Examining Genetic and Environmental Effects on Social Aggression: A Study of 6-Year-Old Twins

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Using a genetic design of 234 six-year-old twins, this study examined (a) the contribution of genes and environment to social versus physical aggression, and (b) whether the correlation between social and physical aggression can be explained by similar genetic or environmental factors or by a directional link between the phenotypes. For social aggression, substantial (shared and unique) environmental effects but only weak genetic effects were found. For physical aggression, significant effects of genes and unique environment were found. Bivariate modeling suggests that social and physical aggression share most of their underlying genes but only very few overlapping environmental factors. The correlation between the two phenotypes can also be explained by a directional effect from physical to social aggression.

Aggressive behavior among children has long been recognized as a major risk factor for subsequent developmental maladjustment, both for the perpetrators and the victims. Until recently, attempts to understand and prevent childhood aggression have been guided by a male-oriented model with a focus on physical aggression. There is mounting evidence, however, that children's aggressive behavior incorporates more than the infliction of physical harm. Thus, children can hurt their peers through more subtle forms of aggression, for example, through social exclusion or rumor spreading (e.g., Bjoerkqvist, Lagerspetz, & Kaukiainen, 1992; Crick, Casas, & Mosher, 1997; Hart, Nelson, Robinson, Olsen, &

McNeilly-Choque, 1998; Willoughby, Kupersmidt, & Bryant, 2001). Notably, these forms of aggression are considered by the victims to be as harmful as physical aggression (Crick, Bigbee, & Howes, 1996; Paquette & Underwood, 1999), with a range of negative effects including anxiety, depression, and even suicide ideation (Owens, Slee, & Shute, 2000).

Different labels have been used to describe these more subtle forms of aggression, specifically, *indirect aggression* (e.g., Bjorkqvist, Lagerspetz, et al., 1992), *relational aggression* (e.g., Crick & Grotpeter, 1995), and *social aggression* (e.g., Galen & Underwood, 1997). All three terms refer to the social manipulation of peer relations to harm another individual, but indirect aggression is mainly covert whereas relational aggression can be both covert (e.g., spreading rumors) and overt (e.g., threatening to withdraw friendship). Social aggression encompasses both overt and covert behaviors and includes nonverbal aggressive behavior (e.g., ignoring someone or making mean faces). In the present study, both covert and overt socially manipulative behaviors are captured; therefore, we use the term *social aggression*. Several studies have suggested that social aggression is especially frequent in girls, although other studies have reported no gender difference or even a gender difference favoring boys' use of social aggression (for

reviews, see Crick et al., 1999; Underwood, Galen, & Paquette, 2001). Many aggressive children seem to use both forms of aggression, however, as indicated by the significant overlap between physical and social aggression ($r = .4$ to $.8$, depending on the source of rating and the age of the participants). Despite this overlap, a nationally representative longitudinal study with Canadian children ages 4 through 11 years showed that social aggression and physical aggression represent two factorially distinct indexes of aggression, whose measurement structure remains stable from the preschool years through preadolescence (Vaillancourt, Brendgen, Boivin, & Tremblay, 2003).

Studying the Etiology of Social Aggression: The Usefulness of a Genetically Informed Approach

The recognition that aggression can be expressed though different means has highlighted the need for a better understanding of the factors contributing to social versus physical aggression. Compared with the multitude of studies investigating the developmental antecedents of physical aggression, however, relatively little is known about the etiology of social aggression. The few existing studies, most of which are cross-sectional, compared the contribution of familial and peer-related environmental characteristics and of individual characteristics of the child to social aggression and to physical aggression. When investigating the relative effects of individual and environmental factors on the etiology of a behavior, however, it is difficult to interpret the findings if only one child per family is assessed. For example, the links between a putative environmental variable such as parenting behavior and aggression in the child may in fact be due to the genetic transmission of problem behaviors. Similarly, peer influences on child aggressive behavior through affiliation with aggressive friends may not reflect only environmental effects but also genetically driven selection processes (DiLalla, 2002; Rhee & Waldman, 2002; Rowe & Osgood, 1984). The use of twin designs allows a better control of this problem (DiLalla, 2002; Plomin, 1994). The comparison of the phenotypic similarity of monozygotic twins (MZ), who are genetically identical, with the phenotypic similarity of same-sex dizygotic twins (DZ), who presumably share only half of their genes, makes it possible to estimate the influence of genetic factors, shared environmental influences (e.g., parental behavior), and nonshared environmental influences (e.g., different peer networks of the two twins in a pair) on phenotypic similarity between twins (Neale & Cardon,

1992). Using such a genetically informed approach, the first goal of the present study was to examine the relative contribution of genetic and (shared and nonshared) environmental factors to social aggression compared with physical aggression. The second goal was to investigate the etiological mechanism that might explain the positive correlation between the two types of aggression.

Etiology of Social Aggression: Genetic Versus Environmental Factors

Several studies have examined heritability effects on aggression in children (for reviews, see Cadoret, Leve, & Devor, 1997; DiLalla, 2002; Miles & Carey, 1997; Rhee & Waldman, 2002). Although the estimates vary, depending on the 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 the literature has reported only a relatively small and often nonsignificant contribution of environmental sources shared between twins. Most studies are based on mother or averaged parent ratings of child aggressive behavior, but similar results have been obtained when juxtaposing different reporting sources such as mothers and fathers (Van den Oord, Verhulst, & Boomsma, 1996) 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 aggressive and antisocial behavior (Rhee & Waldman, 2002; but see Miles & Carey, 1997, for contradictory findings).

No study has examined whether social aggression in children shows a similar heritability as physical aggression, although several scholars have criticized the lack of knowledge in this context (DiLalla, 2002; Rhee & Waldman, 2002). At least theoretically, it is conceivable that social aggression is triggered by genetic mechanisms as much as is physical aggression. For example, genes may affect psychophysiological factors that predispose an individual to aggressive behavior, which may be expressed mainly through physical means in some individuals and mainly through social means in others. Some indirect support of this notion is provided by findings that increasing cortisol levels from morning to afternoon are linked to physical aggression and social aggression in 3- to 8-year-old children (Dettling, Gunnar, &

Donzella, 1999). Moreover, temperamental difficulties in emotion regulation and self-control, which show a high rate of heritability (Cyphers, Phillips, Fulker, & Mrazek, 1990; DiLalla & Jones, 2000), have been associated with both physical aggression (Bates, Bayles, Bennett, Ridge, & Brown, 1991; Caspi, Henry, McGee, & Moffitt, 1995) and social aggression (Isobe & Sato, 2003). Alternatively, the sometimes complex manipulative strategies that are part of social aggression, such as rumor spreading or isolation of an individual from the group, may necessitate a greater amount of social learning than the gross motor skills involved in physical aggression. It has been argued that a large social network—and by extension modeling and reinforcement processes within this social network—are essential preconditions for social aggression (Grotpeter & Crick, 1996). In line with this notion, boys and girls of high centrality in their peer networks are more likely than those of peripheral status to use socially aggressive strategies, whereas the use of physical aggression is not linked to peer network centrality (Xie, Cairns, & Cairns, 2002). Consequently, as suggested by DiLalla (2002), it is also possible that social aggression shows much less heritability than physical aggression.

