Kindergarten Children’s Genetic Vulnerabilities Interact With Friends’ Aggression to Promote Children’s Own Aggression

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ABSTRACT

Objective: To examine whether kindergarten children’s genetic liability to physically aggress moderates the contribution of friends’ aggression to their aggressive behaviors. Method: Teacher and peer reports of aggression were available for 359 6-year-old twin pairs (145 MZ, 212 DZ) as well as teacher and peer reports of aggression of the two best friends of each twin. Children’s genetic risk for aggression was based on their cotwin’s aggression status and the pair’s zygosity. Results: Children’s aggression was highly heritable. Unique environment accounted for most of the variance in friends’ aggression, although there was also a small genetic contribution (15%). Both genetic liability to aggression and having aggressive friends predicted twins’ aggression. However, the contribution of aggressive friends to children’s aggression was strongest among genetically vulnerable children. This result was similar for boys and girls, despite sex differences in both aggression and the level of aggression of friends. Conclusions: Affiliation with aggressive friends at school entry is a significant environmental risk factor for aggression, especially for children genetically at risk for aggressive behaviors. Developmental models of aggression need to take into account both genetic liability and environmental factors in multiple settings, such as the peer context, to more precisely describe and understand the various developmental pathways to aggression. The implications for early prevention programs are discussed. J. Am. Acad. Child Adolesc. Psychiatry, 2007;46(8):1080–1087. Key Words: twins, aggression, friends’ aggression, genotype-environment interaction.

Meta-analyses have shown that the heritability of children’s antisocial behaviors is moderate to high in magnitude (Rhee and Waldman, 2002). Until recently, analyses of the heritability of antisocial behaviors typically omitted environmental measures, thus neglecting the possibility that Genotype–Environment transactions could account for such behaviors (Kendler and Eaves, 1986; Moffitt, 2005). Recent studies, using adoptee or twin designs, have shown that the genetic liability to antisocial behavior moderates the influence of environmental stimuli through genotype × environment interactions (G×E; Button et al., 2005; Jaffee et al., 2005). For example, the contribution of physical maltreatment before or at age 5 to the prediction of severe conduct problems at age 7 was found to vary as a function of the genetic risk for conduct problems (Jaffee et al., 2005).
There are limited data for G×E among young children, and this evidence is essentially based on environmental factors within the family context, such as maltreatment by parents or family dysfunction. However, children’s early aggression is also subjected to the possible influence of peers (Boivin et al., 2005). For instance, the propensity of aggressive children to affiliate with aggressive friends has been reported as early as in kindergarten (Snyder et al., 1997). This tendency could reinforce aggressive behaviors as a means to reach personal and social goals and account for the shaping of current and future antisocial behaviors (Dishion et al., 1995).

Aggressive behaviors are frequent among kindergarten children (NICHD Early Child Care Research Network, 2004); however, only 3% to 10% of the children will develop chronic high levels of aggression (Broidy et al., 2003; NICHD Early Child Care Research Network, 2004). It could be that the vulnerability to early negative peer influences is conditioned by young children’s genetic susceptibility to aggression. Accordingly, the main goal of this study was to examine whether the genetic liability to physical aggression among kindergarten children moderates the contribution of aggressive friends to their physical aggression.

To clearly identify G×E interactions, it is important to first document the extent to which individuals evoke or select features of their environment, such as having aggressive friends, as a function of heritable traits, a phenomenon identified as gene–environment correlation (Moffitt et al., 2005; Scarr and McCartney, 1983). Small heritability has been reported for teacher-rated deviant peer affiliation of 6- to 14-year-old twins (h² = 18%), but not for teacher-rated friends’ externalizing behavior and observational ratings of deviant friendships (Bulloch et al., 2006; see also Rose, 2002). No study has yet examined whether having aggressive friends could be associated to genetic factors at an early age. Thus, the second goal of this study was to ascertain whether genetic factors in the child accounted for his or her friends’ aggression.

Finally, because boys are more aggressive (Archer and Côte, 2005) and associate with more aggressive friends than do girls at age 6 (van Lier et al., 2005a), we also examined whether the putative G×E would apply more to boys than to girls.

