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Peer Victimization and Anxiety in Genetically Vulnerable Youth: The Protective Roles of Teachers' Self-Efficacy and Anti-Bullying Classroom Rules

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Abstract

Many victimized youngsters are at risk of developing internalizing problems, and this risk seems to be especially pronounced when they are genetically vulnerable for these problems. It is unclear, however, whether protective features of the school environment such as anti-bullying classroom policies and teacher's perceived self-efficacy in handling bullying situations can mitigate these negative outcomes. Using a genetically informed design based on twins, this study examined the potential moderating role of classroom anti-bullying policies and teachers' perceived self-efficacy in handling bullying situations in regard to the additive and interactive effects of peer victimization and genetic vulnerability on anxiety symptoms. To this end, 208 monozygotic and same-sex dizygotic twins (120 girls) rated their level of anxiety and peer victimization in grade 6 (mean age = 12.1 years, *SD* = 2.8). Teachers rated their self-efficacy in handling bullying situations and the extent of anti-bullying classroom policies. Multilevel regressions revealed triple interactions showing that genetic disposition for anxiety predicted actual anxiety for twins who were highly victimized by their peers, but only when their teachers had low perceived self-efficacy in handling bullying situations or when anti-bullying classroom rules were absent or rarely enforced. In contrast, for victimized youth with teachers who perceive themselves as effective or in classrooms where anti-bullying classroom policies were strongly enforced, genetic disposition for anxiety was not associated with actual anxiety symptoms. Anti-bullying programs should continue to promote teachers' involvement, as well as the enforcement of anti-bullying classroom policies, in order to diminish peer victimization and its related consequences.

Keywords: anxiety, peer victimization, gene-environment interaction, teachers, classroom policies

Peer Victimization and Anxiety in Genetically Vulnerable Youth: The Protective Roles of Teachers' Self-Efficacy and Anti-Bullying Classroom Rules

Anxiety is a common problem that generally begins in childhood and affects between one and 11% of children and adolescents (Costello et al. 2011). Anxious children and adolescents experience significant psychological impairments and are at risk for a range of psychiatric disorders in later life, such as depression, general anxiety and conduct disorders (Bittner et al. 2007). Genetically informed research indicates that anxiety in children and adolescents is partly explained by genetic factors (Franić et al. 2010). Specific estimates of genetic effect sizes vary widely, however. For instance, Lau and colleagues (2007) showed that heritability of different anxiety symptoms ranged between 5 to 40% in children and between 29 and 50% in adolescents. The large variability of genetic effect size estimates within each age group may be explained by different anxiety types measured across studies (e.g., general, social or separation anxiety). Another possible explanation is that the role of genetic factors in explaining inter-individual differences in anxiety depends on environmental circumstances, a phenomenon called Gene-Environment Interaction (GxE). In line with GxE, Lau and colleagues (2007) found that negative life events such as a sibling leaving home, parents losing their jobs or a serious illness are associated with a stronger genetic contribution to children's separation anxiety symptoms and adolescents' panic anxiety symptoms. Similarly, results from Eaves and colleagues (2003) suggest that female adolescents who are genetically vulnerable for anxiety are more sensitive to the damaging effects of negative life events than their male counterparts. These findings indicate a diathesis-stress process of GxE, which occurs when an environmental stressor exacerbates the expression of an individual’s genetic predisposition for psychopathology or when the role of an environmental stressor in psychopathological outcomes is exacerbated in genetically vulnerable individuals (Brendgen et al. 2011).

Interactive Effects of Genetic Vulnerability to Anxiety and Peer Victimization

Although the aforementioned studies provide initial evidence that negative experiences may trigger genetic vulnerability for anxiety, additional research is needed to identify specific and proximal environmental stressors that may interact with genetic vulnerabilities to foster anxiety in youth (Gregory and Eley 2007). An especially relevant stressor may be victimization by school bullies. Recent studies showed that around 25% of American adolescents suffer from peer victimization, including cyber bullying (Wang et al. 2009). These youngsters are particularly at risk of developing an array of psychosocial problems including anxiety and depression (Craig 1998). For instance, a review of longitudinal studies showed an important link between peer victimization and subsequent internalizing problems (Reijntjes et al. 2010). There is also some, albeit indirect evidence, that peer victimization interacts with genetic vulnerabilities in predicting internalizing problems. Using a molecular genetic design, Sugden and colleagues (2010) found that frequently bullied youth were more likely to develop depressive symptoms when they were carriers of two short alleles (SS) of the Serotonin Transporter Gene Polymorphism (5-HTTLPR) than children with the short-long (SL) or long-long (LL) genotype. Similarly, Benjet and colleagues (2010) showed that adolescent girls with two short alleles of the 5-HTTLPR gene were more vulnerable to depressive symptoms when they were relationally victimized.

