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Does Other People's Plight Matter? A Genetically Informed Twin Study of the Role of Social Context in

the Link Between Peer Victimization and Children's Aggression and Depression Symptoms

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Abstract

Using a genetically informed design, this study examined the additive and interactive effects of genetic risk, personal peer victimization experiences and peer victimization experienced by others on children's aggression and depression symptoms. Of major interest was whether these effects varied depending on whether the victimized others were children's close friends or not. The sample comprised 197 monozygotic and same-sex dizygotic twin pairs reared together (95 female pairs) assessed in grade 4. Each twin's victimization experiences and victimization experienced by his or her friends and other classmates was measured using each individual's report about their own level of peer victimization. Aggression was assessed using peer nominations and depression was measured using self-reports. Indicative of a possible social learning mechanism or the emotional contagion of anger, multi-level regressions showed that personal victimization experiences were related to especially high levels of aggression when close friends where also highly victimized, albeit only in boys. Moreover, in line with social comparison theory, the effect of frequent personal victimization experiences on depressive feelings was much weaker when close friends were also highly victimized than when close friends were not or only rarely victimized. Finally, a high level of peer victimization experienced by other classmates was related to a lower level of aggression in girls and boys, possibly due to a heightened sense of threat in classrooms where many suffer attacks from bullies. All of these results were independent of children's genetic risk for aggression or depression. Theoretical and practical implications are discussed.

Keywords: Peer Victimization, Social Context, Aggression, Depression, Twin Design

Does Other People's Plight Matter? A Genetically Informed Twin Study of the Role of Social Context in the Link Between Peer Victimization and Children's Aggression and Depression Symptoms

Peer victimization is a severe problem with serious repercussions for the victims. Victims of peer abuse develop externalizing problems, most notably aggressive behavior (e.g., Hanish & Guerra, 2002; 2007), as well as internalizing problems such as anxiety and depression symptoms (e.g., Hodges, Boivin, Vitaro, & Bukowski, 1999; Vuijk, van Lier, Crijnen, & Huizink, 2007). In line with a diathesis-stress mechanism of psychopathology, recent evidence also suggests that peer victimization may be especially likely to foster aggression and depression symptoms in children with a pre-existing genetic vulnerability for such problems. Thus, a quantitative genetic study of 6 year-old twins showed that peer victimization was strongly related to aggressive behavior when genetic risk for aggression was high, but this relation was considerably weaker when genetic risk for aggression was low (Brendgen et al., 2008). Moreover, findings from two molecular genetic studies show that the deleterious effect of peer victimization is especially pronounced in youth carrying two 5-HTTLPR short alleles, which increases their vulnerability to developing depression symptoms (Benjet, Thompson, & Gotlib, 2010; Brendgen et al., 2008; Sugden et al., 2010).

Studies examining the link between peer abuse and children's developmental maladjustment have usually focused on children's own, personal experiences of victimization by peers. However, peer victimization is a social phenomenon that goes beyond the bully-victim dyad. When peer victimization occurs in a school class, most students are not only aware of it but are also present when it occurs (O'Connell, Pepler, & Craig, 1999; Salmivalli, 2010). Witnessing such acts of aggression may compromise children's behavioral and emotional adjustment even if they are not bullied themselves, and – as for personal peer victimization experiences – this association may be especially pronounced in children with a genetic predisposition for aggression and depression symptoms. Using a quantitative genetic design based on monozygotic and dizygotic twin pairs, the present study examined the additive and interactive effects of genetic vulnerability, children's own peer victimization experiences and victimization experienced by

others on children's aggressive behavior and depression symptoms. We also examined whether these effects vary depending whether the victimized others are children's close friends or not.

Personal Victimization Experiences, Victimization Experienced by Others, and Developmental Adjustment

Peer victimization usually occurs either on the playground or in the classroom, i.e., in plain view of others (Fekkes, Pijpers, & Verloove-Vanhorick, 2005). Examining coded playground observations of elementary school aged children, Craig and Pepler (1997) found that peers were present in 85% of bullying episodes. Studies also show that most students do nothing to intervene or support the victim (Goossens, Olthof, & Dekker, 2006; Salmivalli, Lagerspetz, Bjoerkqvist, Österman, & Kaukiainen, 1996) and as many as 20–30% of students even encourage the bully (O'Connell, et al., 1999; Salmivalli, 2001). Adults often seem to offer little help as well, as most are either not aware that peer victimization occurs or are unsuccessful in stopping it (Fekkes, et al., 2005). It is therefore not surprising that children who are victimized by their peers are not only at risk of becoming increasingly aggressive themselves but also of developing internalizing symptoms such as feelings of depression. However, peer victimization may also pose a serious threat to children's mental health even if they are not directly targeted themselves. Indeed, witnessing acts of violence in their community has been shown to promote aggression in youth by fostering anger and biased social cognitions such as hostile attributions as well as efficacy beliefs and positive outcome expectancies for aggression (e.g., Calvete & Orue, 2011; Musher-Eizenman et al., 2004; Reid-Quiñones et al., 2011; Schwartz & Proctor, 2000). Moreover, witnessing violence has been shown to trigger fear for their own and others' safety in children and adolescents (Reid-Quiñones, et al., 2011), leading to increased internalizing problems such as anxiety and depression symptoms (Ho & Cheung, 2010; Mrug & Windle, 2010). Only a handful of studies have examined specifically the effects of witnessing peer victimization in school (Flannery, Wester, & Singer, 2004; Ho & Cheung, 2010; Janosz et al., 2008; Rivers, Poteat, Noret, & Ashurst, 2009). Nevertheless, their findings concord with the notion that victimization that happens to others is related to increased levels of externalizing and internalizing

problems in children and adolescents, even when controlling for the effects of own victimization experiences. In all of these studies, however, assessment of both own and others' victimization by peers was entirely based on youth's self reports. Even more convincing evidence for the potential effect of peer victimization suffered by others on children's mental health would be provided if the measurement of others' victimization were based on these individuals' own accounts.

