original scale developed by Dodge and Coie: "Reacts in an aggressive manner when contradicted." This item was added to assess the extent to which children behave reactively aggressively even in a rather benign, less provocative context. The Dodge and Coie teacher-rated measures of proactive and reactive aggression have shown very good external validity in previous studies, as indicated by positive correlations with direct observations of the same types of aggressive behavior (Dodge & Coie, 1987), by distinct relations with early reactive temperament (Vitaro et al., 2002), and by distinct social cognitive processes (Dodge & Coie, 1987; Dodge et al., 1997; Orobio de Castro, Merk, Koops, Veerman, & Bosch, 2005). Moreover, these teacher-rated measures of reactive and proactive aggression have been shown to differentially predict adjustment outcomes including delinquent behavior and depression in adolescents (Brendgen, Vitaro, Tremblay, & Lavoie, 2001; Vitaro et al., 1998, 2002). Responses were given on a 3-point scale (0 = *never,* 1 = *sometimes,* 2 = *often*). For each type of aggression, the respective scores were averaged to yield a total reactive aggression score ($M = 1.33$, $SD = 1.95$) and a total proactive aggression score ($M = 0.68$, $SD = 1.10$). Internal consistency of the total scales was acceptable in the present sample with Cronbach's $\alpha = .88$ for teacher-rated reactive aggression and Cronbach's $\alpha = .72$ for teacher-rated proactive aggression.

Children's level of physical aggression was rated by teachers using three items based on the Preschool Behavior Questionnaire (Behar & Stringfield, 1974). Specifically, the teachers indicated to what extent the child "gets into fights," "physically attacks others," and "'hits, bites, or kicks others"; responses were also given on a 3-point scale (0 = *never,* 1 = *sometimes,* 2 = *often*). This instrument has been shown to possess good criterion validity and high interrater and test–retest reliabilities in both normal and clinical samples (Behar & Stringfield, 1974). Individual item scores were summed up to yield a total physical aggression score (Cronbach's $\alpha = .88$, $M = 0.79$, $SD = 1.39$).

Results

Preliminary Analyses

Preliminary analyses using structural equation modeling with the Mplus statistical package (Muthén & Muthén, 1998–2004) were conducted to examine potential sex differences in regard to the means and covariance structure of proactive, reactive, and physical aggression (Browne & Arminger, 1995). Moreover, because all three aggression variables showed considerable positive skewness, a log transformation was applied to normalize the data prior to all analyses (Tabachnik & Fidell, 2001). Notably, because information was provided from both twins in a pair (i.e., from Twin 1 and Twin 2 of a given pair), who were not independent of each other, all analyses were performed on the basis of a double-entry procedure (for a similar approach, cf. Deater-Deckard, 2000). In the double-entry procedure, information such as means or covariances is considered simultaneously from both twins in a pair, but degrees of freedom are corrected to reflect true sample size, which is equal to the number of pairs, not the number of individuals. Compared with a model in which means were freely estimated, a significant loss of model fit was observed when we fixed the means of physical aggression to be equal across gender, $\chi^2(1, N = 151 \text{ pairs}) = 55.04$, $p < .001$ for MZ twins and $\chi^2(1, N = 100 \text{ pairs}) = 23.70$, $p < .001$ for same-sex DZ twins. Similarly, a significant loss of model fit was observed when fixing the means of reactive aggression to be equal across gender, $\chi^2(1, N = 151 \text{ pairs}) = 9.89$, $p < .01$ for MZ twins and $\chi^2(1, N = 100 \text{ pairs}) = 34.70$, $p < .001$ for same-sex DZ twins. Inspection of the log-transformed means showed that boys were more physically aggressive and more reactively aggressive than were girls. No significant sex difference was found, however, in regard to proactive aggression. Examination of the equality of the multivariate covariance structure of reactive, proactive, and physical aggression, also conducted separately for MZ twins and same-sex DZ

twins, revealed that none of the covariances significantly differed between the two sex groups. Indeed, a model with covariances constrained to be equal across sex groups did not significantly differ from a freely estimated model, $\chi^2(8, N = 151 \text{ pairs}) = 12.75$, $p = .12$ for MZ twin parameters, and $\chi^2(6, N = 100 \text{ pairs}) = 8.75$, $p = .19$ for same-sex DZ twin parameters. These findings suggested that neither the magnitude of the genetic and environmental influences on proactive and reactive aggression and on physical aggression nor the pattern of overlap among the three types of aggression differed between the boys and girls in our sample. Data were therefore pooled combining male and female MZ pairs and combining male and female same-sex DZ pairs, respectively, to maximize statistical power (for a similar approach, cf. Arseneault et al., 2003; Dionne, Tremblay, Boivin, Laplante, & Perusse, 2003; Van den Oord et al., 2000). However, to control for the previously mentioned mean differences between boys and girls, we z-standardized aggression scores within gender prior to the pooling of the data. The correlations as well as the means and standard deviations of the three types of aggression are depicted in Table 1, separately for MZ and same-sex DZ pairs.