The two aforementioned molecular genetic studies did not specifically examine anxiety symptoms. Nevertheless, anxiety and depression symptoms typically show strong correlations and between 25 and 50% of depressed youth have anxiety symptoms (Axelson and Birmaher 2001). This comorbidity seems to be mostly accounted for by a common genetic liability (Middeldorp et al. 2005). It can therefore be expected that a similar GxE may be observed in the link between peer victimization and anxiety, as worry and fear are likely immediate reactions to victimization experiences. Moreover, anxiety symptoms such as worry and fear are considered proximal stress responses that are more frequent than - and often precede - depressive symptoms in children and adolescents (Zahn-Waxler et al. 2000). The first objective of the present study was therefore to examine whether a diathesis-stress

related GxE process can be observed in the link between peer victimization and adolescents' anxiety symptoms, while controlling for depressive symptoms. To reduce the risk of false positive findings of GxE, it was also important to examine and control for a possible gene-environment correlation (rGE) in the link between anxiety and peer victimization (Purcell 2002). An rGE occurs when an individual's genetically determined traits influence the kind of environment he or she will experience (Scarr & McCartney, 1983). For instance, genetically vulnerable children for anxiety may be more likely to evoke aggressive behaviors such as bullying in their peers than other children. A meta-analysis of existing research supports this view, showing that pre-existing internalizing problems such as anxiety are a significant risk factor of peer victimization (Reijntjes et al., 2010). Because rGE and GxE can co-occur (Purcell 2002), rGE was controlled in the analyses examining GxE.

The Potential Moderating Role of Classroom Policies and Teacher's Perceived Efficacy

From a prevention perspective, it would also be important to know whether the heightened risk of anxiety for genetically vulnerable and victimized adolescents can be attenuated by the presence of positive environmental circumstances. Of particular importance in this regard may be characteristics of the school environment where peer victimization most often takes place (Swearer et al. 2010). The implementation and the enforcement of appropriate school or classroom policies is indeed believed to play an important role in reducing bullying situations and its negative consequences (Farrington and Ttofi 2009). School and classroom policies effective in decreasing school victimization encompass training for parents and teachers, increased playground supervision, precise classroom rules and regular parent-teacher associations (PTA) meetings. Classroom policies are therefore key components in many anti-bullying intervention programs (Ttofi and Farrington 2011). Fekkes and colleagues (2006) showed that schools applying an anti-bullying intervention program reported a decline in active bullying, peer victimization and depression among their students when compared to schools without anti-bullying policies. The presence and extent of classroom anti-bullying policies may therefore be an important moderator of the link between peer victimization and youngsters' anxiety. Moreover, teachers are the

first line agents to implement anti-bullying policies and to intervene in their classroom. For instance, Saarento and colleagues (2013) showed that peer victimization was more frequent in classrooms where teachers were perceived as less disapproving of victimization. However, teachers need to feel competent in handling bullying situations in order to effectively deal with these issues in their classroom. Indeed, school staffs, especially teachers, are more likely to intervene and to improve the situation if they feel efficacious and competent in handling bullying situations (Bradshaw et al. 2007; Dedousis-Wallace et al. 2013). Teachers' perceived self-efficacy to deal with bullying situations may thus be another potential moderator that may mitigate youngsters' anxiety symptoms as a consequence of peer victimization, particularly for genetically vulnerable individuals. Hence, the second objective of this study was to examine the potential moderating role of positive environmental factors related to the teacher and the classroom in regard to the additive or combined effects of peer victimization and genetic vulnerability on anxiety symptoms.

The Present Study

To address these issues, the first objective of this study was to test for potential GxE in the link between peer victimization and anxiety symptoms in early adolescent girls and boys, while controlling for depressive symptoms and accounting for potential rGE between anxiety and peer victimization. The second objective was to examine the moderating role of classroom anti-bullying policies and teachers' perceived self-efficacy in handling bullying situations in regard to the (additive or combined) effects of peer victimization and genetic vulnerability on anxiety symptoms. In line with the theoretical and empirical arguments outlined above, it was expected that safe classroom environments characterized by anti-bullying policies or teachers who perceive themselves to be effective in handling bullying situations may help preventing the expression of youngsters' genetic disposition for anxiety as a consequence of peer victimization. The third objective was to examine the potential moderating effect of child sex on the previous associations. Victimized girls may be more likely to develop anxiety symptoms than victimized boys because girls seem to react more often with internalizing problems to

peer stressors whereas boys react more often with externalizing problems (Grills and Ollendick 2002; Troop-Gordon and Ladd 2005). The grade six (i.e., last year of primary school in Canada) period was chosen because peer relationships become increasingly important starting in early adolescence (Berndt 1979). By the same token, the choice of this period avoids confounding effects of school transition, which may act as an additional important stressor that increases anxiety. To test our hypotheses we used a behavioral genetic design based on monozygotic (MZ) and dizygotic (DZ) twins reared together.