### Method

#### Subjects

Participants were part of an ongoing longitudinal study of a population-based sample of twins from greater Montreal, Canada (Quebec Newborn Twin Study [QNTS]) who were recruited at birth between November 1995 and July 1998 (N = 648 twin pairs). Zygosity of same-sex twin pairs was assessed through detailed ratings at 18 months (Goldsmith, 1991) and confirmed in 94% by genotyping (Forget-Dubois et al., 2003). Most families (84%) were of European descent. The sample was followed longitudinally at 5, 18, 30, 48, and 60 months. The present study was conducted at the end of kindergarten (sixth wave), when children were on average 72.7 months old (SD 3.6). Complete data were available for 357 twin pairs. Compared with the original sample, these 357 twin pairs were more likely to be monozygotic (MZ) twins, and their parents/caretakers would have on average a higher income (145 MZ pairs, 48% males, 99 same-sex dizygotic [DZ] pairs, 51% males, and 113 opposite-sex pairs).

#### Measures and Procedures

All of the assessments were administered in the spring of kindergarten. Parental consent was obtained for all of the participating twins, as well as for children who acted as respondent (see below). All of the procedures were approved by the ethics review boards of Laval University and St.-Justine Hospital, as well as by the school boards of the participating schools.

Teacher-rated aggression during kindergarten was assessed using five items dealing with physical aggression and bullying from the Child Social Behavior Questionnaire (Tremblay et al., 1991): got into fights, scared other children to get what he or she wanted, physically attacked others, tried to dominate others, and hit, kicked, or bit others in the past 6 months. Variants of this scale have been shown to predict high aggression, delinquency, and conduct disorders in adolescence (Broidy et al., 2003; Côté et al., 2001; Nagin and Tremblay, 1999). All of the items were scored on a 3-point scale (0 = never, 1 = sometimes, 2 = often). Cronbach’s α was .82.

Peer-rated physical aggression of the twins was assessed using booklets containing photographs of all of the children in a given class. The children were asked to circle the photographs of up to two classmates who best fit several descriptors, including gets into fights and hits, bites, or kicks others. Because they are based on nominations from many respondents, nominations on these items are highly reliable and valid and have been found to predict later conduct disorder, school dropout, and delinquency (Rubin et al., 1998; van Lier et al., 2005a). To optimize the respondent time, the peer assessment of aggression was limited to two items. The total number of nominations received for each aggression item was standardized and the two scores then added to a total score. The correlation between the two items scores was 0.77.

**Friends’ Aggression.** Children were asked to nominate up to four children they considered their friends and to rank order them from the very best friend to the fourth best friend. Teachers scored aggression of each of the target twin’s friends by rating on a 3-point scale (0 = never, 1 = sometimes, 2 = often) how likely each friend in the past 6 months was to physically attack others, try to dominate others, and scare others to get what he or she wanted. The number of items was restricted to three to lighten the task for teachers while ensuring good reliability. Because of our focus on central friends, the friendship nominations of the two most important friends were examined. 

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were considered. Cronbach’s \( \alpha \) was .85 for the aggression score of the first nominated friend and .90 for the second friend. As for the twins’ assessment, peer nominations were also used to assess the friends’ aggression. The two best friends’ physical aggression scores were then averaged.

The teacher and peer assessments of the twins’ aggression were significantly related \( (r = 0.53; p < .01) \) as were the teacher and peer assessment of friends’ aggression \( (r = 0.43; p < .01) \). The standardized teacher and peer assessments were standardized again and summed to a composite twin’s aggression score and a composite friends’ aggression score.

**Statistical Analyses**

We first assessed the genetic–environmental etiology of twin’s physical aggression through the use of ACE models. The ACE model decomposes the variance of aggression into additive genetic (A), shared environmental (C), and nonshared environmental factors (E; Neale and Cardon, 1992). Three criteria were used to select the best fitting and most parsimonious model: the \( \chi^2 \) goodness of fit statistic; the root mean square error of approximation statistic (RMSEA), which gives an index of model discrepancy per degree of freedom (Brown and Cudeck, 1993); and Akaike’s index of parsimony. Lower values are indicative of a better and more parsimonious fit. The ACE models were analyzed using Mx (Neale et al., 2003).

A genetic risk index was computed through combining the cotwin’s risk status and the pair’s zyosity. Such a procedure was proved effective in assessing genetic liability for highly heritable traits such as aggression (Jaffe et al., 2005; Kendler and Kessler, 1995; Moffitt, 2005). A target twin’s genetic risk was considered highest if his or her MZ cotwin was at or above the 80th sex-specific percentile of the composite score of aggression, a cutoff that has been previously reported as an indication of risk (e.g., Achenbach and Rescorla, 2001). The target twin’s genetic risk was considered moderately high if his or her DZ twin was at or above the 80th sex-specific percentile on aggression. It was considered moderately low if his or her DZ twin was below the 80th percentile on aggression. Finally, the target twin’s genetic risk was considered lowest if his or her MZ twin was low on aggression. The genetic risk index was coded on a 0 to 3 ordinal scale, with 0 representing lowest risk and 3 representing highest genetic risk.