Etiology of Social Aggression: Explaining the Overlap With Physical Aggression

In addition to the question whether social aggression shows a similar level of heritability as physical aggression, one might ask which etiological mechanisms explain the positive correlation between the two types of aggression. Hypotheses put forth to explain the link between physical aggression and social aggression can be subsumed under two general developmental models. The first model could be called the shared, or correlated, etiological factors model. This model assumes that both types of aggression are, at least in part, caused by the same genetic or environmental factors, or both. The second model describes a phenotype-to-phenotype effect during development, whereby one phenotype directly influences the occurrence of the other phenotype without sharing any other etiological features. Because the correlated etiological factors model and the phenotype-to-phenotype effect model are not nested options, both alternatives may offer similar fit to the data.

Shared or correlated etiological factors. To the extent that social aggression and physical aggression are expressions of the same underlying aggressive tendency, one might expect not only a comparable heritability of the two types of aggressive behavior but also a considerable amount of overlap between the predisposing genetic factors. This notion is supported by the aforementioned findings that physical and social aggression seem to have similar physiological and temperamental correlates. Moreover, it is possible that the same or correlated features of the (shared or unique) environment may create conditions that simultaneously foster both physically aggressive and socially aggressive behavior. Such conditions may be generated, for example, by negative parenting behaviors such as a lack of responsiveness, coercion, and physical punishment, which have been found to be related to both physical aggression (Dishion, Duncan, Eddy, & Fagot, 1994) and social aggression (Hart et al., 1998) in children. Another important and possibly common environmental influence may be affiliation with similarly aggressive friends, which has not only been shown to foster children's physical aggression (Vitaro, Tremblay, & Bukowski, 2001) but also plays an equally important role in the development of socially aggressive strategies (Werner & Crick, 2004).

Other empirical evidence suggests, however, that social aggression and physical aggression might be influenced by different heritable factors and especially by different environmental characteristics. For example, cognitive ability in middle childhood, which is partly determined by genes (McGue, Bouchard, Iacono, & Lykken, 1993), is differentially linked to physical and social aggression. Specifically, a high level of social intelligence is positively correlated with social aggression but is uncorrelated with physical aggression (Kaukiainen et al., 1999). Similarly, the level of language development, which is also in part genetically determined, has been negatively related to physical aggression in early and middle childhood (Dionne, Tremblay, Boivin, Laplante, & Perusse, 2003; Stevenson, Richman, & Graham, 1985). In contrast, a positive link has been shown between language development and social aggression (Bonica, Arnold, Fisher, Zeljo, & Yershova, 2003). Further support for the notion that not all "risky" environmental conditions may foster social aggression to the same extent as physical aggression comes from a recent experimental study by Coyne, Archer, and Eslea (2004). They showed that viewing social aggression on film mainly increases socially aggressive responses in children, whereas viewing physical aggression mainly increases physically aggressive responses. Taken together, existing evidence is thus equivocal and provides equal support for the notion that physical aggression and social aggression may be fostered either by the same or by different genetic and environmental factors.

Phenotype-to-phenotype contributions. In addition to the possibility of shared etiological factors, there is another scenario that might explain the link between the two types of aggression, namely, a phenotype-to-phenotype effect. This scenario is supported by the theoretical model of aggression proposed by Bjoerkqvist and colleagues (Bjoerkqvist, Lagerspetz, et al., 1992; Bjoerkqvist, Oesterman, & Kaukiainen, 1992). According to this model, very young children aggress against others primarily through physical means because of a lack of other expressive tools. As verbal and social cognitive skills evolve, children begin to use verbal aggression and, at around 5 to 6 years, add social aggression to their repertoire. Because physical and verbal forms of aggression are socially less acceptable, and because social aggression can be as damaging with much less risk of retribution, social aggression eventually becomes the primary strategy. According to the Bjoerkvist et al. model (Bjoerkqvist, Lagerspetz, et al., 1992), high levels of physical aggression should thus lead to high levels of social aggression, although the reverse pattern should not be true. Indirect support for this notion comes from the fact that physical aggression generally diminishes from early childhood onward, whereas social aggression increases (e.g., Cairns, Cairns, Neckerman, Ferguson, & Gariepy, 1989; Osterman et al., 1998; Tremblay, 1999; Tremblay et al., 1996). In contrast, the only study that directly tested the longitudinal associations between physical and social aggression did not find any cross-lagged links (Vaillancourt et al., 2003). However, to the extent that the shift from physical to social aggression mainly occurs during the transition from early to middle childhood, the merging of several age levels in that study might have precluded the detection of directional effects between the two types of aggression.

Research Questions of the Present Study

In summary, the goal of the present study was to examine the following questions: Compared with physical aggression, to what extent is the variance of social aggression explained by genetic, shared environmental, or nonshared environmental factors? To what extent can the covariance between physical aggression and social aggression be explained (a) by the same underlying genetic or shared or nonshared environmental factors (i.e., corresponding to a shared or correlated etiological factors model), or (b) by direct effects from one type of aggression to the other (i.e., corresponding to a phenotype-to-phenotype contribution model)? These questions were addressed comparing two of the most frequently used reporting sources of child aggression: teacher and peer ratings. Peer ratings as well as teacher ratings show a clear factorial distinction between social aggression and physical aggression even for preschool children (Crick et al., 1997; McNeilly-Choque, Hart, Robinson, Nelson, & Olsen, 1996; Tomada & Schneider, 1997; Willoughby et al., 2001). In addition, peer ratings and teacher ratings of social aggression and physical aggression in preschool children show good stability over periods up to 9 months as well as good construct and predictive validity based on concurrent and longitudinal links with related antisocial behaviors and peer difficulties (Crick et al., 1997; McNeilly-Choque et al., 1996; Tomada & Schneider, 1997; Willoughby et al., 2001). Despite their good psychometric properties, however, teacher and peer ratings of young children's social and physical aggression typically show only low to moderate correspondence (Crick et al., 1997; McNeilly-Choque et al., 1996; Tomada & Schneider, 1997). It was thus important to examine whether the same pattern of results with regard to our research questions could be obtained for both peer and teacher reports.

Method

Sample

Participants in the present study were part of an ongoing longitudinal study (Quebec Newborn Twin Study [QNTS]) of a population-based sample of twins from the greater Montreal, Canada area who were recruited at birth between November 1995 and July 1998 ($N = 322$ twin pairs). For same-sex twin pairs ($n = 237$), zygosity was assessed at 18 months based on physical resemblance using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991). For a subsample of these same-sex twin pairs ($n = 123$), a DNA sample was evaluated with respect to 8 to 10 highly polymorphous genetic markers. 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 (Forget-Dubois et al., 2003). 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 average yearly household income (CAN$54,000) in the twin sample was slightly above the national average for couples with children. However, a comparison of family characteristics of

this sample at 5 months of age with an epidemiological sample of singletons from the Montreal area indicated that the samples were very similar regarding parental education, yearly income, age of parents at the birth of the children, and marital status.