Interactive Effects Between Personal Victimization Experiences and Victimization Experienced by Others

In addition to potential additive effects, peer victimization suffered by others might interact with the effects of children's own victimization experiences on externalizing and internalizing problems. For example, through social learning mechanisms such as modeling or reinforcement, victimized children may be especially likely to perceive aggression as a normative way to obtain objects or gain power (and hence become more aggressive themselves) if many others in their class are also treated in the same way. Indirect support for this notion comes from findings showing that children in classrooms with a high mean frequency of aggression are more likely to remain or to become even more aggressive themselves than children in less aggressive classrooms (Mercer, McMillen, & DeRosier, 2009; Thomas et al., 2006). Moreover, this effect seems to be especially pronounced in children who – like many victimized youth – are more prone to displaying aggressive behavior than others to begin with (Kellam, Ling, Merisca, Brown, & Ialongo, 1998). It is also possible that feelings of anger, hostility, as well as fear and anxiety are augmented in a social environment where many fall victim to aggressive acts, thus increasing the risk of eliciting externalizing or internalizing problems in the victims. Indeed, there is considerable evidence for the social contagion of primal emotions such as anger, fear, or sadness between individuals through affective, motor, and somatosensory pathways (Bastiaansen, Thioux, & Keysers, 2009; Hatfield & Rapson, 2000). It has been suggested that this emotional contagion may be especially strong in individuals who – like victims of aggression – are sensitive to such emotions (Hatfield & Rapson, 2000).

With respect to internalizing problems, however, it is also conceivable that the effect of others' and own victimization interact in such a way as to *decrease* instead of increase such problems. Indeed, a large body of research shows that individuals experiencing stressful events such as victimization seek to regulate their emotional reaction by comparing themselves to others with similar experiences (for a review, see Taylor, Buunk, & Aspinwall, 1990). If many others have experienced similar (or even worse) hardships, this 'shared misery' can help mitigate negative emotional reactions such as anxiety and depression through a downward social comparison. Moreover, observing other victims' potentially successful response strategies may also help alleviate internalizing problems by providing valuable information for effective coping as well as hope and motivation. In contrast, if no one else in the social environment is faced with the same stressor, the absence of appropriate targets for social comparison or positive social learning models may lead to increased emotional distress.

The interactive effect of own and others' peer victimization experiences on mental health has, to our knowledge, only been examined directly in two studies so far. In one study (Rivers, et al., 2009), based on adolescents' reports of experienced and witnessed peer victimization over the past nine weeks and using self-reported psychiatric symptoms as outcome, no significant interaction was found. However, the lack of significant interaction could at least in part be due to the correlation of experiencing and witnessing victimization, both of which were based on youths' self-reports. Indeed, a significant interaction between own and others' victimization experiences was observed in the second study (Huitsing, Veenstraa, Sainio, & Salmivalli, in press). In line with social comparison theory, it was found that victimized three-to-fifth graders had a higher self-esteem when they were in classrooms with high average levels of victimization. Further, albeit indirect evidence for an interaction between own and others' victimization experiences comes from a study by Graham and Juvonen (2002). These authors found that, compared to Latino and multiethnic youth, fewer African American adolescents were nominated as victims of peer harassment. However, the few African American youth who were victimized reported more loneliness and lower self-esteem than harassment victims in the other ethnic groups. In line with social comparison theory, the authors interpret these findings as evidence that peer victimization is especially detrimental for youngsters' emotional well-being when this experience deviates from what others in their peer group experience. Similar findings were obtained in another study (Bellmore, Witkow, Graham, & Juvonen, 2004), which showed that peer victimization was related to less anxiety in sixth-graders when their classrooms were characterized by a high level of social disorder (measured by the average of classmates' teacher-rated disruptiveness, aggression, and victimization). Still, given the scarcity and inconsistency of findings, more research on the putative interactive effects of own and others' peer victimization experiences on youngsters' internalizing and externalizing problems is needed.

In Search of Potential Moderators: Considering Victims' Characteristics

When examining the additive and interactive effects youngsters' own and others' peer victimization experiences on youngsters' mental health, one potentially important issue to consider may be the victimized youngsters' own characteristics as well as those of the victimized 'others'. With respect to victimized youngsters' own characteristics, there is considerable evidence that both aggression and depression in children are to a significant extent influenced by genetics (Dick, 2007; DiLalla, 2002; Rice, 2009). As already mentioned, recent findings from behavioral and molecular genetic studies show that the negative effect of being the target of peer victimization on aggression and depression symptoms is especially pronounced in youth with a genetic predisposition for such problems (Benjet, et al., 2010; Brendgen, et al., 2008; Sugden, et al., 2010). A similar diathesis-stress mechanism of gene-environment interaction might be observed in regard to the (additive or interactive) effects of peer victimization experienced by others on children's aggression and depression symptoms. Thus, children with a preexisting genetic disposition for aggressive behavior may be especially susceptible to the previously discussed social learning or emotional contagion processes that may link victimization experienced by others to increased aggression. Similarly, children who are at genetic risk for depression may be especially prone to the potential contagion of fear and anxiety from victimized others, as well as to the potential effects of social comparison on depression symptoms.

With respect to the victimized others' characteristics, a crucial point may be the degree of children's affiliative closeness with the victimized others. Indeed, it has been suggested that behavioral modeling, emotional contagion, as well as social comparison processes are stronger when they involve social agents that individuals feel close to (Bandura & Huston, 1961; Hatfield & Rapson, 2000; Taylor, et al., 1990). Indeed, children spend more time with friends than with nonfriends and are more likely to emulate the social behavior of their close friends than the behavior of others (Hartup, 1996). Furthermore, children are more likely to confess personal problems to friends than to nonfriends (Berndt, 2002), which can foster the emotional contagion of negative feelings (Rose, Carlson, & Waller, 2007). In addition, friends often share many behavioral and other characteristics (Berndt, 1982), which may facilitate social comparison processes. As such, victimization experienced by close friends should have a much greater impact – either additively or interactively with children's own victimization experiences or with genetic risk – on children's externalizing and internalizing problems than victimization experienced by others with whom children are presumably less close. To date, however, none of the few existing studies have considered a possible moderating role of genetic vulnerability or have distinguished between victimized friends and victimized nonfriends when examining the potential effect of victimization experienced by others on children's mental health.