Methods

Participants

The 104 twin pairs (59 MZ pairs, 45 same-sex DZ pairs) 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 by genetic marker analysis of 8-10 highly polymorphous genetic markers and twins were diagnosed as MZ when concordant for every genetic marker. When genetic material was insufficient or unavailable due to parental refusal (43% of cases), zygosity was determined based on physical resemblance questionnaires at 18 months and again at age 9 (Goldsmith 1991; Spitz et al. 1996). The comparison of zygosity based on genotyping with zygosity based on physical resemblance in a subsample of 237 pairs revealed a 94% correspondence rate, which is extremely similar to rates obtained in other studies (Spitz et al. 1996; Magnusson et al. 2013). Eighty-four percent of the families were of European descent, 3% were of African descent, 2% were of Asian descent, and 2% were Native North Americans. The remaining families (9%) did not provide ethnicity information.

The demographic characteristics of the twin families were compared to those of a sample of single births that is representative of the large urban centers in the province of Quebec (SantéQuébec et al. 1998) when the children were 5 months of age. The results showed that the same percentage (95%) of parents in both samples lived together at the time of birth of their child(ren); 44% of the twins compared to 45% of the singletons were the first born children in the family; 66% of the mothers and

60% of the twins' fathers were between 25 and 34 years old compared to 66% of mothers and 63% of fathers for the singletons; 17% of the mothers and 14% of the twins' fathers had not finished high school compared to 12% and 14% of mothers and fathers respectively for the singletons; the same proportion of mothers (28%) and fathers (27%) in both samples held a university degree; 83% of the twin parents and 79% of singleton parents were employed; 10% of the twin families and 9% of the singleton families received social welfare or unemployment insurance; finally 30% of the twin families and 29% of the singleton families had an annual total income of less than CAN$30,000, 44% (42%) had an annual total income between CAN$30,000 and CAN$59,999; and 27% (29%) had an annual total income of more than CAN$60,000. These results indicate extremely similar socio-demographic profiles in the twin sample and the representative sample of single births.

The sample was followed longitudinally at 5, 18, 30, 48, and 60 months focusing on a variety of child-related and family-related characteristics. Follow-up data collections were completed when the children were in kindergarten, grade one, and grades three, four and six. The present paper describes findings from the grade six data collection. The average age at assessment in grade six was 12.1 years (2.8 SD). Attrition in the sample was approximately 3% per year, such that 294 twin pairs participated in grade six. In 220 (74.8%) of these twin pairs, the two twins did not attend the same class. For statistical analyses with twin samples, the same environmental variable (e.g., classroom anti-bullying policies) 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 classes, or at the level of the pair, as was the case when the two twins of a pair were in the same class. Because the two twins of a pair were mostly in different classes, only these pairs were included in the present study. One hundred and sixteen teachers refused to fill out the study questionnaire due to labor disputes, such that one hundred and two twin pairs had complete data for both teacher-rated and self-reported variables. Occasional missing data (i.e., less than 1% of data points) for self-reported anxiety symptoms or self-reported peer victimization levels were imputed with the Estimation Maximization algorithm (Schafer and Olsen 1998), resulting in a final

sample of 208 individual twin children. The nonsignificant Little's MCAR test (χ^2 = 1.985 (2), p = .37) suggested that data were missing completely at random. Twins included in this study did not significantly differ from those excluded in regard to child temperament or any of the socio-demographic measures mentioned previously at 5 months.

Measures

Anxiety symptoms. Anxiety symptoms were assessed via the twins' self-reports using the short version (10 items) of the Revised Children's Manifest Anxiety Scale, Second version (RCMAS-2) (Reynolds and Richmond 2008). The RCMAS-2 is a widely used instrument that evaluates physiological, emotional, and cognitive symptoms of anxiety in youth from 6 to 19 years of age and has shown good reliability and validity in previous studies (Turgeon and Chartrand 2003). Participants indicated, for example, whether in the past month they "were nervous", "were worried", "were afraid of many things", "had trouble falling asleep", "were worried about what other people think about them" and "had stomach-aches". Response options ranged from 1 (never), 2 (once or twice), 3 (several times), to 4 (very often). Item scores were averaged to yield a global anxiety symptoms score (Cronbach's alpha = .80, *ME* = 1.61, *SD* = 0.50).