Assessing the Relative Contribution of Genes and Environment to Aggressive Behaviors

The twin design makes it possible to assess the relative role of genetic factors and environmental factors associated with a given phenotype (Neale & Cardon, 1992). By comparing within-pair (i.e., intraclass) correlations for MZ twins and DZ same-sex twins, one can estimate sources of variability of a given phenotype in terms of genetic and environmental factors (Falconer, 1989). Typically, the relative strength of additive genetic factors on individual differences (i.e., a^2) is approximately twice the MZ and same-sex DZ correlation difference. The relative strength of shared environmental factors that affect twins within a pair in a similar way (c^2) can be approximated by subtracting the MZ correlation from twice the DZ correlation. And, finally, nonshared environmental factors that uniquely affect each twin in a pair (e^2) are approximated by the extent to which the MZ correlation is less than 1. Prior to examining these correlational patterns, we compared data from twins frequenting the same classroom (i.e., twins who were rated by the same teacher) with those from twins in different classrooms (i.e., twins who were rated by different teachers) to exclude the possibility of inflated heritability estimates (Towers et al., 2000). Although no mean differences were observed, a chi-square difference test between the multivariate correlation matrices of twin pairs in the same and those in different classes revealed a significant difference, $\chi^2(19, N = 151 \text{ pairs}) = 74.02$, $p < .001$ for MZ twins, and $\chi^2(19, N = 100 \text{ pairs}) = 35.67$, $p < .05$ for same-sex DZ twins. The sample size ($n = 79$ pairs, 48 MZ and 31 same-sex DZ twins) in the group of twins who were in the same class was too small for us to conduct a formal four-group genetic model test (i.e., DZ–MZ by same–different class). Therefore, only data from twins who were in different classrooms and who thus were evaluated by different raters ($n = 172$ pairs, 55 MZ girls and 48 MZ boys, 33 DZ girls and 36 DZ boys) were used in subsequent analyses. It is important to note that twins from different classrooms did not significantly differ from twins in the same classroom with respect to any of the collected sociodemographic variables.

As can be seen in Table 1, the MZ intraclass correlations appear to be considerably higher than the same-sex DZ intraclass correlations for reactive, proactive, and physical aggression, which suggests a significant contribution of genetic factors to the three types of aggressive behavior. By the same token, this correlational pattern suggests that shared environmental influences may play only a relatively small role —if any—in the etiology of the three types of aggressive behavior. The overall magnitude of the correlations is relatively modest, however, indicating that nonshared environmental factors may account for a significant proportion of the variance in the three variables.

Although the comparison of intrapair correlations across MZ and DZ twins can provide rough approximations of the relative contributions of genetic, shared environmental, and nonshared environmental factors to a given phenotype, structural equation modeling using a maximum likelihood fit function enables a more precise estimation of the genetic and environmental parameters (for a detailed description of the use of structural equation modeling in behavioral genetic analyses, see Neale & Cardon, 1992). Specifically, univariate modeling of variance decomposition provides estimations of the contribution of latent additive genetic (A), latent shared environmental (C), and latent nonshared environmen-

Table 1
Zero-Order Correlations, Intraclass Correlations, and Means and Standard Deviations of Reactive Aggression, Proactive Aggression, and Physical Aggression

Variable	Rea Twin 1	Pro Twin 1	Phy Twin 1	Rea Twin 2	Pro Twin 2	Phy Twin 2	MZ *M*	MZ *SD*	DZ *M*	DZ *SD*
Rea Twin 1	—	0.60	0.72	**0.13**	0.15	0.15	0.88	1.47	0.95	1.58
Pro Twin 1	0.51	—	0.47	0.15	**0.22**	0.25	0.58	0.97	0.42	0.84
Phy Twin 1	0.66	0.60	—	0.15	0.25	**0.16**	0.56	1.12	0.48	1.03
Rea Twin 2	**0.41**	0.33	0.40	—	0.60	0.72	0.88	1.47	0.95	1.58
Pro Twin 2	0.33	**0.42**	0.41	0.51	—	0.47	0.58	0.97	0.42	0.84
Phy Twin 2	0.40	0.41	**0.59**	0.66	0.60	—	0.56	1.12	0.48	1.03

Note. MZ correlations are presented below the diagonal; same-sex DZ correlations are presented above the diagonal. All correlations are significant at $p < .05$ or less. Intraclass correlations between twins in a pair with respect to the same phenotype are in bold. Means and standard deviations are based on raw (i.e., untransformed) data to facilitate interpretation. Rea = Reactive Aggression; Pro = Proactive Aggression; Phy = Physical Aggression; MZ = monozygotic twins; DZ = dizygotic twins.

tal (E) factors to the observed variance of a given phenotype (for an example, see the basic ACE model in Figure 1). This model is fitted to the data in a two-group model in which correlations between twins in a pair are fixed to 1.0 for MZ twins and to 0.5 for DZ twins. The estimated coefficients a, c, and e, which are fixed to be equal across the two twins in a pair and across MZ and DZ twins, are the factor loadings that provide information about the relative contribution of the latent factors A, C, and E to the total variance of each phenotype P, with $P = a^2 + c^2 + e^2$. In the first series of analyses, such a univariate model was fitted to the data, separately for reactive aggression, proactive aggression, and physical aggression. Specifically, we fitted a series of models comparing the full ACE model with a series of submodels (i.e., AE, CE, E). To determine the most probable model given the pattern of intercorrelations observed within twin pairs, we assessed model fit on the basis of the chi-square statistic, the Akaike information criterion, the Bayesian information criterion, the comparative fit index, and the root-mean-square error of approximation. The latter two statistics are two parsimony indexes that take into account both the model chi-square and the associated degrees of freedom, thus correcting for model complexity. Table 2 provides a summary of the tested univariate models. We also fitted additional models specifying a dominance genetic effect D for the different types of aggression. The D path was not significant for any of the three phenotypes, and these models are therefore not reported here.

As can be seen, the variance of all three types of aggression was best described by an AE model, with the estimate of the shared environment factor C being equal to zero in the ACE model in each case. In the best fitting model (AE), heritability accounted for 39% of the variance of reactive aggression, whereas the remaining 61% was explained by nonshared environmental sources. Similarly, heritability accounted for 41% of the variance of proactive aggression, whereas 59% was explained by nonshared environmental sources. For physical aggression, estimates indicated around 62% of heritability and 38% of nonshared environmental influences, which is similar to findings obtained in previous studies (e.g., Dionne et al., 2003).