The sample was followed longitudinally at 5, 18, 30, 48, and 60 months focusing on a variety of child- and family-related characteristics. A sixth wave of data collection was completed at 6 years of age to assess children's 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 ($SD = 3.6$). Attrition in the sample averaged approximately 2 % per year, resulting in a total of 234 twin pairs for the data collections at age 6 years (MZ males = 44, MZ females = 50, DZ males = 41, DZ females = 32, DZ mixed sex = 67). Twins remaining in the study at 6 years of age did not differ from those lost with regard to zygosity status, family status, mother's level of education, and parent-rated temperament at 5 months of age. However, fathers in the remaining study sample had a slightly higher level of education than did fathers of the twins who were lost 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 the following description of measures). 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 Institutional Review Board (IRB) and the school board administrators. Before data collection, active written consent from the parents of all the children in the classroom was obtained. Data collection took place in the spring of the kindergarten year to ensure that children and teachers had gotten to know each other. In 175 of the 234 twin pairs, the two twins did not attend the same classroom, whereas in 59 (25.2%) pairs, the two twins attended the same classroom. Overall, sociometric data (i.e., peer nominations) and teacher ratings were thus obtained from a total of 409 classrooms. The sociometric procedure took approximately 45 min per class. Although the collection of sociometric data was highly time consuming and costly, it did provide valuable information about children's aggressive

behavior from the peers' perspective, which is not normally available for twin samples. Children were encouraged not to share their responses with each other. At the same time, teachers completed the behavior questionnaires for the twin(s) in their class.

Teacher ratings. Kindergarten teachers rated the children's level of social aggression and physical aggression using items based on the Preschool Social Behavior Scale (PSBS–T; Crick et al., 1997) and on the Direct and Indirect Aggression Scales (Bjoerkqvist, Lagerspetz, et al., 1992). With regard to social aggression, the teachers in the present study indicated to what extent the child "tries to make others dislike a child," "becomes friends with another child for revenge," and "says bad things or spreads nasty rumors about another child." With regard to physical aggression, the teachers indicated to what extent the child "gets into fights," "physically attacks others," and "hits, bites, or kicks others." Responses were given on a 3-point scale (0 = never, 1 = sometimes, 2 = often). For each type of aggression, the respective individual item scores were summed to yield a total social aggression score and a total physical aggression score. Internal consistency of the total scales was high in the present sample, with Cronbach's $\alpha = .89$ for teacher-rated physical aggression ($M = 0.78$, $SD = 1.41$) and Cronbach's $\alpha = .82$ for teacher-rated social aggression ($M = 0.72$, $SD = 1.22$). Because both teacher-rated social aggression and teacher-rated physical aggression showed considerable positive skewness, an inverse transformation was applied to normalize the data (Tabachnik & Fidell, 2001).

Peer ratings. In addition to teacher ratings, peer reports were obtained for the twins' level of social and physical aggression. For this purpose, booklets of photographs of all the children in a given class were handed out. Two research assistants ensured that all children recognized the photos of all their classmates by presenting them individually. The children were then asked to circle the photos of three children who best fit a behavioral descriptor. On the following page, children were asked to circle the faces of three children who best fit another behavioral descriptor, and so forth. Two behavioral descriptors were used for social aggression ("tells others not to play with a child" and "tells mean secrets about another child") and two others for physical aggression ("gets into fights" and "hits, bites, or kicks others"). Most twins (82%) received at least one nomination as being among the three most socially aggressive children in the classroom on at least one of the items. Similarly, most twins (62%) received at least one nomination as being among the three most physically aggressive children in the

classroom on at least one of the items. For each behavioral descriptor, the total number of received nominations was calculated for each child and z standardized within the classroom to account for differences in classroom size. For each type of aggression, the respective z-standardized individual item scores were then summed to yield a total social aggression score and a total physical aggression score. Internal consistency of the total scales in the present sample was Cronbach's $\alpha = .87$ for peer-rated physical aggression ($M = -0.19$, $SD = 0.73$) and Cronbach's $\alpha = .62$ for peer-rated social aggression ($M = -0.26$, $SD = 0.77$). Similar to teacher ratings, peer ratings of social and physical aggression were positively skewed, and a logarithmic transformation was applied to normalize the data (Tabachnik & Fidell, 2001).

Results

Preliminary Analyses

Preliminary analyses were conducted to examine potential sex differences with regard to physical and social aggression as well as the amount of overlap between the two phenotypes. Results showed that, according to teachers, boys were more physically aggressive than girls, $t(466) = 4.51$, $p = .000$, and girls were more socially aggressive than boys, $t(466) = -2.79$, $p = .006$. According to peers, boys were more physically aggressive and more socially aggressive than girls, $t(466) = 11.06$, $p = .000$, and $t(466) = 2.53$, $p = .012$, respectively. Because of the relatively small sample size, it was not possible to perform sex-limited genetic analyses to test for potential moderating effects of gender with regard to the genetic and environmental effects on social versus physical aggression. However, to control for the mean differences between boys and girls in the subsequent genetic analyses, teacher- and peer-rated social and physical aggression were z standardized within gender. Social and physical aggression showed a modest, yet significant, overlap for teacher ratings ($r = 0.43$, $p = .000$) and for peer ratings ($r = 0.41$, $p = .000$). Correlations between teacher and peer ratings were moderate for social aggression ($r = 0.33$, $p = .000$) and for physical aggression ($r = 0.25$, $p = .000$).

Assessing the Relative Contribution of Genes and Environment to Social Aggression Versus Physical Aggression

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 correlations for MZ twins and same-sex DZ twins, sources of variability of a given phenotype can be estimated 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, $a^2 = 2(r_{MZ} - r_{DZ})$. The relative strength of shared environmental factors that affect twins within a pair in a similar way (c^2) can be estimated by subtracting the MZ correlation from twice the DZ correlation, $c^2 = 2r_{DZ} - r_{MZ}$. 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, $e^2 = 1 - r_{MZ}$. Notably, because estimation of the basic genetic models rests on the comparison of MZ and same-sex DZ twins (Neale & Cardon, 1992), mixed-sex DZ pairs were not included in the subsequent analyses.

A preliminary examination of the equality of the bivariate covariance structure of social and physical aggression between boys and girls was conducted by means of χ^2-difference tests, separately for MZ twins and same-sex DZ-twins and separately for teacher ratings and peer ratings. These tests revealed that none of the covariances differed significantly between the two sex groups. Indeed, a model with means and covariances constrained to be equal across sex groups did not differ significantly from a freely estimated model: teacher-rated MZ twin parameters, $\chi^2(14) = 19.59$, $p = .14$; peer-rated MZ twin parameters, $\chi^2(14) = 19.43$, $p = .15$; teacher-rated same-sex DZ twin parameters, $\chi^2(14) = 19.06$, $p = .16$; and peer-rated same-sex DZ twin parameters, $\chi^2(14) = 14.74$, $p = .40$. These findings suggested that neither the magnitude of the genetic and environmental influences on social and physical aggression nor the pattern of overlap between the two 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 pair to maximize statistical power (for a similar approach, see Dionne et al., 2003; Arseneault et al., 2003; Van den Oord, Boomsa, & Verhulst, 2000).