The Present Study

Addressing the limitations of the extant literature, the principle objective of the present study was to examine the additive and interactive effects of own peer victimization experiences and victimization experienced by others on children's aggressive behavior and depression symptoms. Of major interest in this context was whether these effects varied depending on (a) whether the victimized others were children's close friends or not, or (b) children's genetic risk for aggression or depression. To avoid the risk of inflated estimates due to shared source variance in previous studies, children's own victimization experiences and victimization experienced by their friends and nonfriends was assessed using each individual's report about his or her personal level of victimization by peers. In line with social learning principles and emotional contagion theory, we expected that children especially those who are themselves highly victimized - would be more likely to show elevated levels of aggression if many of their friends (and, to a lesser extent, many of their nonfriends) are also treated in the same way. Moreover, according to emotional contagion theory, children should also show elevated levels of depression symptoms under these circumstances. According to social comparison theory, however, highly victimized children should show *fewer* internalizing problems if many others – and particularly their friends – also fall prey to peer victimization. In contrast, victimized children's internalizing problems should be especially elevated when only few other children share the same fate. These additive and interactive effects of own peer victimization experiences and victimization experienced by others on children's aggression and depression were expected to be especially pronounced in children with a high genetic risk for these mental health problems. Potential moderating effects of child sex in these associations were also tested.

The study objectives were addressed using a quantitative genetic design based on data from monozygotic (MZ) and dizygotic (DZ) twin pairs reared together. Despite significant advances in knowledge, many of the specific genes at play in the etiology of aggression and depression symptoms are still largely unknown. Twin designs thus provide an ideal framework to study the interplay between environmental and genetic influences on development (see description of analytical procedure below). Importantly, empirical evidence suggests that the nature of twins' peer relations (e.g., the number of friends and friendship features) does not differ from that of non-twin children (Koch, 1966; Thorpe, 2003). Moreover, twin samples and singleton samples do not differ with respect to social-psychological adjustment, including aggression and depression symptoms, during childhood (Moilanen, 1999).

Method

Sample

The 197 twin pairs (MZ males = 60, MZ females = 51, DZ males = 42, DZ females = 44) participating in this study were part of a population-based sample of 448 MZ and same-sex DZ twin pairs

from the greater Montreal area who were recruited at birth between November 1995 and July 1998. Zygosity was assessed at 18 months based on physical resemblance via 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 8-10 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-seven percent of the families were of European descent, 3% were of African descent, 3% were of Asian descent, and 1% were Native North Americans. The remaining families did not provide ethnicity information. The demographic characteristics of the twin families were comparable to those of a sample of single births representative of the urban centers in the province of Quebec. At the time of their child(ren)'s birth, 95% of parents lived together; 44% of the twins were the first born children; 66% of mothers and 60% of fathers were between 25 and 34 years old; 17% of mothers and 14% of fathers had not finished high school; 28% of mothers and 27% of fathers held a university degree; 83% of the parents held an employment; 10% of the families received social welfare or unemployment insurance; 30% of the families had an annual income of less than \$30,000.

The sample was followed longitudinally at 5, 18, 30, 48, and 60 months focusing on a variety of child-related and family-related characteristics. New data collections were completed when the children were in kindergarten, grade one, and grades three and four. The present paper describes findings from the grade four data collection (mean age = 10.04 years, SD = .26). Attrition in the sample was approximately 6% per year, such that 307 twin pairs participated in grade four. In the majority of twin pairs (i.e., 197 or 64.2%), the two twins did not attend the same classroom. For statistical analyses with twin samples, the same environmental variable (e.g., classmates' peer victimization) needs to be measured consistently either at the level of the individual twin, as was the case when the two twins of a pair were in different classrooms, or at the level of the pair, as was the case when the two twins of a pair were in the same classroom. Because in most cases the two twins of a pair were in different classrooms, only these pairs

were included in the present study. Notably, no twin child went to the same class as any other twin child in our study sample. The twin pairs in the final study sample did not differ from those who were lost through attrition in regard to family status, parental education or parents' age, although family revenue was higher in the remaining study sample. Moreover, a comparison in regard to mother-rated aggressive and anxious behavior assessed in early childhood (ages 18 to 48 months) revealed no significant differences between those who were included in the present study and those who were excluded.

Measures

Aggressive behavior of each target child (i.e., the twins) was measured using peer nominations from their classmates. The participation rate per class - based on active parental consent for each child in a given class - varied between 73% and 80% in the different classrooms. A roster with the names of all children in a given class who had received parental consent to participate was handed out to all participating children in the classroom. The children were then asked to nominate up to three classmates who best fit a specific behavioral descriptor. Six behavioral descriptors were used based on similar items from the Social Behavior Questionaire (Tremblay et al., 1991) (e.g., "...those who most often push and hit other children; ...those who most often say mean things to other children; ...those who fight most often with other children"). For each behavioral descriptor, the total number of received nominations was calculated for each child in the class and *z*-standardized within classroom to account for differences in classroom size. The *z*-standardized individual item scores were then averaged for each child and again z-standardized within classroom (Cronbach's alpha = .90, M = .12, SD = 1.08).

Depression symptoms of each target child (i.e., the twins) were measured using 10 self-reported items from an abbreviated version of the Children's Depression Inventory (Kovacs, 1992). Responses on the CDI are given on a three-point Guttman-type scale ranging from 0 to 2. Item scores were summed to yield a self-perceived depression symptoms score for each target child (Cronbach's alpha = .74, M = 3.94, SD = 3.52).

Friendship nominations. During the peer nomination procedure, children were asked to nominate up to three best friends in the classroom, regardless of whether the friend participated or not. Limiting friendship nominations to the classroom does not seem to overly restrict selection of friends because the vast majority of elementary school children select a best friend from among their classmates even when they can nominate a friend from outside the classroom (Parker & Asher, 1993). Moreover, classroom composition remained stable throughout the year and students spent all day together. A participant was considered to have a reciprocal friend when the peer the participant had nominated had in turn rated the participant as one of his/her three best friends. Of the participating twins, 85% had at least one reciprocal close friend in the class (M = 1.64, SD = .99, range = 0 - 3). This percentage was similar to that reported in research with singletons (Parker & Asher, 1993). Among those with at least one reciprocal friend, the mean number of reciprocal friends was M = 1.93, SD = .78.