Assessing Common Etiological Factors Between Reactive and Proactive Aggression

Next, we examined the sources of covariation between reactive and proactive aggression in a series of multivariate models. To this end, we first examined a bivariate correlated or common factors model that included reactive and proactive aggression but that did not take into account the overlap of these two variables with physical aggression. In a bivariate context, shared sources of variance can be estimated in terms of a latent genetic correlation (RG), a latent shared environment correlation (RC), and a latent nonshared environment correlation (RE), indicating the extent to which latent genetic and environmental factors associated with one phenotype overlap with the latent genetic and environmental factors associated with the other phenotype (for a detailed description, see Neale & Cardon, 1992). For example, a significant RG estimate of .9 would indicate that reactive and proactive aggression share 81% of the same genes, whereas a nonsignificant genetic correlation estimate would indicate that the two types of aggression do not share any genetic influences. In the second series of analyses, such a correlated factors model was fitted for reactive aggression and proactive aggression. It is important to note that in this second series of analyses we were interested in the links between only proactive and reactive aggression, that is, without controlling for physical aggression. Therefore, only data including proactive and reactive aggression were used for the subsequent analyses to ensure proper model fit. Univariate analyses had already indicated that an AE model best explained the variance of all three types of aggression (i.e., reactive, proactive, and physical aggression). We thus performed the correlated factors analyses specifying an AE model for both reactive and proactive aggression, with an RG and an RE as the only two estimates of overlap. The bivariate correlated factors model based on an AE model for both reactive and proactive aggression is denoted in Figure 2. The results from the bivariate correlated factors analyses are presented in the middle column of Table 3.

The results reported in Table 3 indicate a good fit of the bivariate correlated factors model. Similar to the previous univariate analyses, 40% of the variance of reactive aggression was

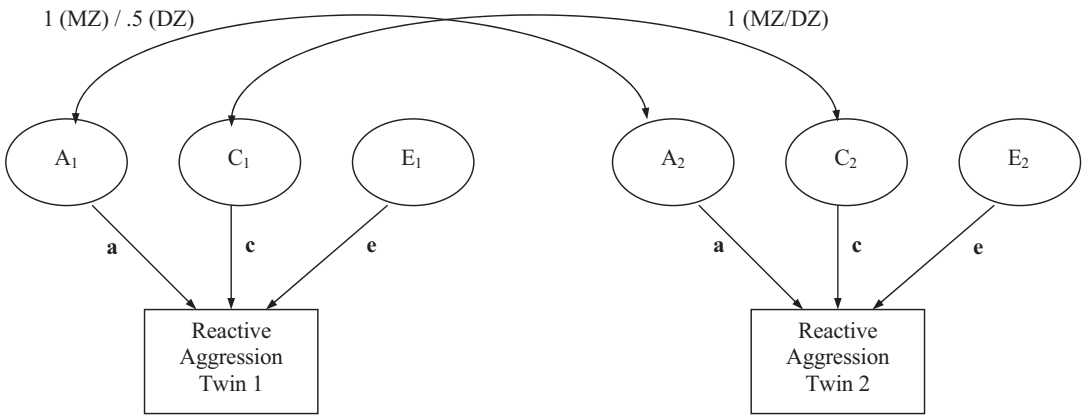

Figure 1. Basic univariate ACE model using reactive aggression as an example. Latent factors A, C, and E refer to additive genetic, shared environmental, and nonshared environmental factors, respectively. Letters in bold are coefficients. MZ = monozygotic; DZ = dizygotic.

Table 2
Amount of Variance Explained in Reactive Aggression, Proactive Aggression, and Physical Aggression: Univariate Models

Aggression Models	Latent Factors: A UPE	A CI	A %	C UPE	C CI	C %	E UPE	E CI	E %	AIC	BIC	CFI	RMSEA	χ^2	*df*	*p*
Reactive																
ACE	.62	.25, .99	39	.00		0	.77	.68, .86	61	1,759.25	1,774.30	1.00	.00	0.13	1	.72
AE*	.62	.47, .76	**39**				.77	**.68, .86**	**61**	**1,757.25**	**1,768.54**	**1.00**	**.00**	**0.09**	**2**	**.76**
CE*				.55	.41, .68	31	.82	.74, .90	69	1,762.94	1,774.23	1.00	.00	3.26	2	.19
E*							.99	.93, 1.0	100	1,792.69	1,800.21	.41	.14	13.81	3	.001
Proactive																
ACE	.63	.20, 1.0	41	.00		0	.76	.66, .86	59	1,755.96	1,771.01	1.00	.00	0.28	1	.60
AE*	**.63**	**.51, .75**	**41**				**.76**	**.66, .86**	**59**	**1,753.96**	**1,765.25**	**1.00**	**.00**	**0.37**	**2**	**.83**
CE*				.55	.44, 1.0	32	.82	.73, .91	68	1,761.56	1,772.85	1.00	.00	3.48	2	.17
E*							.99	.91, 1.0	100	1,792.68	1,800.21	.64	.12	13.30	3	.001
Physical																
ACE	.78	.38, 1.0	62	.00		0	.61	.50, .72	38	1,687.71	1,702.76	1.00	.00	0.23	1	.64
AE*	**.78**	**.63, .94**					**.61**	**.50, .71**	**38**	**1,685.71**	**1,696.99**	**1.00**	**.00**	**0.29**	**2**	**.86**
CE*				.69	.55, .83	49	.71	.62, .80	51	1,708.19	1,719.48	.94	.07	6.55	2	.04
E*							.99	.92, 1.0	100	1,792.69	1,800.21	.37	.19	21.88	3	.000