Although 70% of the twins were in different classrooms, nonindependence of the data was likely. We estimated whether the tests on G×E using standard regression were affected by nonindependence of the data by using a sandwich estimator (Williams, 2000).

**RESULTS**

**Descriptive Statistics**

Mean (SD) aggression scores were 0.43 (1.84) for males and −0.66 (1.07) for females \( (F_{1,713} = 93.47; p < .001) \). Mean friends’ aggression scores were 0.19 (0.77) for males and −0.39 (0.49) for females \( (F_{1,689} = 141.02; p < .001) \). Forty-eight MZ twins (42% males) and 95 DZ twins (40% males) were considered at high risk, whereas 242 MZ (49% males) and 329 DZ twins (53% males) were at low risk. Logistic regression analyses revealed that MZ and DZ twins were equally likely to score at or above the 80th percentile on physical aggression (odds ratio 1.3; 95% confidence interval 0.88–1.95).

School authorities in Quebec tend to separate twins at school entry, although not systematically. Consequently, the majority of the twins (70%) were in different classrooms. This proportion was similar for MZ and DZ twin pairs \( (\chi^2_1 [N = 357] = 0.00; p > .05) \). No differences in aggression were found between twins who shared or did not share classrooms \( (F_{1,713} = 1.43; p > .05) \). Because friendship nominations were limited to classrooms, sharing friends was only possible among twins that shared classrooms (106 pairs). Thirty-five of these pairs shared first or second best friend, but only five pairs shared both friends, which was similar for MZ and DZ twins \( (\chi^2_1 [N = 357] = 2.81; p > .05) \). Friends’ aggression scores were not available for 25 twins because parents of the friends did not agree to their participation. Twins for which a friends’ aggression score was missing did not differ from the other twins with respect to sex \( (\chi^2_1 [N = 714] = 2.28; p > .05) \) or aggression \( (F_{1,713} = 3.33; p > .05) \).

**Validity of the Genetic Risk Index**

**Genetic–Environment Etiology of Twins’ Physical Aggression at 72 Months.** The validity of the genetic risk index requires that individual differences in aggression are substantially accounted for by genetic factors. The intraclass correlations (ICCs) for MZ (ICC 0.70; \( p < .001 \)) versus DZ twins (ICC 0.18; \( p < .05 \)) suggested this was the case. Because we included opposite-sex twins, the genetic modeling of twins’ aggression used sex-specific covariance matrices to allow for possible sex differences in variances and means in aggression. Full ACE and nested models were tested, considering both sex-specific and sex-invariant variances and means. Table 1 shows that the best fitting model was an AE model in which the variances across sexes were held equal and 66% of the variance was accounted for by genetic factors. The significant deterioration of subsequent models, in which the genetic parameter was fixed at 0 for males and females (E model), indicated that the genetic parameter was significant for both males and females (Table 1). This finding was a first condition for examining the putative G×E interaction.
Twins’ Friends Aggression.

MZ and DZ twins did not differ in their friends’ aggression (mean 0.01, SD 1.70 for MZ; mean 0.02, SD 1.63 for DZ; \( F_{1,689} = 0.59; p > .05 \)). The validity of our testing of a G×E rests on the assumption that the putative environmental factor, friends’ aggression, is minimally associated with genetic factors and thus is a genuine environmental factor. Accordingly, ACE and nested models were fitted for friends’ aggression, again using sex-specific means and variance differences across twins’ sex (78% of males and 79% of females had same-sex friends; allowing for sex-specific means and variances takes into account mean differences based on friend sex). The best fitting model revealed a significant but marginal genetic effect (15%) for both males and females. Furthermore, the correlation between the genetic risk index and friends’ aggression was zero, indicating that friends’ aggression is unrelated to the genetic liability of the twins. Therefore, the data met the conditions for the further investigation of G×E.