Rough estimations of genetic and environmental sources of variance can be obtained by comparing within-pair correlations for MZ and same-sex DZ twins on each phenotype. These correlations are depicted in Table 1. As can be seen, MZ correlations appear to be almost twice as high as same-sex DZ correlations for physical aggression for both teacher

Table 1

Zero-Order Correlations and Means and Standard Deviations for Teacher Ratings and Peer Ratings

	SA Twin 1	PA Twin 1	SA Twin 2	PA Twin 2	MZ *M*	MZ *SD*	DZ *M*	DZ *SD*
			Teacher ratings					
SA Twin 1	1.00	.44	.34	.16	− .09	.92	.11	1.07
PA Twin 1	.42	1.00	.17	.25	− .03	.96	.03	1.04
SA Twin 2	.35	.17	1.00	.49	− .10	.94	.13	1.04
PA Twin 2	.30	.61	.37	1.00	− .03	.94	.04	1.06
			Peer ratings					
SA Twin 1	1.00	.51	.40	.25	− .16	1.03	.08	.86
PA Twin 1	.50	1.00	.34	.28	− .19	.95	.14	1.03
SA Twin 2	.50	.22	1.00	.36	− .07	1.01	− .07	.99
PA Twin 2	.37	.49	.36	1.00	− .17	.93	− .02	1.00

Note. Monozygotic (MZ) correlations are presented below the diagonal; dizygotic (DZ) correlations are presented above the diagonal. All correlations greater than .16 are significant at $p < .05$ or less. SA = social aggression; PA = physical aggression.

and peer ratings, which suggests a substantial contribution of genetic factors on individual differences for physical aggression irrespective of the reporting source. This finding also suggests, however, that shared environmental influences may play a negligible role in the etiology of physical aggression. In contrast, MZ and same-sex DZ correlations for social aggression are similar, both for teacher and peer ratings, which suggests that this phenotype may be explained more by shared environmental factors than by genetic factors. The overall magnitude of the correlations is modest, however, indicating a significant contribution of nonshared environmental factors to both physical aggression and social aggression.

Structural equation modeling using a maximum likelihood fit function applied to the twin data enables a more precise estimation of the genetic and environmental parameters. Univariate modeling of variance decomposition provides estimations of ad-ditive genetic (A), shared environmental (C) and nonshared environmental (E) factors, separately for social aggression and physical aggression (see the basic ACE model in Figure 1). Parameters A, C, and E are expressions of portions of the total variance of each phenotype. In the first series of analyses, such univariate estimates of variance decomposition were calculated using the Mx statistical package (Neale, 1999). Specifically, a series of models was fitted comparing the full ACE model to a series of sub-models (i.e., AE, CE, E). These models were tested separately for social aggression and physical aggression and separately for the two reporting sources. To determine the most probable model given the pattern of intercorrelations observed within twin pairs, model fit was assessed based on the χ^2 statistic, the Akaike information criterion (AIC), and the Bayesian information criterion (BIC). The latter two statistics are parsimony indexes that take into account both the model chi-square and the
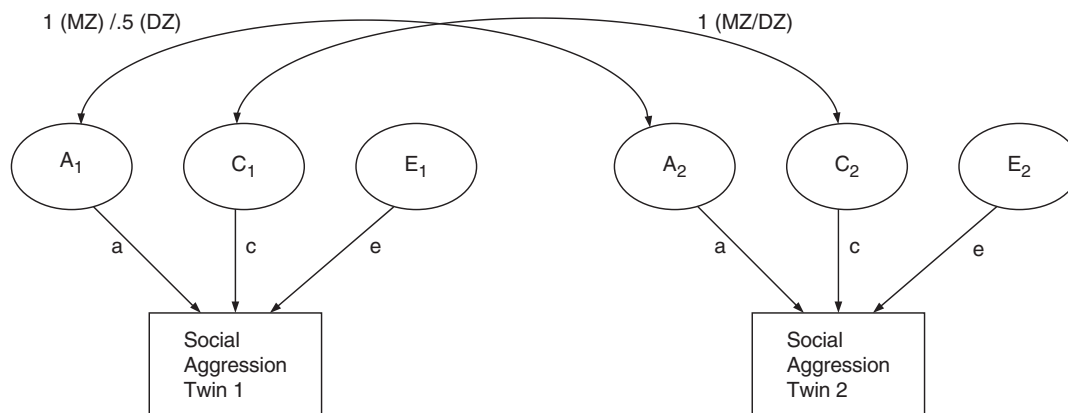

Figure 1. Basic univariate ACE model. Parameters A, C, and E refer to additive genetic, shared environmental, and nonshared environmental factors, respectively.

Table 2

Amount of Variance Explained in Physical Aggression and Social Aggression: Univariate Models

	A	C	E	AIC	BIC	χ^2	df	p	$\Delta\chi^2$	Δdf	Δp
			Teacher-rated physical aggression								
ACE model	.63 (.35, .73)	.00 (.00, .23)	.37 (.27, .51)	−9.44	−32.31	2.55	6	.86			
AE model[a]	**.63 (.49, .73)**		**.37 (.27, 51)**	**−11.45**	**−38.12**	**2.55**	**7**	**.92**	**0**	**1**	**1.00**
CE model[a]		.44 (.31, .55)	.56 (.45, .69)	1.39	−25.29	15.39	7	.03	12.84	1	.00
E model[a]			1.00	34.58	4.09	50.58	8	.00	48.03	2	.00
			Teacher rated social aggression								
ACE model	.17 (.00, .55)	.23 (.04, .47)	.60 (.45, .78)	−6.43	−29.30	5.57	6	.47			
AE model[a]	.43 (.26, .57)		.57 (.43, .74)	−7.22	−33.90	6.77	7	.45	1.20	1	.27
CE model[a]		.35 (.21, .48)	.65 (.52, .79)	−8.01	−34.69	5.99	7	.54	0.42	1	.52
E model[a]			1.00	12.09	−18.40	28.09	8	.00	22.52	2	.00
Constrained ACE model[a]	**.20 (.12:.27)**	**.20 (.12:.27)**	**.60 (.46, .75)**	**−8.41**	**−.35.09**	**5.59**	**7**	**.59**	**0.02**	**1**	**.85**
			Peer-rated physical aggression								
ACE model	.54 (.20, .67)	.00 (.00, .43)	.46 (.33, .63)	−7.96	−30.16	4.04	6	.67			
AE model[a]	**.54 (.38, .67)**		**.46 (.33, .62)**	**−9.96**	**−35.86**	**4.04**	**7**	**.78**	**.00**	**1**	**1.00**
CE model[a]		.42 (.27, .55)	.58 (.45, .77)	−5.81	−31.71	8.19	7	.31	4.15	1	.04
E model[a]			1.00	17.85	−11.75	33.85	8	.00	29.81	2	.00
			Peer-rated social aggression								
ACE model	.10 (.00, .58)	.35 (.10, .56)	.55 (.40, .71)	−6.87	−29.37	5.13	6	.52			
AE model[a]	.47 (.31, .60)		.53 (.39, .69)	−7.14	−33.39	6.86	7	.44	1.73	1	.19
CE model[a]		.44 (.28, .56)	.56 (.44, .71)	−8.75	−34.99	5.25	7	.63	0.13	1	.72
E model[a]			1.00	16.05	−13.94	32.05	8	.00	26.92	2	.00
Constrained ACE model[a]	**.23 (.15, .30)**	**.23 (.15, .30)**	**.54 (.40, .69)**	**−8.63**	**−34.88**	**5.37**	**7**	**.62**	**0.24**	**1**	**.62**

Note. Best fitting models are in bold. AIC = Aikaike information criterion. BIC = Bayesian information criterion. Confidence intervals for parameter estimates are given in parentheses. Parameters A, B, and C are additive genetic (A), shared environmental (C), and nonshared environmental (E) factors.
[a]Model is compared with its respective ACE model through a χ^2-difference test. $\Delta\chi^2$ − difference test statistics.

associated degrees of freedom, thus correcting for model complexity. In addition, nested χ^2-difference tests were conducted to examine whether the more parsimonious models (i.e., AE, CE, or E) would provide a significantly better or worse fit to the data than the complete ACE model. Table 2 provides a summary of the tested univariate models. We also fitted additional models specifying a dominance genetic effect (D) for social aggression and for physical aggression, separately for teacher and peer ratings. The D path was not significant for both phenotypes and both reporting sources; therefore, these models are not reported here.