Note. Best fitting models are in bold. UPE = unstandardized parameter estimate; CI = confidence interval; AIC = Akaike information criterion; BIC = Bayesian information criterion; CFI = comparative fit index; RMSEA = root-mean-square error of approximation.
*indicates a model that is nested within the preceding ACE model.

explained by heritable factors (a_{Rea}), and 60% was explained by unique environmental influences (e_{Rea}). Similarly, 43% of the variance of proactive aggression was explained by genes (a_{Pro}) and 58% of the variance was explained by unique environmental factors (e_{Pro}). The results also showed a very strong correlation between the latent genetic factors contributing to reactive and proactive aggression, RG_{ReaPro} = .87 (.71, 1.00 confidence interval [CI]), but a much more modest, albeit significant, correlation between the latent nonshared environmental factors contributing to reactive and proactive aggression, RE_{ReaPro} = .34 (.19, .49 CI). The fact that the respective 95% CIs do not overlap indicates that these correlations significantly differ from each other. In other words, RG^2 = 76% of the genetic factors influencing reactive and proactive aggression seem to be the same, compared with only a RE^2 = 12% overlap between the unique environmental factors that influence the two types of aggression.

In sum, the results from the bivariate model indicate that the correlation between reactive and proactive aggression in kindergarten children seems to be attributable mainly to overlapping genes and much less so to overlapping environmental conditions. As mentioned previously, however, the genetic and/or environmental links between reactive aggression and proactive aggression may be explained by genetic or environmental effects influencing a common underlying form such as physical aggression. In other words, when one takes into account the overlap with physical aggression, the correlation between the genetic and/or environmental factors contributing to reactive and proactive aggression might be considerably reduced or might even disappear. This possibility was tested in the next series of analyses.

Assessing Common Etiological Factors While Controlling for the Overlap With Physical Aggression

In the next series of analyses, a multivariate model that included reactive aggression, proactive aggression, and physical aggression was fitted to the data. According to the hypothesis outlined previously, this multivariate correlated factors model specified common genetic and nonshared environmental influences for all three phenotypes of physical, reactive, and proactive aggression (A_{PRP}, E_{PRP}) as well as specific genetic and nonshared environmental influences for reactive aggression (A_{Rea}, E_{Rea}) and for proactive aggression (A_{Pro}, E_{Pro}; see Figure 3). The common genetic and nonshared environmental influences for all three phenotypes tested the possibility that the overlapping etiological factors influencing reactive and proactive aggression are related to a common underlying form (i.e., physical aggression). The specific genetic and nonshared environmental influences for reactive and proactive aggression, respectively, assessed the remaining (i.e., specific) genetic and environmental effects on the different functions of aggression (i.e., reactive and proactive aggression) after controlling for a common underlying form. Finally, this model also tested whether any remaining shared sources of variance between reactive and proactive aggression existed that were not accounted for by their overlap with physical aggression. These remaining shared sources of variance were estimated in terms of a latent genetic correlation (RG_{ReaPro}) and a latent nonshared environment correlation (RE_{ReaPro}). These correlations indicate the extent to which the remaining specific genetic and environmental factors associated with reactive aggression overlap with the remaining specific genetic and environmental factors associated with proactive aggression after one controls for the joint overlap of reactive and proactive aggression with physical aggression.

The results reported in the right column of Table 3 indicate a good fit of the multivariate common factors model. Similar to the corresponding univariate analyses, 61% of the variance of physical aggression was explained by genes ($a_{PRP\text{-}Phy}$), whereas 39% was explained by unique environmental factors ($e_{PRP\text{-}Phy}$). As expected, the same genetic and environmental factors that influenced physical aggression also explained part of the variance of both