TABLE 1
Estimates of Genetic and Environmental Parameters for Best Fitting Models (Sex-Invariant AE) and Nested Sex-Specific and Sex-Invariant Models

<table>
<thead>
<tr>
<th>Parameter Estimates</th>
<th>Males</th>
<th>Females</th>
<th>Fit Indices</th>
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<tbody>
<tr>
<td></td>
<td>A</td>
<td>C</td>
<td>E</td>
</tr>
<tr>
<td><strong>Aggression</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE (sex invariant)</td>
<td>0.66</td>
<td>—</td>
<td>0.34</td>
</tr>
<tr>
<td>E-AE (male A = 0)</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>AE-E (female A = 0)</td>
<td>0.67</td>
<td>—</td>
<td>0.33</td>
</tr>
<tr>
<td>E (sex invariant A = 0)</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td><strong>Friends’ aggression</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE (sex invariant)</td>
<td>0.15</td>
<td>—</td>
<td>0.85</td>
</tr>
<tr>
<td>E-AE (male A = 0)</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>AE-E (female A = 0)</td>
<td>0.26</td>
<td>—</td>
<td>0.74</td>
</tr>
<tr>
<td>E (sex invariant A = 0)</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
</tbody>
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*Note: All of the models estimate sex-specific means, constraining means to be equal across sexes significantly worsened the fits of all models (not shown). The best fitting model for aggression and friends’ aggression is highlighted in bold. A = additive genetic factors; C = shared environmental factors; E = nonshared environmental factors or measurement error; RMSEA = root mean square error of approximation; AIC = Akaike’s index of parsimony.
* \( p < .001 \).

Twins’ Friends Aggression. MZ and DZ twins did not differ in their friends’ aggression (mean = 0.01, SD 1.70 for MZ; mean 0.02, SD 1.63 for DZ; \( F_{1,689} = 0.59; p > .05 \)). The validity of our testing of a G×E rests on the assumption that the putative environmental factor, friends’ aggression, is minimally associated with genetic factors and thus is a genuine environmental factor. Accordingly, ACE and nested models were fitted for friends’ aggression, again using sex-specific means and variance differences across twins’ sex (78% of males and 79% of females had same-sex friends; allowing for sex-specific means and variances takes into account mean differences based on friend sex). The best fitting model revealed a significant but marginal genetic effect (15%) for both males and females. Furthermore, the correlation between the genetic risk index and friends’ aggression was zero, indicating that friends’ aggression is unrelated to the genetic liability of the twins. Therefore, the data met the conditions for the further investigation of G×E.

Gene–Environment Interaction
The results that are germane to G×E are presented in Table 2. Sex (being male), genetic risk, and friends’ aggression independently contributed to twins’ aggression. To test for G×E, the product term between genetic risk and friends’ aggression was added. This interaction term was significant.

To illustrate this interaction, the level of aggression for each of the four risk categories was plotted as a function of friends’ aggression. As shown in Figure 1, twins’ aggression was significantly higher as both genetic risk and friends’ aggression increased. However, this increase was more marked for twins with the highest genetic risk (MZ low genetic risk: \( \beta = .15; p = .02 \) for the lowest genetic risk [coded 0]; \( \beta = .25; p = .05 \) for moderately low genetic risk; \( \beta = .22; p = .001 \),

TABLE 2
Results of Multiple Regression Analyses for Variables Predicting Children’s Physical Aggression

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>( \beta )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex (male)</td>
<td>1.16</td>
<td>0.12</td>
<td>.34*</td>
</tr>
<tr>
<td>Genetic risk</td>
<td>0.72</td>
<td>0.06</td>
<td>.36*</td>
</tr>
<tr>
<td>Friends’ aggression</td>
<td>0.11</td>
<td>0.04</td>
<td>.11*</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex (male)</td>
<td>1.15</td>
<td>0.12</td>
<td>.33*</td>
</tr>
<tr>
<td>Genetic risk (G)</td>
<td>0.72</td>
<td>0.06</td>
<td>.36*</td>
</tr>
<tr>
<td>Friends’ aggression (E)</td>
<td>0.01</td>
<td>0.05</td>
<td>.01</td>
</tr>
<tr>
<td>G×E</td>
<td>0.12</td>
<td>0.04</td>
<td>.14*</td>
</tr>
</tbody>
</table>

*Note: \( R^2 = 0.27 \) for Step 2.
* \( p < .001 \).
for moderately high genetic risk; and $\beta = .39; p = .01$ for the highest genetic risk [coded $4$]). When sex was added in a three-way interaction to test for sex differences in G×E, it did not add significantly to the prediction ($\beta = .10; p = .25$), indicating that the G×E stood for both males and females.

To assess the robustness of the G×E, we ran a number of tests. We first tested whether the G×E findings hold for teacher and peer ratings separately. Despite the loss of power and precision (i.e., aggregating indicators generally yields a more reliable estimate), significant G×E effects were found for both teacher-rated aggression ($\beta_{g \times e} = .11; p < .01$), and peer-rated aggression ($\beta_{g \times e} = .05; p < .10$).