Peer victimization. All participants (i.e., the twins as well as their classmates) provided information about their personal experiences of victimization by peers by answering eight items inspired by the Social Experiences Questionnaire (Crick & Grotpeter, 1996) (e.g., "During this school year, how many times has a child at your school... called you names or said mean things to you?,... said mean things about you to other children?, stopped you from playing with his or her group when you wanted to play?, pushed, hit or kicked you?"). Responses were given on a three-point scale ranging from 0 (never) to 2 (often). Item scores were summed to yield a self-perceived peer victimization score for each participating child (Cronbach's alpha = .80). Next, three victimization variables were calculated for each target child (i.e., each twin): a) The target child's *Personal Victimization*, which was equivalent to their self-reported peer victimization score, M = 5.79, SD = 3.54, b) the target child's *Friends' Victimization*, which was an average of the self-perceived victimization scores of the target child's nonfriend *Classmates' Victimization*, which was an average of the self-perceived victimization scores of the target child's nonfriends, M = 5.14, SD = 2.17, and c) the target child's nonfriend *Classmates' Victimization*, which was an average of the self-perceived victimization scores of the target child's classmates, excluding his or her nominated friends, M = 4.93, SD = 1.22. Of note, because no twin child went to the same class as any other twin child in our study

sample, the level of friends' peer victimization as well as the level of classmates' peer victimization were variables that were unique for each twin child.

Results

Calculation of Genetic Risk for Aggression and Depression

The twin design makes it possible to assess the relative role of genetic factors and environmental factors associated with a given phenotype (Falconer, 1989). The relative strength of additive genetic factors on individual differences is approximately twice the MZ and same-sex DZ correlation difference. The relative strength of shared environmental factors that affect twins within a pair in a similar way can be estimated by subtracting the MZ correlation from twice the DZ correlation. Nonshared environmental factors that uniquely affect each twin in a pair are approximated by the extent to which the MZ correlation is less than 1. The MZ intra-pair correlations of both aggression (r = .62) and depression (r = .37) were considerably larger than the corresponding DZ intra-pair correlations (r = .42 for aggression and r = .09 for depression). This pattern suggests a substantial contribution of genetic factors in explaining the two phenotypes, whereas shared environmental influences seem to play only a small role. The overall magnitude of the MZ correlation was still well below 1.0 for both phenotypes, however, indicating a significant contribution of nonshared environmental factors as well.

Next, each target child's genetic risk for depression and aggression, respectively, was calculated as a function of his or her co-twin's level of that phenotype and the pair's zygosity (Andrieu & Goldstein, 1998). This method has been used in several studies that examined gene-environment interactions with an epidemiological twin design (Brendgen, et al., 2008; Jaffee et al., 2005; Wichers et al., 2009). Specifically, one twin from each twin pair was selected as the "target twin" and the second twin as the "co-twin." Each twin pair was represented in the data set twice, first with the elder twin as the target and the younger twin as the co-twin, and second with the younger twin as the target and the elder twin as the co-twin. Separate continuous scores of genetic risk for depression and for aggression were computed as a function of (a) zygosity and (b) the presence or absence of depression (or aggression, respectively) in the co-twin. To this end, we first dichotomized the Depression scale using the 70th percentile of the overall sample distribution as the cut-off, which is similar to the cut-off used in previous studies to distinguish between children with depressive behavior symptoms from others (Brendgen, Vitaro, Turgeon, & Poulin, 2002; Rudolph & Clark, 2001). This cut-off was chosen because it captured children with relatively elevated levels of depression symptoms in our sample distribution while at the same time ensuring sufficient sample size for subsequent analyses. The 70th percentile cut-off for the depression scale in the present sample corresponded to experiencing at least five of the ten assessed depression symptoms at least sometimes. Children whose depression score was at or above the 70th percentile value of the sample distribution were considered depressed, whereas children whose depression score was below the 70th percentile value of the sample distribution were considered not depressed. In order to create comparable groups for depression and aggression, the same cut-off was used to dichotomize the aggression scale.

The presence or absence of depression in the co-twin was then combined with information on the pair's zygosity into an ordinal index of genetic risk for depression. Thus, the target twin's genetic risk for depression was considered to be highest when he/she was part of an MZ pair, who share 100% of their genes, and when the co-twin was depressed (19% of the sample). The target twin's genetic risk for depression was somewhat lower when he/she was part of a DZ pair, who share on average only 50% of their genes, and when the co-twin was depressed (16%). The target twin's genetic risk for depression was even lower when he/she was part of a DZ pair and when the co-twin was not depressed (27%). The target twin's genetic risk for depression was lowest when he/she was part of an MZ pair and when the co-twin was not depressed (38%). Using the analogous rationale, an ordinal index of genetic risk for aggression was calculated, with 20% of target children being at highest genetic risk, 13% at high genetic risk, 31% at low genetic risk, and 36% at lowest genetic risk.

For the logic of the genetic risk indexes, it was important to ensure that zygosity differences in aggression or depression could not account for any effect of genetic risk on these outcomes. A test of equality of means in a saturated model for a continuous phenotype revealed that MZ and DZ twins did

not differ in regard to aggression or depression (p = .262 for aggression and p = .464 for depression), based on a test of equality of means in a saturated model. It was also important to ensure that zygosity differences in regard to their own victimization, their friends' victimization or their nonfriend classmates' victimization could not account for any difference among the genetic risk groups in regard to aggression or depression. MZ and DZ twins did not differ with respect to the mean level of their own victimization (p = .577), their friends' level of victimization (p = .553) or the level of victimization experienced by their nonfriend classmates (p = .153).

Bivariate Correlations Between the Study Variables

Correlations between the study variables, derived from a multivariate within twin-pair correlation matrix, are presented in Table 1. As can be seen, boys were more aggressive and had a higher genetic risk for aggression than girls. Boys also reported higher levels of personal peer victimization than girls. Aggression and depression symptoms were significantly, albeit weakly, correlated and the two phenotypes were significantly correlated with their respective genetic risk indexes. A high genetic risk for aggression was significantly related with an elevated level of personal peer victimization experiences, but not with the level of victimization experienced by friends or other classmates. A high genetic risk for depression symptoms was also significantly related with an elevated level of personal peer victimization experiences and with an elevated level of classmates' (but not friends') victimization. These correlations are noteworthy, as they suggest the presence of a gene-environment correlation, whereby specific heritable personal characteristics increase an individual's probability of experiencing specific environmental conditions (Rutter, Moffitt, & Caspi, 2006; Shanahan & Hofer, 2005). Finally, children's personal peer victimization experiences were positively correlated with their friends' level of peer victimization, which, in turn, was positively correlated with the general level of peer victimization experienced by the other classmates.