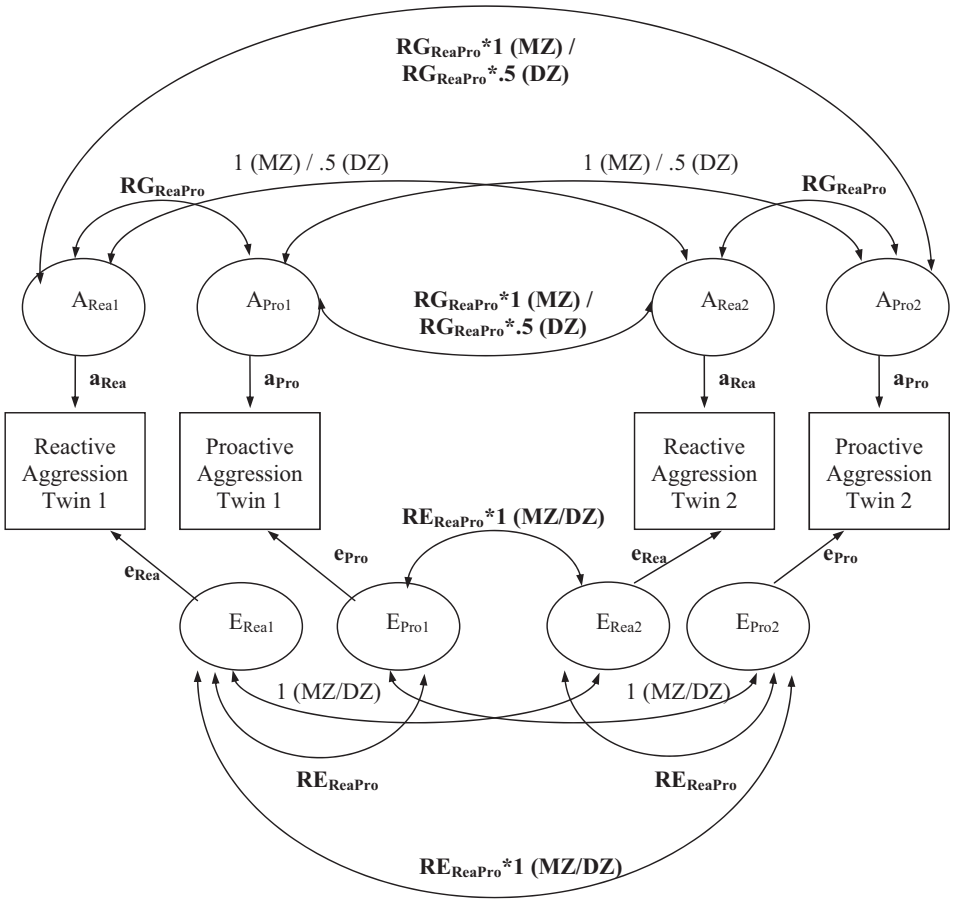

Figure 2. Bivariate correlated factors model based on an AE model for two phenotypes (i.e., reactive aggression and proactive aggression). Latent factors A and E refer to additive genetic and nonshared environmental factors, respectively. Latent factors A_{Rea}, E_{Rea}, A_{Pro}, and E_{Pro} refer to the genetic and nonshared environmental influences on reactive aggression and proactive aggression, respectively. Lowercase letters a_{Rea}, e_{Rea}, a_{Pro}, and e_{Pro} refer to the factor loadings (i.e., estimated parameters) associated with the genetic and nonshared environmental influences on reactive aggression and proactive aggression, respectively. Connotations 1 and 2 refer to Twin 1 and Twin 2, respectively. RG_{ReaPro} refers to the genetic correlation, which represents the overlap of latent genetic influences across phenotypes. RE_{ReaPro} refers to the nonshared environment correlation, which represents the overlap of latent nonshared environment influences across phenotypes. *1 (MZ) / *.5 (DZ) indicate the factor by which the estimated correlation parameters are multiplied for MZ twins and DZ twins, respectively, in the model specification. Estimated parameters are indicated in bold. MZ = monozygotic; DZ = dizygotic.

reactive and proactive aggression. Specifically, the genetic factors influencing physical aggression also explained 29% of the variance of reactive aggression ($a_{PRP\text{-}Rea}$) and 27% of the variance of proactive aggression ($a_{PRP\text{-}Pro}$). The unique environmental factors influencing physical aggression also explained 18% of the variance of reactive aggression ($e_{PRP\text{-}Rea}$) but only 5% of the variance of proactive aggression ($e_{PRP\text{-}Pro}$).

Both reactive aggression and proactive aggression, however, were significantly influenced by additional specific genetic and unique environmental factors that were not related to physical aggression. Specifically, 11% of the variance of reactive aggression was due to specific genetic influences that were not related to physical aggression (a_{Rea}). Similarly, 7% of the variance of proactive aggression was explained by specific genetic influences that were not related to physical aggression (a_{Pro}). The extent of specific environmental influences was considerably larger but seemed to differ for reactive aggression and proactive aggression. Thus, 42% of the variance of reactive aggression was explained by specific environmental influences that were not related to physical aggression (e_{Rea}) compared with 51% of the variance of proactive aggression (e_{Pro}). A model in which these two coefficients were constrained to be equal indeed showed a marginally significant drop in fit compared with an unconstrained model, χ^2 difference $(1, N = 172 \text{ pairs}) = 3.63$, $p = .056$. Apart from the relative extent of the specific genetic and unique environmental factors influencing reactive and proactive aggression, respectively, we also examined the amount of correlation or overlap between these factors in the multivariate model. The results from the multivariate model

Table 3
Multivariate Models of Reactive Aggression, Proactive Aggression, and Physical Aggression

	Correlational models					
	without physical aggression[a]			with physical aggression[b]		
Variable	UPE	CI	%	UPE	CI	%
a_{Rea}	.63	.49, .78	40	.33	.21, .45	11
e_{Rea}	.77	.68, .85	60	.64	.57, .71	42
a_{Pro}	.64	.52, .76	42	.28	.16, .41	7
e_{Pro}	.76	.66, .85	58	.74	.66, .82	51
$a_{PRP\text{-}Rea}$				.53	.39, .68	29
$e_{PRP\text{-}Rea}$				.42	.30, .53	18
$a_{PRP\text{-}Pro}$				.54	.40, .69	27
$e_{PRP\text{-}Pro}$				.23	.07, .38	5
$a_{PRP\text{-}Phy}$				.77	.62, .93	61
$e_{PRP\text{-}Phy}$				.61	.51, .72	39
RG_{ReaPro}	.87	.71, 1.0	76[c]	.46	−.19, .70	0[c]
RE_{ReaPro}	.34	.19, .49	12[c]	.24	.11, .36	6[c]

Note. All models are based on an AE-model for each phenotype. UPE = unstandardized parameter estimates; CI = confidence interval; Rea = Reactive aggression; Pro = Proactive aggression; Phy = Physical Aggression. Percentages represent explained variance of a given phenotype except where otherwise noted.
[a] Akaike information criterion = 3,298.37; Bayesian information criterion = 3,328.46; comparative fit index = 1.00; root-mean-square error of approximation = 0; $\chi^2 = 2.78$; $df = 4.00$; $p = .60$. [b] Akaike information criterion = 4,562.38; Bayesian information criterion = 4,618.81; comparative fit index = 1.00; root-mean-square error of approximation = 0; $\chi^2 = 6.82$; $df = 6.00$; $p = .34$. [c] For the genetic and environmental correlations RG and RE, percentages indicate amount of overlap. Correlational models with and without physical aggression are not nested within each other.