As can be seen, the variance of teacher-rated physical aggression was best described by the AE model, with the estimate of the shared environment factor C being equal to zero in the ACE model. In the best fitting model (AE), heritability accounted for 63% of the variance of physical aggression whereas nonshared environmental sources explained the remaining 37% of variance. The variance of social aggression seemed at first glance to be best described by the CE model, but the AE model showed a similarly good fit to the data. As such, it was impossible

to favor either the AE or the CE model over the more complete ACE model. Moreover, the ACE model yielded relatively similar estimates of shared environmental and genetic influences on social aggression, although the contribution of the genetic factor A did not reach statistical significance. We therefore tested an additional ACE model where the relative effects of genetic (A) and shared environmental (C) sources were constrained to be equal. This model yielded the best fit to the data. In this model (constrained ACE), genetic and shared environmental factors each accounted for 20% of the variance of social aggression, whereas the remaining 60% were explained by nonshared environmental sources.

Essentially the same pattern of results was obtained for peer ratings. Specifically, the variance of peer-rated physical aggression was best described by an AE model, with the shared environment factor C turning out to be nonsignificant in the ACE model. In the best fitting model (AE), heritability accounted for 54% of the variance of physical aggression whereas nonshared environmental sources explained the remaining 46% of variance. The variance of social aggression again seemed at first glance to be best

described by the CE model, but the AE model showed a similarly good fit to the data. Again, the ACE model yielded relatively similar estimates of shared environmental and genetic influences on social aggression, although the contribution of the genetic factor A did not reach statistical significance. We therefore tested another ACE model where the relative effects of genetic (A) and shared environmental (C) sources were constrained to be equal. Because this model did not differ significantly from the unconstrained ACE model, it was chosen as the model that best fit the data. In this model (constrained ACE), genetic and shared environmental factors accounted each for 23% of the variance of social aggression, whereas nonshared environmental sources explained the remaining 54% of variance.

Assessing Shared Etiological Factors

In addition to univariate models of variance decomposition, sources of covariation between two phenotypes can be estimated in a multivariate model. Specifically, shared sources of variance can be computed in terms of a genetic correlation (R_G), a shared environment correlation (R_C), and a nonshared environment correlation (R_E), which indicate the extent to which genetic and environmental factors associated with one phenotype overlap with the genetic and environmental factors associated with the other phenotype (for a detailed description, see Neale & Cardon, 1992). A complete correlated factors model based on an ACE–ACE model for two phenotypes, in this case social aggression and physical aggression, is denoted in Figure 2. In the second series of analyses, this correlated factors model was fitted for social aggression and physical aggression, separately for teacher ratings and peer ratings. However, univariate analyses had already indicated that for both teacher ratings and peer ratings, an AE model best explained the variance of physical aggression, whereas an ACE model best explained the variance of social aggression. We thus performed the correlated factors analyses specifying an AE model
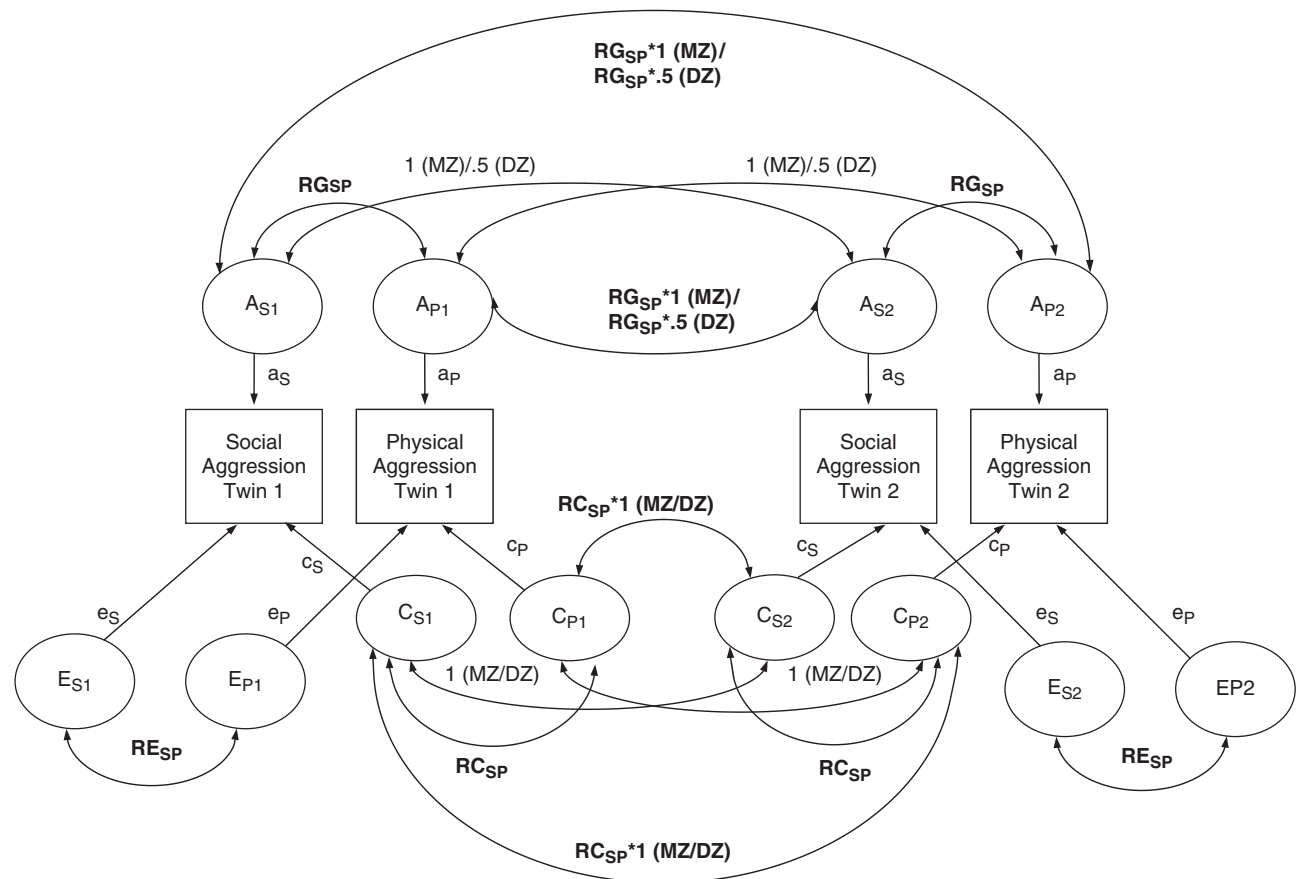

Figure 2. Complete correlated factors genetic model based on an ACE – ACE model for two phenotypes. R_G refers to the genetic correlation, which represents the overlap of genetic influences across phenotypes. R_C refers to the shared environment correlation, which represents the overlap of shared environment influences across phenotypes. R_E refers to the nonshared environment correlation, which represents the overlap of nonshared environment influences across phenotypes.