Multilevel Regression Analyses Predicting Aggression

Using multi-level regression analyses with the Mplus Version 6 software package (Muthén & Muthén, 1998-2010), we next examined the additive and interactive effects of child sex, genetic risk, and personal, friends', and classmates' peer victimization experiences on children's aggression. In a two-level model, a hierarchy consists of lower-level observations (i.e., level 1 unit of analysis) nested within higher-level observations (i.e., level 2 unit of analysis). In the context of the present study, each individual child (level 1) is nested within a twin pair (level 2). Due to the genetic structure of the data, both the within-pair (i.e., level 1) variance and the between-pair (i.e., level 2) variance may differ between MZ and DZ twins. Therefore, separate estimates for level 1 and level 2 variances in MZ twins and DZ twins, respectively, were included in the multilevel model. Level 1 predictors were included as fixed effects.

A series of consecutive models of increasing complexity were estimated where each subsequent model was compared to the preceding one to evaluate whether the inclusion of additional predictors provided a better fit to the data. Goodness of fit for each model was evaluated based on the –2log likelihood estimate and a likelihood ratio test was used to evaluate the difference in fit between subsequent models. For each model, the fixed effects of the predictor variables, the level 1 and level 2 variance parameters, the model fit (i.e., –2log likelihood), and the likelihood ratio are provided. To account for occasional missing data (5.7% of data points), all models were estimated using multiple imputations generated from a Markov Chain Monte Carlo simulation within Mplus Version 6 (for a detailed description, see Asparouhov & Muthén, 2010b). Specifically, for the purposes of the present study, ten independent imputed missing data sets were generated, which were then used for subsequent model tests using maximum likelihood estimation. Separately for each tested model, parameter estimates were averaged over the imputed data sets and the associated standard errors were computed using the formula developed by Rubin (1987). For nested model comparisons, adjusted Likelihood ratio difference tests were performed based on the procedures for imputed data sets described by Asparouhov and Muthén (2010a).

Table 2 presents the results from the first series of multilevel analyses predicting aggression. The first model included the level 1 (i.e., child-specific) predictors as main effects. As can be seen, children with greater genetic risk, $\beta = .50$, p < .001, and those who were frequently victimized themselves, $\beta = .20$, p < .001, were more aggressive. In contrast, a high level of peer victimization experienced by classmates was related to slightly reduced levels of aggression in the children, $\beta = -.09$, p < .05, whereas the level of their friends' peer victimization had no main effect on children's aggression. However, results from the second model revealed a significant two-way interaction of children's own victimization with their friends' victimization, $\beta = .10$, p < .05, but not with their other classmates' victimization. The third model showed that the interaction between children's own and their friends' victimization was significantly moderated by child sex, $\beta = -.20$, p < .05. Further probing revealed that children's personal experiences significantly interacted with their friends' victimization experiences in predicting aggression in boys, $\beta =$.18, p < .001, but not in girls, $\beta = -.02$, p = .73. A break-down of the significant two-way interaction (see Figure 1) revealed that personal victimization experiences were unrelated to boys' aggression when their friends experienced very low levels (1 SD below the mean) of peer victimization, $\beta = -.01$, p = .83. In contrast, frequent personal victimization experiences were related to especially high levels of aggression in boys whose friends where also highly victimized (1 SD above the mean), $\beta = .34$, p < .001. Interestingly, however, the figure also shows that boys who experienced an exceptionally low rate of personal peer victimization but whose friends were very frequently victimized showed the lowest level of aggression. For girls, only a main effect of personal victimization experiences on aggression emerged that was independent of their friends' victimization experiences, $\beta = .16$, p < .001.

In the next two models, we tested whether personal victimization interacted with genetic risk in predicting aggression (model 4) and whether this interaction varied for girls and boys (model 5). None of these interactions was significant. Similarly, there were no signification interactions of genetic risk with

either friends' or classmates' victimization experiences (model 4) nor was there any significant sex moderation of these interactions (model 5).

Notably, given that boys were more aggressive than girls in our sample, relatively more boys and relatively fewer girls were considered at high or highest genetic risk for aggression with the genetic risk variable calculated based on the overall 70th percentile cut-off than if it had been calculated using a gender-specific 70th percentile cut-off. The genetic risk factor for aggression based on the overall cut-off used in the preceding analyses thus reflects, at least partly, the gender difference in aggression. This, however, did not unduly bias the additive or interactive effects of the genetic risk variable reported above, because gender was also a predictor variable in the analyses. This was confirmed when we re-ran the analyses predicting to aggression with a newly calculated genetic risk factor based on the gender-specific 70th percentile cut-off. With only minor variations in the regression coefficients, these new analyses yielded the same pattern of significant and nonsignificant main effects and interaction effects as when genetic risk was based on the overall 70th percentile cut-off. The conclusions from these additional analyses are thus the same as those from the analyses reported above.

Multilevel Regression Analyses Predicting Depression Symptoms

Similar multi-level regression analyses were performed to predict depression symptoms (see Table 3). The results from the first model showed that children with greater genetic risk, $\beta = .42$, p < .001, and those who were frequently victimized themselves, $\beta = .25$, p < .001, reported more depression symptoms. Neither the level of their friends' nor the level of their classmates' peer victimization had a significant main effect on children's depression symptoms. Model 2, however, revealed a significant interaction of children's own victimization with their friends' victimization, $\beta = .11$, p < .05, but not with their other classmates' victimization. Model 3 showed that the interaction between children's own and their friends' level of peer victimization did not vary for girls and boys. Further probing of this significant two-way interaction (see Figure 2) showed that frequent personal victimization experiences were related to

especially high levels of depressive feelings in children whose friends were only rarely victimized (1 SD below the mean), $\beta = .39$, p < .001. In contrast, the effect of a high level of personal victimization experiences on depressive feelings was much weaker, albeit still significant, for children whose friends were also highly victimized (1 SD above the mean), $\beta = .17$, p < .05. Subsequent model tests showed that neither children's personal peer victimization experiences, nor the level of peer victimization experienced by their friends or their classmates significantly interacted with children's genetic risk in predicting depression symptoms.

Discussion

The objectives of the present study were a) to examine the additive and interactive effects of own peer victimization experiences and victimization experienced by their friends and by their classmates on children's aggressive behavior and depression symptoms, b) to test whether these effects varied depending on children's genetic risk for such mental health problems, and c) to investigate potential moderating effects of child sex in this context.