showed that the correlation between the specific genetic influences on reactive aggression and proactive aggression was not statistically significant, $RG_{ReaPro} = .46$ (−.19, .70 CI). An additional model, in which this correlation was fixed to zero, did not differ from the unconstrained model based on a nested chi-square difference test, $\Delta\chi^2$ (1, $N = 172$ pairs) = 1.32, $p = .25$. In other words, when the overlap with physical aggression was taken into account, there was no significant association between the genetic factors influencing reactive and proactive aggression, respectively. In contrast, the correlation between the specific environmental

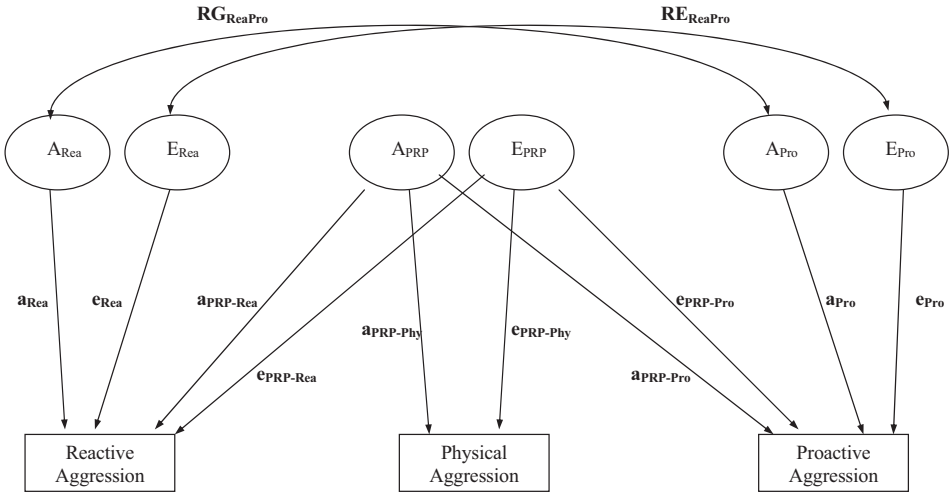

Figure 3. Multivariate correlated factors model based on an AE model for all three phenotypes (i.e., reactive aggression, proactive aggression, and physical aggression). A simplified model is depicted, which does not show parameter specifications within twin pairs and across monozygotic and dizygotic pairs. Latent factors A_{PRP} and E_{PRP} refer to common genetic and nonshared environmental influences on all three phenotypes. Latent factors A_{Rea}, E_{Rea}, A_{Pro}, and E_{Pro} refer to specific genetic and nonshared environmental influences on reactive aggression and proactive aggression, respectively. Latent factors RG_{ReaPro} and RE_{ReaPro} refer to the correlations between the specific genetic and environmental influences, respectively, on reactive aggression and proactive aggression. Estimated parameters are indicated in bold.

influences on reactive aggression and proactive aggression was statistically significant, $RE_{ReaPro} = .24$ (.11, .46 CI). Indeed, an additional model, in which this correlation was fixed to zero, showed a significantly worse fit than did the unconstrained model based on a nested chi-square-difference test, $\Delta\chi^2$ (1, $N = 172$ pairs) = 12.21, $p < .001$.

Taken together, the results from the multivariate model thus suggest that, when controlling for a common underlying form such as physical aggression, reactive aggression and proactive aggression seem to be influenced by different genetic factors. The overall contribution of these specific genetic factors is small, however, in both reactive and proactive aggression. In contrast, specific environmental factors explain the largest portion of the variance in both reactive and proactive aggression, but the overlap between these environmental factors is also small ($RE^2_{ReaPro} = 6\%$).

Discussion

Genetic and Environmental Influences on Reactive and Proactive Aggression

Our goal in the present study was to examine the relative contribution of genetic and environmental effects to reactive and proactive aggression, respectively, as well as the extent to which the two types of aggression show common or distinct etiological factors. A first answer to this question was provided in separate univariate analyses of the two phenotypes. Both reactive aggression and proactive aggression seem to be influenced by genetic factors and by nonshared environmental effects to a similar extent. Specifically, heritability accounted for 39% of the variance of reactive aggression and for 41% of the variance of proactive aggression. The remainder of the variance of both reactive and proactive aggression was accounted for by nonshared environmental factors. These findings concord with the idea that both reactive aggression and proactive aggression have a biological—and presumably heritable—basis in addition to being influenced by environmental contingencies (Raine, 2002). In order to better understand the genetic and environmental influences on reactive and proactive aggression, however, one also needs to consider the overlap between these two functions of aggression as well as the etiological mechanisms that may account for this overlap.

Overlap of Proactive and Reactive Aggression: Shared Etiology Due to a Common Underlying Form

As in previous studies (see Vitaro & Brendgen, 2005), a strong positive correlation was observed between reactive and proactive aggression. In addition, both types of aggression showed similarly strong correlations with physical aggression. Little et al. (2003) showed that the strong overlap between reactive and proactive aggression (i.e., two different functions of aggression) are mainly due to their joint overlap with a common underlying form such as physical aggression. We therefore expected that reactive and proactive aggression would indeed share some—but not all—common etiological factors and that these common (genetic and environmental) factors would be the same ones that also influence physical aggression. We also expected that both reactive and proactive aggression would be influenced by additional genetic and environmental factors that are not linked to a common underlying form such as physical aggression. Moreover, to the extent that physical aggression could entirely explain the overlap between proactive and reactive aggression, these additional genetic and/or environmental factors were expected to be uncorrelated and thus to be specific to proactive and reactive aggression, respectively.