Table 3
Multivariate Models of Physical Aggression and Social Aggression

	R_G	R_E	$p\ X_1 \Rightarrow X_2$	AIC	BIC	χ^2	df	p
			Teacher ratings					
Correlational model	.79 (.37, 1.00)	.31 (.13, .48)		− 28.84	− 114.42	9.17	19	.97
Directional models								
PA ⟺ SA			p PA ⇒ SA .36 (.12, .83)	− 24.99	− 101.57	9.01	17	.94
			p SA ⇒ PA .17 (− 1.13, .76)					
PA ⇒ SA			p PA ⇒ SA .45 (.33, .65)	− 26.84	− 107.91	9.17	18	.96
			p SA ⇒ PA .00					
			Peer ratings					
Correlational model	1.00 (.50, 1.00)	.12 (− .08, .32)		− 24.49	− 108.43	3.51	19	.81
Directional models								
PA ⟺ SA			p PA ⇒ SA .53 (.48, 1.54)	− 21.06	− 96.17	12.94	17	.74
			p SA ⇒ PA .32 (− 1.00, .86)					
PA ⟺ SA			p PA ⇒ SA .73 (.44, 1.12)	− 22.49	− 102.02	13.52	18	.76
			p SA ⇒ PA .00					

Note. All models are based on a multivariate AE – ACE model for physical and social aggression; parameters A, B, and C are additive genetic (A), shared environmental (C), and nonshared environmental (E) factors. R_G is the genetic correlation, which indicates the overlap of the influence of the latent factor A on the two phenotypes; R_E is the nonshared environment correlation, which indicates the overlap of the influence of the latent factor E on the two phenotypes. The shared environment correlation R_C is necessarily zero given that the underlying model for physical aggression is an AE model. $p\ X_1 \Rightarrow X_2$ indicates the directional effect from one phenotype to the other. PA = physical aggression. SA = social aggression. Confidence intervals for parameter estimates are given in parentheses.

for physical aggression and an ACE model for social aggression, with a genetic correlation (R_G) and a nonshared environment correlation (R_E) as the two estimates of overlap (see Figure 2). Because there was no shared environment contribution for physical aggression, the shared environment correlation (R_C) was therefore necessarily zero in the correlated factors model.

The results reported in Table 3 indicate a good fit of this model for both teacher ratings and peer ratings. For teacher ratings, there was a strong correlation between the genetic factors contributing to physical and social aggression, $R_G = .79$ (.37–1.00 CI), but a much more modest correlation between the nonshared environmental factors contributing to physical and social aggression, $R_E = .31$ (.13–.48 CI). In other words, ($R_G^2 =$) 62% of the genetic factors influencing social aggression and physical aggression seem to be the same according to teacher ratings, whereas there is only a ($R_E^2 =$) 9% overlap of unique environmental factors influencing the two types of aggression. An even more extreme picture emerged for peer ratings. Here, the correlation between the genetic factors was estimated to be perfect, $R_G = 1.00$ (.50–1.00 CI), whereas the correlation between the nonshared environmental factors was weak and did not reach statistical significance, $R_E = .12$ (−.08–.32 CI). As such, according to peer ratings, social aggression and physical aggression share 100% of their genes but no overlapping envi-

ronmental influences. In sum, these results indicate that the correlation between social aggression and physical aggression in kindergarten children may be attributable mainly to overlapping genes and only to a small extent, if at all, to overlapping environmental conditions. As mentioned previously, however, the link between physical and social aggression may not only be explained by shared etiological factors, as indicated in a correlational model, but also by a direct phenotype-to-phenotype effect. This possibility was tested in the next series of analyses.

Phenotype-to-Phenotype Contribution Models

In traditional single-birth sample designs, correlation or covariance matrices based on longitudinal data can be used to assess directional (i.e., unidirectional or reciprocal) effects between two phenotypes. These directional effects can be specified either (a) as cross-time (i.e., cross-lagged) directional effects or (b) as more instantaneous directional effects between the two phenotypes (see the longitudinal model with instantaneous directional effects in Figure 3; Neale & Cardon, 1992). The latter approach can be considered a special case of the traditional longitudinal directional effects model and is ideal to illustrate the basic logic of the cross-sectional genetic directional model described next. In a longitudinal design with two variables X and Y assessed at two time points, the directional path a denoted in
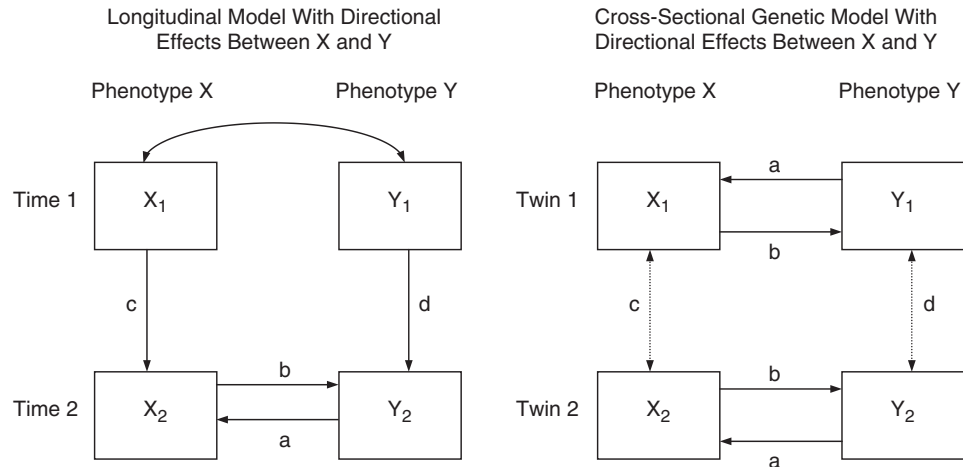
Figure 3. Longitudinal directional design and cross-sectional genetic directional design.

Figure 3 is a function of the correlation between Y1 and Y2, the correlation between X2 and Y2, and the correlation between Y1 and X2. Similarly, the directional path b is a function of the correlation between X1 and X2, the correlation between X2 and Y2, and the correlation between X1 and Y2. The assessment of the directional paths a and b is possible because the patterns of correlations or covariances across time and across measures are not necessarily symmetrical. That is, the correlation between X1 and Y2 is not necessarily identical to the correlation between X2 and Y1. Moreover, the correlations between X1 and X2 may differ from the correlations between Y1 and Y2. Given this potential asymmetry, the cross-phenotype contributions in one direction or the other (or both) can be compared in standard structural equation models.