Personal Peer Victimization, Others' Peer Victimization, and Child Aggression

In line with previous studies (e.g., Hanish & Guerra, 2002; Lamarche, et al., 2007), girls and boys who were frequently victimized by their peers showed elevated levels of aggressive behavior. At least in boys, the link between personal peer victimization and aggressive behavior was augmented even further when their friends were also highly victimized. Social learning mechanisms such as modeling or reinforcement of aggression might play a role in explaining this interaction effect. Specifically, victimized boys may be especially likely to consider aggression as a justified response if they frequently see their victimized friends use aggressive strategies when harassed by peers. In support of this notion, peer victimization has been linked with a stronger increase in first grade boys' (but not girls') reactive aggression when they had moderately to highly reactively aggressive friends (Lamarche, et al., 2007). Many of these friends were likely also victimized, given that reactive aggression is a significant risk factor

of peer victimization (Schwartz et al., 1998). This interpretation of the present findings necessarily remains speculative, however, because friends' aggressive responses to peer victimization were not assessed in the present study.

An additional explanation for our finding of an increased level of aggression in victimized children whose friends are also frequently harassed may be the contagion of negative emotions, particularly anger and hostility, among victimized children and their friends (Bastiaansen, et al., 2009; Hatfield & Rapson, 2000). Indeed, research has shown that witnessing bullying is uniquely associated with feelings of hostility even when own victimization experiences are controlled (Rivers, et al., 2009) and these feelings may be aggravated when the targets of bullying are close friends. Some albeit indirect support for this notion comes from recent findings that children reported increased levels of anger when they thought of their friends being victimized by others (Rocke Henderson & Hymel, 2011). Anger over their friends' plight may augment victimized boys' already pent up negative emotions even further. Given that retaliation is a key feature for understanding aggressive behavior in victimized children (Camodeca, Goossens, Meerum Terwogt, & Schuengel, 2002; Camodeca, Goossens, Schuengel, & Terwogt, 2003; Salmivalli & Nieminen, 2002), the accumulation of these negative emotions may eventually trigger aggressive outbreaks in an attempt to revenge against their tormenters. The fact that, compared to girls, boys more readily consider aggressive behavior as an acceptable response to peer provocation (Goldstein, Tisak, & Boxer, 2002) may explain why this interactive effect between friends' and own victimization was only found in boys.

As hypothesized, frequent peer victimization experienced by others only contributed to predicting elevated levels of victimized children's aggressive behavior insofar as others' victimization experiences concerned children's friends. These findings are in line with propositions from both social learning and emotional contagion theory that modeling and reinforcement of behaviors such as aggression, as well as the contagion of primal emotions such as anger, are stronger when they involve social agents that individuals feel close to (Bandura & Huston, 1961; Hatfield & Rapson, 2000; Taylor, et al., 1990).

Nevertheless, in addition to friends' peer victimization experiences, peer victimization experienced by other classmates was also significantly, albeit weakly, associated with children's aggressive behavior. Somewhat unexpectedly, however, whereas friends' frequent peer victimization experiences augmented victimized children's aggression, frequent victimization experienced by nonfriend classmates was related to *lower* levels of aggression in both victimized and nonvictimized children. A possible explanation for this finding may be that children show reduced aggression due to a heightened sense of threat when many of their classmates suffer attacks from bullies. Under these circumstances, children may want to avoid provoking potential bullies by being somewhat less dominant and aggressive – and hence less noticeable. Indeed, even if they are not victimized themselves, children and adolescents who witness violence report increased fear for their safety and tend to react by withdrawing and fleeing from rather than engaging in violent interactions (Reid-Quiñones, et al., 2011). Notably, a similar process may also explain why boys who are not victimized themselves but whose friends were very frequently victimized showed very low levels of aggression. These children, who likely often witness their close friends' frequent suffering firsthand, may be especially motivated to use social strategies other than aggression to negotiate conflict with peers in order to avoid becoming bullied themselves.

Personal Peer Victimization, Others' Peer Victimization, and Child Depression Symptoms

Neither friends' nor classmates' victimization were directly related to children's level of depression. These results seem to reflect those of other studies showing that witnessing violence may lead to desensitization (Buka, Stichick, Birdthistle, & Earls, 2001) and that children generally worry little about the suffering of schoolmates who are victims of peer harassment (Perry, Williard, & Perry, 1990; Rigby & Slee, 1991). Instead, similar to what was found for aggression, the extent of friends' peer victimization interacted with children's own victimization experiences in predicting depression. The nature of this interaction, however, was in the opposite direction to that found for aggression. Specifically, highly victimized children reported considerably *fewer* depression symptoms when their friends were also frequently victimized than when their friends were not or only rarely victimized by peers. These results

concord with findings from three previous studies that victims have fewer internalizing problems when their classrooms are characterized by a high level of peer victimization or social disorder (Bellmore, et al., 2004; Huitsing, et al., in press) or when they belong to ethnic groups where many others are also victimized (Graham & Juvonen, 2002). The findings are also in line with social comparison theory, which dictates that individuals experiencing stressful events seek to compare themselves to others (Taylor, et al., 1990). Downward social comparisons with others who have experienced similar (or even worse) hardships can help mitigate negative emotional reactions such as anxiety and depression. As suggested by Graham and Juvonen (2002), victimized children might feel less bad about themselves when they see others share their plight because they may be less likely to engage in self-blaming attributions. Under these circumstances, victims may perceive their experiences as more or less random events for which they are not responsible. In contrast, if few others are the target of peer harrassment, victims may be more likely to conclude that they are targeted because of specific personal characteristics, thus leading to increased emotional distress. The importance of self-blame for explaining the link between personal peer victimization experiences and subsequent depression has been shown in previous research (Graham & Juvonen, 1998). Finally, it is also possible that victimized children who have the opportunity to see others successfully respond to such attacks are more likely to develop effective coping skills and to ward off emotional distress.

Importantly, the present study showed that the alleviating effect of shared suffering against depression only seems to emerge when the fellow victims are close friends, but not when the fellow victims are other classmates the child is not befriended with. This result is in line with the notion that social comparison as well as social learning processes are stronger when they involve social agents that individuals feel close to (Bandura & Huston, 1961; Taylor, et al., 1990). Friends often share many behavioral and other characteristics (Berndt, 1982), which may facilitate social comparison. Moreover, because friends are more likely than nonfriends to confess personal problems to each other (Berndt, 2002),

victimized friends may also talk more with each other about their negative experiences with bullies and help each other find ways to overcome their predicament.