The findings from the present study supported most of these hypotheses. Thus, initial bivariate genetic analyses showed that proactive and reactive aggression shared 76% of their genetic influences and 12% of environmental influences. However, the inclusion of physical aggression in the model revealed that the genetic factors that were shared by reactive and proactive aggression seemed to be those that accounted for a common underlying form, that is, physical aggression. Common environmental effects that were related to a common underlying form were also found, although, as we discuss later, these effects did not entirely explain the initially observed overlap of environmental influences in the bivariate model. Overall, our findings thus support the notion that much of the overlap in etiological factors influencing reactive aggression and proactive aggression can be explained by a shared underlying form such as physical aggression. Apart from the common genetic and environmental effects due to form, reactive and proactive aggression shared no other genetic effects and only a few environmental effects, although additional specific genetic and environmental effects were observed for both reactive and proactive aggression.

Specific Genetic and Environmental Effects on Reactive and Proactive Aggression

Specific genetic effects that were not related to a common underlying form accounted for 11% of the variance of reactive aggression and for 7% of the variance of proactive aggression. Although small, these specific genetic effects may provide important information about the etiology of reactive aggression and proactive aggression because they might help explain the distinct physiological and temperamental correlates of reactive and proactive aggression found in previous studies (Dodge et al., 1997; Price & Dodge, 1989; Vitaro et al., 2002). For example, the presence of genes responsible for a low physiological arousal threshold may predispose a child to angry outbursts of reactive behavior when faced with provocations or threats. In contrast, a genetic predisposition to physiological underreactivity in the sympathetic nervous system may render a child less sensitive to the same stressors. By extension, physiological underreactivity may also render a child less sensitive to aversive social or physical consequences of the aggressive behavior (i.e., punishment), thus increasing his or her likelihood of using aggressive behavior as an instrumental means of goal achievement. The fact that these physiological—and presumably heritable—predispositions are mutually exclusive (i.e., an individual has either a low or high arousal threshold but not both) might explain why the specific genetic effects on proactive and reactive aggression were not significantly correlated in our data once physical aggression was included in the model. In sum, the present results thus suggest that both reactive aggression and proactive aggression have at least some distinct heritable roots, which may explain why some children seem to be especially prone to display one rather than the other type of aggressive behavior, that is, the so-called reactive-only or proactive-only children iden-

tified in previous person-centered studies (e.g., Dodge et al., 1997; Vitaro et al., 1998).

Despite their statistical significance, genetic effects specific to either reactive aggression or proactive aggression accounted for only a very small amount of the interindividual variability of the two phenotypes. Environmental factors that do not explain the form of aggression (i.e., physical aggression) but that specifically influence the function of aggressive behavior seem to play the most essential role in the development of reactive and proactive aggression. Thus, 42% of the variance of reactive aggression and 51% of the variance of proactive aggression were determined by such specific environmental effects that were unrelated to physical aggression. It is important to note that only a fraction (6%) of these environmental effects seems to be shared between reactive and proactive aggression. What may these specific environmental influences be? As suggested in Dodge's (1991) theoretical model of reactive and proactive aggression, a harsh, threatening, and unpredictable environment may particularly foster outbursts of anger and reactive aggression in the child. Conversely, an overly lenient and "supportive" social environment may create conditions that teach children to use aggressive behavior as an instrument for goal achievement. Although parental behavior might reflect such specific environmental conditions, the family environment has been traditionally viewed as a part of the latent C (i.e., shared environment) factor, which did not significantly contribute to reactive and proactive aggression in our data. Differential experiences with the peer group thus might be a more likely source of specific environmental influences on reactive and proactive aggression, especially given that only twin pairs in which the two twins were in different classrooms were used for the analyses.

When entering kindergarten—if not earlier—children become exposed to a relatively large social context of same-age peers. At this point, the establishment of a social position in the peer group and the formation of new dyadic friendships become important developmental tasks (Boivin, Vitaro, & Poulin, 2005). Interaction with peers thus affords new opportunities to shape children's behaviors, and children who experience difficulties in the peer context may be at risk of maintaining or further increasing already-existing behavior problems, including reactive and proactive aggression. Empirical evidence suggests, however, that the specific type of aggressive behavior affected may depend on the specific type of problematic peer experience. Thus, in addition to being at risk for maltreatment by adults, reactively aggressive children have been found to be at high risk for maltreatment by peers, including peer rejection and victimization by classmates (Dodge et al., 1997; Poulin & Boivin, 2000a; Price & Dodge, 1989; Prinstein & Cillessen, 2003). In turn, maltreatment by peers has been shown to particularly aggravate children's reactively aggressive behavior (Dodge et al., 2003). Contrary to reactive aggression, proactive aggression seems to be more tolerated and even reinforced by peers. Thus, proactively aggressive children often have friends who are similarly proactively aggressive, whereas reactively aggressive children are often friendless (Poulin & Boivin, 2000b). Moreover, friends' proactive aggressiveness has been shown to increase children's own proactive aggressiveness, whereas friends' reactive aggressiveness seems to have no influence on children's own aggressiveness profile (Poulin & Boivin, 2000b). Once more, these results concur with the view that reactive aggression is related to—or results from—aversive experiences, whereas proactive aggression is supported by relatively "positive" socialization experiences. Indeed, these "positive" socialization experiences seem to be crucial for the development of proactive aggression, as they explained most of the interindividual variance associated with this phenotype.