Peer victimization. Twins rated their level of peer victimization with eight items based on the Social Experiences Questionnaire (Crick and Grotpeter 1996). Victimization was defined as being the target of physical, verbal or psychological attacks at the hand of one or more peers (i.e., behaviors that are different from mere teasing or conflict among equals). Sample items are: "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 1 (never) to 3 (often). Item scores were averaged to yield a global peer victimization score (Cronbach's alpha = .81, *ME* = 1.52, *SD* = .37).

Anti-bullying classroom policies. Anti-bullying classroom policies were assessed through teacher ratings of five items adapted from the Anti-Bullying and Behavioral Policies (ABBP) evaluation scale (Woods and Wolke 2003). The ABBP evaluation scale is based on the core whole-school intervention approach advocated by Olweus (1993) and indicates to what extent policy elements are applied at the school, the classroom or the individual student level. Focusing on three items for the classroom level and two items for the individual student level, teachers indicated to what extent they "had special class meetings about the topic of bullying", "had parent-teacher association (PTA) meetings about the topic of bullying", "had precise anti-bullying classrooms rules", "had serious (formal) talks with both bullies and victims following bullying incidents" and "had serious (formal) talks with the parents of bullies and victims following bullying incidents". Responses were given on a three-point scale, ranging from 1 (never) and 2 (once) to 3 (at regular intervals) for the first three items and from 1 (never) and 2 (sometimes) to 3 (always) for the last two items. Because anti-bullying policies can only be effective if they are consistently applied and reinforced (Smith et al. 2004), each policy item was then dichotomized such that a value of 1 was given when a specific policy was consistently reinforced (i.e., at regular intervals or always) and a value of 0 otherwise (i.e., never or only once or sometimes). Item values were then summed to yield a global anti-bullying classroom policies score (*ME* = 2.48, *SD* = 1.29). The global anti-bullying classroom policies measure can thus be conceived as a count variable: policies can take different forms in different classroom environments but having more policies that are consistently reinforced can generally be considered as being better than having only a limited number of reinforced anti-bullying policies.

Teachers' perceived self-efficacy. The teachers were asked to rate their perceived level of self-efficacy in handling bullying situations on six items adapted from the previously mentioned ABBP evaluation scale (Woods & Wolke, 2003), which are similar to a self-efficacy measure recently used by Oldenburg and colleagues (2014). Specifically, teachers were asked how efficacious they felt about applying certain anti-bullying policies on a five-point scale ranging from 1 (definitely not able) to 5

(definitely able). For example: "Do you feel able to… support a child who is a victim of bullying?,… have serious talks with bullies/victims/parents?,… talk to children who have witnessed bullying about their responsibilities?, … intervene in such a way that the bullying stops?". Item scores were averaged to yield a global teachers' self-efficacy score (Cronbach's alpha = .88, *ME* = 4.13, *SD* = .65).

Depressive symptoms. Depressive symptoms were assessed via the twins' self-reports using the brief version of the Children's Depression Inventory (CDI; Kovacs 1992). The Short Form of the CDI evaluates affective, cognitive, motivational and somatic symptoms of depression and has been validated using normative and clinic-referred samples, showing relatively high internal consistency and stability (Allgaier et al. 2012). Participants indicated, for example, whether in the past two weeks they "felt like crying", "found it hard to make choices", "wondered if someone loves them", "felt alone" and "didn't like themselves". Response options ranged from 1 (rarely), 2 (often), to 3 (always). Item scores were averaged to yield a depressive symptoms score (Cronbach's alpha = .74, *ME* = 1.24, *SD* = .31).

Procedure

All instruments were administered in either English or French, depending on the language spoken by the children and the teachers. Instruments that were administered in French but were originally written in English were first translated into French and then translated back into English. Bilingual judges verified the semantic similarity between the back-translated items and the original items. Children's verbal assent as well as active written consent from the parents were obtained. Data collection took place in the spring. Teacher measures were assessed via paper-and-pencil questionnaires that covered the current school year and were completed either in the school office or at home. Twins' self-report measures were completed during a home visit during which twins completed a paper-and-pencil questionnaire alone in separate rooms. The instruments were approved by the Institutional Review Boards of the University of Quebec at Montreal and the Ste. Justine Hospital.