In a similar fashion, cross-sectional data from pairs of relatives can be used to assess cross-phenotypic contributions at a single measurement time (for a detailed description of the cross-sectional genetic directional model, see Neale & Cardon, 1992). The statistical strategy is similar to the one used in the single-birth longitudinal design: A path analysis model is fit where measures on both twins of a pair, respectively, substitute for Time 1 and Time 2 measures of a longitudinal model (see cross-sectional genetic model with directional effects in Figure 3). The inference of a unidirectional phenotypic contribution between two phenotypes rests on the asymmetry of the cross-twin/cross-phenotype correlations. Specifically, the correlation between phenotype X in Twin 1 and phenotype Y in Twin 2 is not equal to the correlation between phenotype Y in Twin 1 and phenotype X in Twin 2 if different directional paths exist between phenotypes. Note that

paths c and d, which estimate the cross-time stability within phenotypes in the longitudinal model, are represented in the cross-sectional genetic model by the cross-twin correlations within phenotypes. The cross-sectional genetic directional model also assumes that the directional relationship between the two phenotypes is the same for both twins (i.e., paths a and b are equal for Twins 1 and 2), whereas the relationship between the two phenotypes in the longitudinal model is typically expressed as a directional path only at Time 2 but as a correlational link at Time 1.

To assess whether a directional model can provide an alternative explanation of the positive correlation between social and physical aggression, a structural equation model specifying reciprocal paths between social and physical aggression was fitted first. As was the case for the correlational model, the phenotypic contribution model specified an ACE model for social aggression and an AE model for physical aggression (see univariate models). Figure 4 illustrates the reciprocal phenotypic contribution model for social and physical aggression. Because such models can be sensitive to measurement error specifications, a separate measurement error parameter (R) was also specified in the model (Neale & Cardon, 1992). Notably, the measurement error estimate was equal to 0 in models specifying measurement errors to be equal across measures as well as in models with phenotype-specific measurement error where degrees of freedom permitted it. As can be seen in Table 3, the phenotypic contribution model showed an equally good fit to the data as the correlational model, both for teacher ratings and for peer ratings of aggression. Inspection of the directional paths revealed, however, that only the path from physical
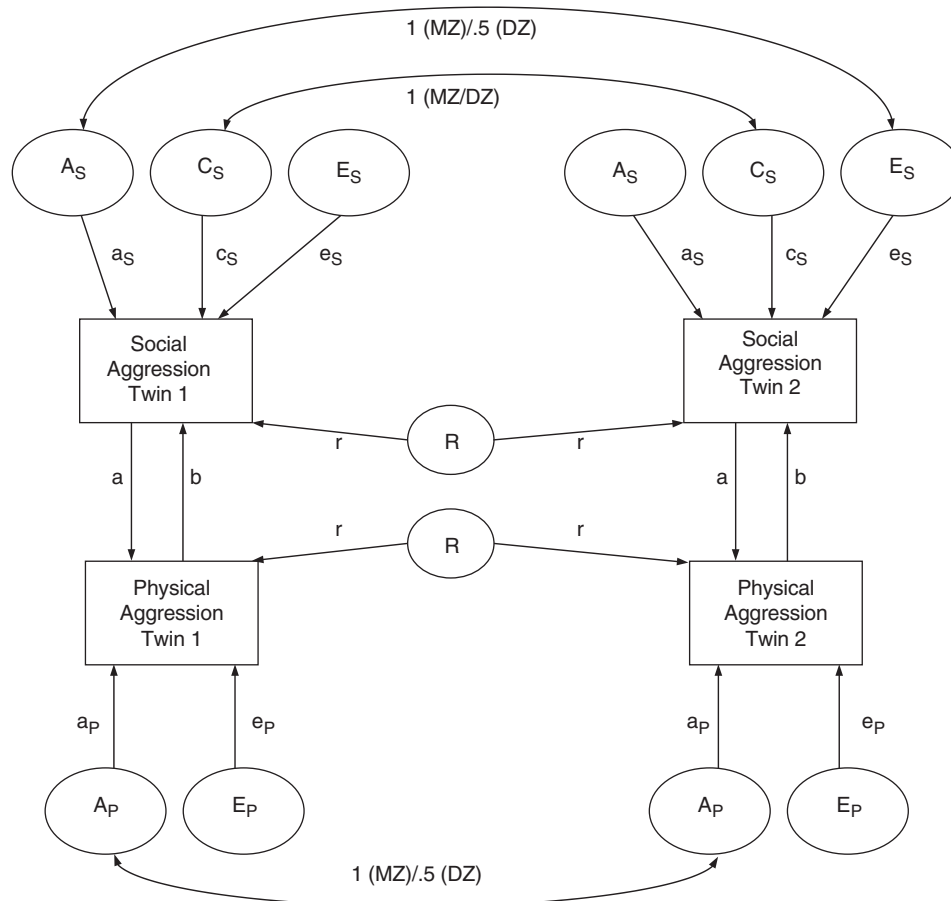
Figure 4. Phenotype-to-phenotype effects model. Paths a and b indicate the direction and size of effects between phenotypes (possible values between −1 and 1). R is the measurement error.

to social aggression was statistically significant, both for teacher ratings and for peer ratings of aggression. We therefore tested an additional unidirectional model where the path from social to physical aggression was fixed to zero. This model did not differ significantly from the previous model based on χ^2-difference tests: teacher ratings, $\chi^2(1) = 0.16$, $p = .69$, and peer ratings, $\chi^2(1) = .58$, $p = .45$. Directional links between the two types of aggression thus appear to be based entirely on a phenotypic contribution from physical to social aggression, p PA $\Rightarrow$ SA = .44 (.33–.65 CI) for teacher ratings, and p PA $\Rightarrow$ SA = .73 (.44–1.12 CI) for peer ratings.

Discussion

The goals of the present study were to examine (a) to what extent the variance of social aggression, compared with physical aggression, is explained by genetic, shared environmental, or nonshared environmental factors, and (b) to what extent the covariance between physical aggression and social aggression is explained either by the same underlying genetic or shared/nonshared environmental factors or by direct effects from one type of aggression to the other. These questions were examined comparing results obtained from teacher ratings and peer ratings of aggression.

Genetic and Environmental Influences on Physical and Social Aggression

Although many children in our study were rated as nonaggressive, we found observable individual differences in social aggression as well as in physical aggression at 6 years of age, both when based on teacher ratings and when based on peer ratings. In this respect, our findings corroborated previous research (e.g., Crick et al., 1997; Hart et al., 1998; McNeilly-Choque et al., 1996) indicating that even relatively young children are capable of using complex manipulative strategies in addition to physical

means to harm their peers. Where does this aggressive behavior come from? Similar to findings from previous studies on physical aggression in kindergarten and school-age children (e.g., Cadoret et al., 1997; DiLalla, 2002; Miles & Carey, 1997; Rhee & Waldman, 2002), our results indicated that approximately 50% to 60% of the variance of physical aggression was explained by heritable factors and the rest was attributable to unique environmental effects. Even more pronounced environmental effects, however, were revealed with respect to social aggression. In the best fitting model, genetic effects accounted for only around 20% of the variance of social aggression, another 20% was explained by shared environmental influences, and about 60% was explained by unique environmental factors. The present study is thus the first to show that, in line with suggestions by DiLalla (2002), social aggression seems to be determined to a lesser extent by genetic factors and to a greater extent by environmental factors than physical aggression. This pattern of results holds regardless of whether it is based on teacher or peer ratings of social and physical aggression.