As is the case for the different physiological and temperamental correlates of reactive and proactive aggression, the different peer-related experiences related to reactive and proactive aggression appear—at least at first glance—to be mutually exclusive. Mutually exclusive socialization processes would indeed concur with our finding that the specific environmental factors influencing reactive and proactive aggression are largely uncorrelated, showing an overlap of only 6%. Despite its small size, however, the overlap between the specific environmental factors influencing reactive and proactive aggression was statistically significant, and the question arises about what factors might explain this commonality. One possible explanation may be that certain problematic peer experiences, albeit being differentially linked to reactive and proactive aggression, can influence each other. For example, as argued by Dishion (1990), rejection by the peer group might limit access to friendships with conventional peers, which may prompt some rejected children to seek the company of those peers who are available, namely less conventional and aggressive peers. This pattern may not hold for all rejected children, however, many of whom do not have any friends (e.g., Brendgen, Little, & Krappmann, 2000; e.g., Parker & Asher, 1993). The fact that rejection does not lead to affiliation with aggressive friends for all children may thus explain why the overlap between the specific environmental factors influencing reactive and proactive aggression is relatively limited.

Limitations and Conclusion

This study is the first to compare the relative contribution of heritability and environmental effects on reactive and proactive aggression in children. Nevertheless, like many other studies, this study has several limitations that need to be considered when interpreting the present results. The most obvious limitation is the small sample size, which may have precluded the detection of sex differences in regard to genetic and environmental effects on reactive and proactive aggression. As such, future studies need to replicate the present findings using larger samples before definite conclusions can be drawn in regard to the relative contribution of genetic versus environmental effects on reactive and proactive aggression and potential sex differences in this context. In addition, caution needs to be exercised in trying to generalize the present results to other sociocultural contexts or beyond the assessed age. Some previous studies on aggression and antisocial behavior have suggested that genetic effects diminish with age, whereas the magnitude of nonshared environmental influences increases (Rhee & Waldman, 2002). As such, it is possible that heritable factors may play a larger role initially in placing a child at risk for reactively or proactively aggressive behavior but that later socialization experiences determine whether the child overcomes this risk. Another limitation concerns the fact that physical aggression was the only form of aggressive behavior examined in the present study. Although physical aggression has been identified as the most likely source of confound in the teacher-rated measure of reactive and proactive aggression commonly used in

the literature (Little et al., 2003), it will be important to also include other forms such as verbal or indirect aggression in future studies. Indeed, inclusion of these additional forms may result in correlation estimates of zero for both the specific genetic effects and the specific environmental effects on reactive and proactive aggression. Because of the statistical complexity of a genetic model including four or more phenotypes, however, such a test will necessitate a much larger sample than the one available in the present study. The present findings also need to be replicated with measurement sources other than teacher ratings, preferably in a multisource, multimethod framework. Although peer reports of reactive and proactive aggression have been found to be unreliable in young children (Poulin & Boivin, 2000a) and self-reports may be equally problematic at that age, parent ratings and especially observational measures in future studies may provide important complementary information in this context.

Another limitation shared with many other twin studies is that neither genes nor environmental features were explicitly measured, but their effects were estimated on the basis of the pattern of covariance between twins. Because the specific genes associated with proactive and reactive aggression have yet to be identified, however, findings from twin designs such as those used in the present study can provide important first insights into whether the two types of aggression are—at least to some extent—heritable and whether they share the same genetic and environmental influences (Jaffee et al., 2005; McGue, Elkins, Walden, & Iacono, 2005a; Moffitt, 2005). In turn, the results from the present study can guide future research attempting to identify candidate genes for proactive and reactive aggression (e.g., Caspi et al., 2002; McGue et al., 2005a). Finally, it should be noted that the tested models are based on the assumption that genetic contribution is additive. However, not only is it possible that genes interact in their effects on behavior but behavior is also most likely shaped by the interaction between genes and environment (Greenberg, 2005). Whereas gene–gene interactions are not testable without measuring specific genes, interactions between heritable and environmental factors can be demonstrated with the twin paradigm and specific measures of environmental features (e.g., Eaves, Silberg, & Erkanli, 2003; Jaffee et al., 2005). Similar tests of gene–environment interactions should be incorporated in future studies to assess under which specific environmental conditions genetic vulnerability may play a role in the development of proactive and reactive aggression. In a related vein, a possible gene–environment correlation may have been masked in the present findings of additive genetic and environmental effects. However, genetic factors may impact behavior indirectly by affecting the environments that children experience (McGue, Elkins, Walden, & Iacono, 2005b), either through active choice (e.g., affiliation with aggressive friends) or through the reactions of the social environment to a genetically driven behavioral disposition (e.g., peer rejection and victimization), and this possibility should also be assessed in future studies.

Despite its limitations, we believe the present study demonstrates that genetic designs can make an important contribution to the question of the etiology of reactive and proactive aggression. Specifically, the present study is the first to show that both reactive aggression and proactive aggression seem to be determined not only by environmental factors but also by genetic factors. However, most of the genetic effects contributing to reactive and proactive aggression seem to be associated with a common underlying form of aggression (i.e., physical aggression). Additional distinct genetic effects on reactive and proactive aggression also exist, but they play a comparatively small role in explaining the two functions of aggressive behavior. The most important contribution to both reactive and proactive aggression seems to come from environmental effects that are unrelated to physical aggression and that are—for the most part—specific to each of the two functions of aggressive behavior. In other words, whether a child uses aggressive behavior more for reactive or more for proactive purposes seems to be determined to a small extent by specific heritable traits and mostly by the child's specific socialization experiences. The results from the present study thus pave the way for future studies aimed at isolating the specific genes and environmental variables affecting the two functions of aggressive behavior. Finally, the present results also have important implications for preventive interventions as they suggest that intervention approaches tailored to the particular temperamental and socialization needs of reactively or proactively aggressive children are necessary if we are to effectively curb aggressive behavior (e.g., Phillips & Lochman, 2003).

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