The Role of Children's Genetic Risk for Aggression and Depression Symptoms

Notably, all of the above mentioned findings were obtained while controlling for children's genetic risk for aggression and depression symptoms, respectively, which explained a considerable portion of inter-individual differences in these problems. Contrary to expectations, however, genetic risk for these problems did not moderate (i.e., augment) the associations of either personal peer victimization or of friends' or classmates' peer victimization with aggression or depression. Thus, the additive and interactive effects of children's own and others' peer victimization experiences on children's externalizing and internalizing problems seem to occur even in children without a strong genetic disposition for such problems. At least in part, this lack of Gene-Environment Interaction may be explained by the presence of a Gene-Environment Correlation. Indeed, children with a strong genetic disposition for either aggression or depression symptoms in our sample were more likely to be victimized by their peers. These results lend further credence to findings from studies with singleton samples showing that both pre-existing externalizing and internalizing problems put children at risk of becoming the target of peer harassment (e.g., Barker et al., 2008; Boivin, Hymel, & Hodges, 2001; Hanish & Guerra, 2000; Hodges, et al., 1999; Lamarche, et al., 2007; Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999). Although geneenvironment interaction (GxE) and gene-environment correlation (rGE) can and do co-occur (Eaves, Silberg, & Erkanli, 2003; Purcell, 2002), finding statistical support for GxE in the presence of rGE is difficult.

However, it is also entirely possible that – at least for the specific age group studied here – peer victimization is an important stressor that negatively affects the mental health of most children regardless of their genetic risk. Indeed, contrary to the elementary-school aged children of the present sample, the few studies that have found evidence of genetic moderation of the link between peer victimization and depression symptoms included early to mid-adolescents (Benjet, et al., 2010; Sugden,

et al., 2010). In contrast, evidence of genetic moderation of the link between peer victimization and aggression has so far been found in kindergarten girls only (Brendgen, et al., 2008). Some evidence suggests that the relative influence of genetic factors on depression increases from childhood to adolescence (Rice, 2009), whereas the opposite pattern is true for aggression (Rhee & Waldman, 2002). As such, it is possible that GxE processes linking own and others' peer victimization with depression symptoms may indeed be mainly observed in adolescence, whereas a similar GxE with respect to aggression may be found only in young children. More research is necessary to clarify potential developmental changes in gene-environment interplay linking children's own and others' peer victimization experiences with mental health outcomes.

Strengths, Limitations, and Conclusions

The present study is the first to examine the additive and interactive effects of own peer victimization experiences and victimization experienced by others on children's aggressive behavior and depression symptoms while testing whether these effects varied depending on (a) whether the victimized others were children's close friends or not, or (b) children's genetic risk for externalizing or internalizing mental health problems. In this context, a major strength of our study is the use of friends' and classmates' own reports of their victimization experiences instead of relying on target children's (in this case, the twins') accounts of their friends' and classmates' peer victimization. A further asset of the present study is the use of mostly different reporting sources for the different variables at play (i.e., peer nominations, self-reports) to reduce bias due to shared source variance.

In addition to these strengths, the present study also has several limitations that need to be considered when interpreting the findings. One limitation concerns the cross-sectional nature of the data due to budget constraints, which limits the interpretability of the direction of observed effects. However, the control for genetic risk in our regression analysis was akin to a control for pre-existing personal dispositions for these outcomes, similar to what the control for previous levels of the outcome in longitudinal studies is trying to achieve. Moreover, because both genetic risk and the purported environmental variables were included as predictors in the regressions, the correlation between these variables was statistically controlled. As previously mentioned, such gene-environment correlations reflect the child's effects on his or her environment insofar as heritable personal characteristics (such as a disposition for aggression or depression) may shape environmental experiences (such as a greater risk of being victimized by peers or of having friends who are victimized). By controlling for gene-environment correlations statistically in the regressions, the observed associations between the environmental risk factors (i.e., personal and others' peer victimization experiences) and child behavior (aggression and depression) likely reflect the effects of these environments on child behavior and not the reverse (i.e., child effects on the environment). Nevertheless, longitudinal studies are needed to disentangle directionality of effects even more conclusively. Such studies should preferably include short-term longitudinal data within the same school year, because children's social context (i.e., friends, classmates) may change considerably from one year to the next and may thus have immediate rather than long-term effects on children's behavioral and emotional adjustment.

Another limitation relates to the fact that we did not test the purported mechanisms underlying the observed associations, for instance by assessing friends' behavioral responses to victimization or by measuring victims' emotional or cognitive responses to their own and others' victimization. As such, while the present findings may be explained by social learning, emotional contagion, or social comparison processes, future studies are needed for a direct test of these hypothesized mediating pathways. A further limitation is the relatively small sample size, which was in part due to the fact that twin pairs where both members attended the same classroom had to be excluded from analyses to ensure consistency in classroom level data for the multi-level regression analyses. Although the final sample did not seem overly biased and statistical power was sufficient to detect significant interaction effects, future studies need to replicate the present findings with larger samples. It also needs to be kept in mind that the present results are based on a normative sample and may not generalize to children who are diagnosed with conduct disorder or major depression. As mentioned, the results may also differ for other age groups. Moreover,

findings may vary depending on the specific form of peer victimization children and their agemates experience. Indeed, a recent study showed that witnessing indirect forms of peer victimization such as rumor spreading or social exclusion was unrelated to adolescents' externalizing and internalizing problems (Fitzpatrick & Bussey, 2011). However, that study did not differentiate whether friends or nonfriends where the targets of peer victimization nor did it examine interactive effects with children's own victimization experiences. Future research will need to investigate these issues while considering potential differences between the various forms of peer victimization.

Despite these limitations, the present study offers important new insights into the role of social context in the link between peer victimization and child adjustment. Our findings suggest that, while children with a genetic disposition for either externalizing or internalizing problems are at higher risk than others of being victimized by their peers, these experiences may in turn promote further mental health problems. Whether and to what extent these negative effects occur, however, seems to depend on whether others – particularly close friends – also fall prey to peer attacks or not. Aggressive behavior seems to increase even more in victims whose friends are also frequently harassed. Paradoxically, however, the same negative social context seems to protect victims against depressive feelings. The results from our study also have important implications for anti-bullying interventions. On a general level, our findings lend support to current perspectives on bullying intervention that favor the consideration of the larger peer context instead of a sole focus on victims and bullies, because negative behaviors may be modeled or reinforced by others in the group (Rivers, Duncan, & Besag, 2007; Salmivalli, 2010). Our findings also suggest that the evaluation of anti-bullying interventions should take into account how interventions influence the position of victims in the classroom. As such, it may be useful for parents and practitioners to know whether a victimized child who presents with emotional or behavioral difficulties is the only victim or one of several in the group. As noted by Huitsing and colleagues (in press), if an anti-bullying intervention reduces the number of victims in a classroom to only one, the remaining victim might be worse off because there may be no one left with whom to share the plight. Importantly, these latter

findings should not be interpreted as a justification for disregarding the danger of peer harassment for the victims. Rather, they suggest that victimized children need social support from and should be encouraged to share their troubles with others who have similar experiences. At the same time, clinicians, educators, and parents need to help victimized children find nonaggressive ways to cope with their negative experiences in order to prevent a further escalation of the cycle of violence.