As in previous studies (e.g., Crick & Grotpeter, 1995; Vaillancourt et al., 2003), social aggression and physical aggression showed a significant, albeit moderate, amount of overlap. To account for the observed correlation, we first examined the possibility of shared origins of social aggression and physical aggression by examining the correlations between the genetic factors and between the unique environmental factors influencing each type of aggressive behavior. This model offered an excellent fit to the data and showed that the positive link between physical and social aggression can be explained mostly by overlapping genes but only to a small extent, if at all, by overlapping environmental conditions. The substantial overlap of genetic factors influencing physical and social aggression could reflect heritable biological and physiological components that predispose some children to aggressive behavior in general. Such genetic liabilities may be evidenced in biological markers of a reactive, irritable temperament and difficulties in emotion regulation and self-control (Bates et al., 1991; Caspi et al., 1995). The specific form of aggression used, however, seems to depend on the degree of children's exposure to certain environmental conditions, which show only little commonality between physical aggression and social aggression.

Among the main environmental contributors to child aggression are parental behaviors (e.g., Dishion, 1990). A parental behavior that could be particularly relevant for the development of social—as opposed to physical—aggression in the child is psychological control (Barber, 1996). Psychological control includes relationally manipulative behaviors such as love withdrawal or induction of shame and guilt, which are highly similar to the manipulative tactics that define social aggression (e.g., social exclusion, threat of friendship withdrawal). Experiencing parental psychological control may thus foster the development of social aggression in the child. Empirical evidence for an effect of parental psychological control on child social aggression is still lacking, although other negative parenting behavior such as coercion and lack of responsiveness have been linked to social aggression in children (Hart et al., 1998). Of course, parents are not the only source of environmental influence on child behavior. Already among preschoolers, prolonged interaction with aggressive peers has been related to an increase in observed and teacher-rated aggression over a 3-month interval (Snyder, Horsch, & Childe, 1997). A particularly important source of peer influence on children's aggressive behavior may be deviancy training through modeling and reinforcement from aggressive friends, which has been observed in children as young as 5 years of age (Snyder et al., in press). Empirical evidence for the specific effect of socially aggressive friends on children's own social aggression comes from a recent study with second through fourth graders by Werner and Crick (2004). These authors showed that a higher level of reciprocal friends' social aggression predicted increases in social aggression, whereas a higher level of reciprocal friends' physical aggression predicted increases in physical aggression.

Notably, parental influence has been traditionally viewed as a part of the latent C (i.e., shared environment) component. Some parental behaviors, such as psychological control directed to the spouse and observed by both twins, may indeed affect both children in a family equally and thus fall in this category. Conversely, parental psychological control directed to the child may differ enough between twins in one family that each child experiences the parental behavior in a unique fashion, thus reflecting unique environmental effects on child behavior.

Similar to parental influences, peers can also represent a shared as well as a nonshared environmental influence, for example, depending on whether twins in a pair affiliate with the same friends. Members of twin pairs who are in different classrooms might have more opportunities to form unique, independent friendship relations with their respective classmates than twins who are in the same classroom. The comparison of twin pairs from the

same and from different classrooms in future studies, along with measures of friends' level of aggressiveness, may help clarify the relative contribution of shared and nonshared peer environment on children's social aggression.

Toward a Developmental Model of Physical Aggression and Social Aggression

Apart from common underlying genetic and (to a limited extent) environmental factors, our results suggest that the overlap between social aggression and physical aggression can also be explained by a directional effect. Specifically, a high level of physical aggression seems to lead to a high level of social aggression, whereas the opposite effect does not seem to be true. Again, this pattern of results held for both teacher ratings and peer ratings of aggression. The finding of a directional effect from physical aggression to social aggression is in line with the developmental sequence of aggressive behavior proposed by Bioerkqvist, Lagerspetz, et al. (1992). Specifically, some children may exhibit strong aggressive tendencies already at an early age, which initially will be expressed through physical means. As suggested by the present findings, these (initially physically) aggressive tendencies may be, to a large extent, genetically based. Physical aggression is generally not socially accepted, however, and often leads to punishment. At the same time, socializing agents such as parents or peers may foster an alternative yet similarly aggressive strategy, namely social aggression, either by direct modeling or through reinforcement. As such, many children who initially display physically aggressive behavior may soon learn to use socially more acceptable and less risky aggressive strategies.

Our finding that physical and social aggression seem to share most if not all genetic components is also in line with the notion that a generalized, largely genetically driven individual disposition for aggressive behavior may shift from physical to social aggression as children mature (Bjoerkqvist, Lagerspetz, et al., 1992). Whether and when this shift occurs, however, seems to be determined by the extent to which the child is exposed to a social environment that specifically promotes the use of social aggression. The potentially crucial role of the environment in the extent and timing of the development shift in aggressive behavior may also explain the present finding that social aggression is determined to a larger extent than physical aggression by environmental factors. However, because the use of social aggression requires sufficient verbal and cognitive skills that gradually develop over early and middle childhood, this developmental shift is likely a gradual one. This gradual change from physical to social aggression might explain why, at least in middle childhood, many children still exhibit both types of aggression.

Strengths, Limitations, and Conclusion

This study is the first to assess the question of heritability versus environmental effects on social aggression. By the same token, this study presents a new way of examining the mechanisms underlying the association between social aggression and physical aggression. A main advantage of the present study is it assessed behavior not only by teachers but also by peers, who are rarely employed as a reporting source in twin studies. It is interesting that essentially the same results were obtained despite the commonly found moderate concordance between teacher ratings and peer ratings of social and physical aggression.

Our study also has several limitations, however, that need to be kept in mind when interpreting the results. The most obvious limitation is the small sample size, which precluded the examination of sex differences with regard to genetic and environmental effects on social versus physical aggression. The high costs associated with sociometric peer ratings in 409 classrooms of young children unable to read, however, rendered the assessment of a larger sample difficult. Nevertheless, future studies need to replicate the present findings using larger samples before definite conclusions can be drawn with regard to the relative contribution of genetic versus environmental effects on social aggression and potential sex differences in this context. In addition, caution needs be taken in trying to generalize the results beyond the assessed age. Physical aggression is already diminished at school entry (Nagin & Tremblay, 2001) whereas social aggression may not be fully developed until age 8 (Bjoerkqvist, Lagerspetz, et al., 1992). Moreover, some previous studies on aggression and antisocial behavior have suggested that both genetic and shared environmental effects diminish with age, whereas the magnitude of nonshared environmental influences increases (Rhee & Waldman, 2002). As such, the mechanisms explaining social aggression and its link with physical aggression that are identified in adolescence may differ from those present by the end of early childhood, when that behavior first develops.

Despite its limitations, we believe the present study demonstrates that genetic designs can make a

substantial contribution to the question of the etiology of social aggression compared with physical aggression. Specifically, by revealing the importance of environmental influences on social aggression, the present study paves the way for future studies aimed at isolating the effects of particular environmental variables on this type of aggressive behavior. Moreover, multivariate models such as the phenotype-to-phenotype model allowed testing theoretical assumptions regarding the overlap of social aggression with physical aggression even when using cross-sectional data. Nevertheless, longitudinal genetic data are preferable to provide a clear picture of the mechanisms underlying the association and developmental trends between the two types of aggression. The present results have important implications for preventive interventions as they suggest that reducing physically aggressive behavior at an early age might also help prevent the development of social aggression. In this context, it will be important to identify in future studies the putative moderating factors that might qualify the pathway from physical to social aggression. Identification of such moderating conditions will help improve the existing preventive intervention programs aimed at reducing aggressive behavior in all its forms.

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