Second, because of the sex differences in aggression and the possible distortions associated with the inclusion of opposite-sex DZ pairs, G×E analyses were performed on same-sex pairs only. The results remained the same ($\beta_{g \times e} = .12; p < .01$).

Third, to ascertain the direction of the G×E process, we tested whether genetic risk moderated the contribution of twins’ aggression to their friends’ aggression, which it did not. The moderation thus seems to operate on friends’ aggression in predicting twin’s aggression rather than the reverse.

Fourth, we tested whether the findings would stand using reciprocal nominations to qualify friendship ($n = 471$) in one test and excluding twins whose best friend was of the opposite sex (19% of the cases, $n = 579$) in a second test. In both cases, the G×E finding persisted ($\beta_{g \times e} = .15, p < .01, \beta_{g \times e} = .12; p < .01$, respectively).

Finally, we also explored whether the nonindependence of the data accounted for the results by adjusting SEs for clustering of the data using a sandwich estimator, assuming that all of the twin pairs shared the same classroom. Despite this conservative approach, the G×E effect remained ($\beta_{g \times e} = .14, SE = .05; p < .05$).

**DISCUSSION**

High levels of aggression in early elementary school predict chronically high aggression through adolescence and related maladjustment problems such as delinquency (Broidy et al., 2003; Nagin and Tremblay, 1999). The goal of this study was to examine whether the genetic liability to aggression moderated the association between having aggressive friends and displaying aggression among kindergarten children. We found that both genetic liability and having aggressive friends were generally associated with aggression, a finding consistent with previous research that explored each of these phenomena separately (Rhee and Waldman, 2002; Snyder et al., 1997). However, as hypothesized, these two factors combined nonlinearly in predicting aggressive behaviors; that is, high levels of aggression in kindergarten children were most likely when the most genetically vulnerable children interacted with aggressive friends.
This finding suggests that children who are genetically vulnerable to aggression are more susceptible to the processes of influence associated with interacting with aggressive peers (e.g., the reinforcement of deviant behavior and the learning from deviant behavior by friends), which resulted in increased aggression. This interpretation builds on previous findings showing that interactions with aggressive peers in kindergarten led to increased aggressive behaviors over time (Snyder et al., 1997). Specifically, this study was the first, to our knowledge, to document the contribution of friends’ aggression to children’s aggression while taking into account the role of genetic liability to aggression. Most important, the present study indicates that this negative impact is most marked in children genetically at high risk for aggressive behaviors.

This differential susceptibility to aggressive peer influence could be accounted for by genetically mediated endogenous predispositions, such as those governing emotion regulation and social-cognitive processes, and perhaps shared with other comorbid conduct problems such as attention-deficit/hyperactivity disorder problems (Boivin et al., 2005; Vitaro et al., in press). Future studies should investigate the role of these possible genetically mediated predispositions, not only in terms of vulnerability but also with respect to resilience because children with low genetic risk for aggressive behaviors were less affected by friend’s aggression.

It has been reported that aggressive children may actively select and befriend aggressive peers with whom they share similar attitudes and interests with respect to aggression-related activities, such as rough play and dominance assertion (Boivin et al., 2005). In theory, this proactive affiliation process could be accounted for by the same genetic vulnerability underlying both the children’s aggressive tendencies and their selection of aggressive peers (rGE). The present study indicates that this was not true: having aggressive friends was essentially accounted for by environmental factors. The negligible heritability of friends’ aggression was consistent with that of a previous study on an age-heterogeneous sample of children (Bullock et al., 2006). However, age could be an important factor with respect to the selection of aggressive friends. Indeed, because children more actively select their social environment with age, we can speculate that active rGE will be more likely. Specifically, aggressive children may be more likely to select aggressive friends and do so on the basis of their genetic vulnerability (Scarr and McCartney, 1983; see Rose, 2002; Rushton and Bons, 2005 for rGE in friends selection). However that may be, we should bear in mind that G×E and rGE processes are not mutually exclusive but rather complementary pieces of the same developmental phenomenon. To the extent that children genetically at risk for aggression are more influenced by other aggressive children (G×E), they may progressively be more likely to actively select other aggressive children (rGE) with whom they could more likely to experience deviancy training (Dishion et al., 1994). Future longitudinal and genetically informative studies are necessary to more precisely examine the complex developmental interplay of genetic factors and peer experiences leading to the selection of deviant and more aggressive friends.