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Table 1

Bivariate Correlations of Study Variables

	1	2	3	4	5	6	7	8
1. Child Sex								
2. Genetic Risk for Aggression	31***							
3. Genetic Risk for Depression	04	.08						
4. Aggression	39***	.46***	.09					
5. Depression	06	.14*	.27***	.10*				
6. Personal Victimization	25***	.32***	.16**	.38***	.32***			
7. Friends' Victimization	08	.06	.05	.08	.00	.15**		
8. Classmates' Victimization	.07	.01	.12*	11*	.10	.04	.22***	

Note. Correlations are derived from a multivariate within twin-pair correlation matrix, * = p < .05, ** = p < .01, *** = p < .001; Child sex is coded such that 1 indicates girls and 0 indicates boys.

Table 2

Multilevel Analyses Predicting Aggression

Model	Predictor	Fixed effect (se)	Level 1 variance (se)	Level 2 variance (se)	log likelihood (np)	ΔLikelihood ratio (df)	р
1			MZ = .69 (.05) DZ = .71 (.13)	MZ = .00 (.00) DZ = .00 (.00)	-475.8 (10)	81.2 (5)	p = .00
	Child Sex Genetic Risk	31*** (.09) .50*** (.07)					
	Personal Victimization	.20*** (.07)					
	Friends' Victimization Classmates' Victimization	.03 (.04) 09* (.04)					
2			MZ = .68 (.08) DZ = .70 (.09)	MZ = .00 (.00) DZ = .00 (.00)	-472.5 (12)	6.2 (2)	p = .04
	Personal Vic x Friends' Vic Personal Vic x Classmates' Vic	.10* (.04) .01 (.04)					
3			MZ = .70 (.08) DZ = .69 (.09)	MZ = .00 (.00) DZ = .00 (.00)	-469.9 (14)	4.7 (2)	p = .09
	Personal Vic x Friends' Vic x Sex Personal Vic x Classmates' Vic x Sex	20* (.09) 01 (.09)					
4			MZ = .71 (.09) DZ = .69 (.05)	MZ = .00 (.00) DZ = .00 (.00)	-467.5 (17)	4.1 (3)	p = .25
	Genetic Risk x Personal Vic	.01 (.03)					

	Genetic Risk x Friends' Vic	.04 (.04)					
	Genetic Risk x Classmates' Vic	.05 (.04)					
5			MZ = .72 (.09)	MZ = .00 (.00)	-466.8 (20)	1.1 (3)	p = .77
			DZ = .70 (.09)	DZ = .00 (.00)	-400.8 (20)		p – .77
	Genetic Risk x Personal Vic x Sex	.05 (.06)					
	Genetic Risk x Friends' Vic x Sex	01 (.08)					
	Genetic Risk x Classmates' Vic x Sex	06 (.08)					

Note. Vic = Victimization; se = standard error ; np = number of parameters; df = degrees of freedom. Δ Likelihood ratio tests are based on multiple imputation procedures described by Asparouhov and Muthén (2010a). The first model is compared to an unconditional model.

Table 3

Multilevel Analyses Predicting Depression Symptoms

Model	Predictor	Fixed effect (se)	Level 1 variance (se)	Level 2 variance (se)	log likelihood (np)	ΔLikelihood ratio (df)	р
1			MZ = .87 (.10) DZ = .96 (.10)	MZ = .00 (.00) DZ = .00 (.00)	-517.2 (10)	55.5 (5)	p = .00
	Child Sex	01 (.08)					
	Genetic Risk	.42*** (.07)					
	Personal Victimization	.25*** (.05)					
	Friends' Victimization	07 (.05)					
	Classmates' Victimization	.05 (.05)					
2			MZ = .84 (.10)	MZ = .00 (.00)	-513.9 (12)	6.3 (2)	$\mathbf{n} = 0.1$
			DZ = .85 (.09)	DZ = .00 (.00)			p = .04
	Personal Vic x Friends' Vic	11* (.05)					
	Personal Vic x Classmates' Vic	.00 (.04)					
3			MZ = .85 (.10)	MZ = .00 (.00)	512 0 (14)	2.1 (2)	n - 21
			DZ = .84 (.09)	DZ = .00 (.00)	-512.8 (14)		p = .34
	Personal Vic x Friends' Vic x Sex	.01 (.10)					
	Personal Vic x Classmates' Vic x Sex	14 (.10)					
4			MZ = .85 (.10)	MZ = .00 (.00)	-510.4 (17)	4.5 (3)	p = .21
			DZ = .84 (.09)	DZ = .00 (.00)			
	Genetic Risk x Personal Vic	01 (.04)					

	Genetic Risk x Friends' Vic	.05 (.04)					
	Genetic Risk x Classmates' Vic	09 (.04)					
5			MZ = .82 (.10)	MZ = .00 (.00)	-508.7 (20)	3.0 (3)	p = .39
			DZ = .84 (.09)	DZ = .00 (.00)			p – .59
	Genetic Risk x Personal Vic x Sex	05 (.08)					
	Genetic Risk x Friends' Vic x Sex	15 (.09)					
	Genetic Risk x Classmates' Vic x Sex	01 (.09)					

Note. Vic = Victimization; se = standard error ; np = number of parameters; df = degrees of freedom. Δ Likelihood ratio tests are based on multiple imputation procedures described by Asparouhov and Muthén (2010a). The first model is compared to an unconditional model.



Figure 1. Illustration of the interaction effect between personal peer victimization experiences and close friends' peer victimization experiences on boys' aggressive behavior.



Figure 2. Illustration of the interaction effect between personal peer victimization experiences and close friends' peer victimization experiences on children's (i.e., girls' and boys') depressive feelings.

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