Other than the mean differences in own and friends’ aggression, boys and girls showed the same gene–environment etiological pattern. Thus, despite the differences in exposure, genetic liability similarly moderated the contribution of such exposure to aggression in both sexes. However, this finding must be interpreted cautiously because our sample had a limited power to detect sex differences. Furthermore, the G×E concerned physical aggression, which is typical of boys, whereas girls manifest more social or relational forms of aggression (Crick and Zahn-Waxler, 2003). These forms of aggression are not mutually exclusive and may share genetic origins (Brendgen et al., 2005). However, it will be important to document gene–environment transactions for multiple forms of aggression to more fully understand the development of aggressive behavior from a peer ecological perspective.

Limitations

The main limitation of this study was that it was cross-sectional and correlation based, which limits the inferences about social and developmental processes. For instance, other peer contextual influences that covary with friends’ aggression, such as peer rejection, could partly account for the results. Another constraint of this study was the limited sample size, which prevented us from drawing firm conclusions about the gene–environment etiology of friends’ aggression (see Posthuma and Boomsma, 2000), as well as about sex-specific G×E, as discussed previously. The effect size found for GxE was robust but modest. As Figure 1 illustrates, the GxE was mainly accounted for by the
twins at the highest genetic risk (i.e., MZ twins who are limited in number).

Seventy percent of the twins of the same family did not share classrooms. On the one hand, this is an asset of the study as it provided more independent assessments than when the twins were in the same classrooms. On the other hand, the fact that 30% of the twins shared the same classroom may have biased the evaluations and induced contrast effects among DZ twins. The low DZ ICC for aggression (vs. MZ twins) indeed suggests a modest contrast effect whereby the raters (teacher and peers) tend to oppose the two twins in their ratings. Such a bias may have artificially increased the heritability estimates for aggression. However, the limitation of some twins sharing the same classroom was mitigated by the minimal overlap in sharing friends and the fact that aggression was unrelated to the condition of sharing or not sharing the same classroom. Separation has been associated with differential adjustment to the school (e.g., internalizing problems, see Tully et al., 2004; van Leeuwen et al., 2005), but this question is beyond the scope of this report.

The findings of the present study may also be limited to kindergarten children. As mentioned previously, children may become more selective of their friends with age (Boivin and Vitaro, 1995; Dishion et al., 1994), which could bring about changes in the genetic–environment landscape of the association between physical aggression and friends’ aggression.

Implications

The implications of the present study are manifold. First, teacher and peer assessments can be combined to reliably assess individual differences in aggressive behavior at school entry. Given the well-documented predictive validity of these forms of assessments for later negative outcomes (Broidy et al., 2003; Moffitt, 1993; Nagin and Tremblay, 1999; Tremblay, 2003), their use is highly advisable for screening children for early prevention purposes. Second, this study emphasizes the complex interplay of genetic and environmental factors, rather than a deterministic genetic model, in the development of aggression. To understand individual differences in aggressive behaviors, we need to appreciate how children’s genetic vulnerability leads to aggression, how their cumulative exposure to environmental risks, including their association with aggressive friends, leads to increased aggression, and why children at genetic risk for aggression are more likely than other children to be affected by aggressive friends.

Finally, the findings underscore the importance of preventing aggressive behavior by intervening in situations in which peers interact. Reducing the likelihood of encountering friends who aggress could reduce aggression, especially among those who are at high genetic risk. This may be of particular importance during early childhood when active selection of friends may be less established. School-based interventions have shown to not only reduce aggressive behavior but also to prevent the further ostracism of aggressive children and to facilitate affiliation with nonaggressive, normatively developing children (van Lier et al., 2005b; Vitaro and Tremblay, 1994). Our results could mean that children at high genetic risk for aggression may benefit most from such preventive programs.

Disclosure: The authors have no financial relationships to disclose.

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Superhero-Related Injuries in Paediatrics: A Case Series  Patrick Davies, Julia Surridge, Laura Hole, Lisa Munro-Davies

Five cases of serious injuries to children wearing superhero costumes, involving extreme risk-taking behaviour, are presented here. Although children have always displayed behaviour seemingly unwise to the adult eye, the advent of superhero role models can give unrealistic expectations to the child, which may lead to serious injury. The children we saw have all had to contemplate on their way to hospital that they do not in fact possess superpowers. The inbuilt injury protection which some costumes possess is also discussed.


GENETIC RISK × FRIENDS’ AGGRESSION