Results

Estimation of Genetic and Environmental Effects on Children's Anxiety

The twin design makes it possible to assess the relative role of genetic factors and environmental factors associated with a given phenotype (e.g., a behavior or trait; Falconer, 1989). The examination of intra-pair correlations for MZ twins (who are assumed to share 100% of their genes) and same-sex DZ twins (who on average share 50% of their genes) can be used to roughly estimate the sources of variability of anxiety in terms of genetic and environmental factors. The relative strength of additive genetic factors on individual differences (a^2) is approximately twice the difference between the MZ and the same-sex DZ intra-pair correlations, $a^2 = 2(r_{MZ} – r_{DZ})$. The relative strength of shared environmental factors that affect twins within a pair in a similar way (c^2) can be estimated by subtracting the MZ intra-pair correlation from twice the DZ intra-pair correlation, $c^2 = 2r_{DZ} – r_{MZ}$. Non-shared environmental factors that uniquely affect each twin in a pair (e^2) are approximated by the extent to which the MZ intra-pair correlation is less than 1, $e^2 = 1–r_{MZ}$. In the present study, the MZ intra-pair correlation for anxiety symptoms (r = .24) appears to be three time as high as the corresponding same-sex DZ correlation (r = .08), suggesting a significant contribution of genetic factors, whereas shared environmental influences may play only a small role. Still, the overall magnitude of the MZ intra-pair correlation was well below 1.0, indicating a significant contribution of nonshared environmental factors. However, these correlations represent additive effects of genetic and environmental factors and do not consider potential gene-environment interactions.

Calculation of Genetic Risk for Anxiety Symptoms

An ordinal scale of genetic risk for anxiety symptoms was computed based on a formula developed by Ottman (1994). This method has been used in several studies to test the presence of GxE with an epidemiological twin design (Jaffee et al. 2005; Wichers et al. 2009; Brendgen et al. 2013). Each twin pair was represented in the data set twice, with each twin serving as “the target twin” and also as the other twin’s “co-twin”. For each target twin, genetic risk for anxiety was computed as a function of (a) zygosity and (b) the presence or absence of anxiety symptoms in the co-twin. To represent presence or absence of anxiety symptoms, the continuous global anxiety scale was

dichotomized using 0.5 SD above the sample mean as the cut-off, which corresponds to a value of 1.86 or higher on the continuous global anxiety scale. Children whose anxiety score was at or above 0.5 SD above the mean of the sample distribution were considered as being anxious, the others were considered as not being anxious. The presence or absence of anxiety in the co-twin was then combined with information on the pair's zygosity into an index of genetic risk for anxiety symptoms ranging from 0 to 3. The target twin's genetic risk for anxiety was therefore considered to be highest (3) when he or she was part of an MZ pair and when anxiety symptoms were present in the co-twin. The target twin's genetic risk for anxiety was somewhat lower (2) when he or she was part of a DZ pair and when anxiety symptoms were present in the co-twin. The target twin's genetic risk for anxiety was even lower (1) when he or she was part of a DZ pair and when the co-twin was not anxious. Finally, the target twin's genetic risk for anxiety was lowest (0) when he or she was part of an MZ pair and when the co-twin was not anxious. Notably, all analyses described below were also performed using different cut-offs (i.e., the mean level and 1SD above the sample mean), yielding very similar results. However, the cut-off of 0.5 SD above the sample mean was chosen because it indicated more elevated levels of anxiety than the sample mean (i.e., it corresponded to experiencing four anxiety symptoms several times and one symptom at least once or twice) while at the same time ensuring a sufficient sample size at the different levels of the genetic risk factor and therefore enough statistical power for subsequent analyses. The number of boys and girls at each level of genetic risk for anxiety is provided in Table 1. For the logic of the ordinal genetic risk index, it was important to ensure that MZ and DZ twins did not differ in regard to their anxiety symptoms. Multilevel regressions using generalized estimating equations (GEE) to account for the interdependence of twin data revealed that zygosity was not significantly associated with self-reported anxiety symptoms ($ß$ = -.12, *SE* = .11, *ns*).

Main Analyses: Analytical Rationale

Before conducting the main analyses, bivariate correlations were examined to assess associations between study variables (see Table 2). Twin's depressive symptoms (r = .23), anxiety

symptoms (r = .38) and being a girl (r = .22) were associated with higher levels of peer victimization. Genetic risk for anxiety (r = .15) was also associated with higher levels of peer victimization, indicating the presence of rGE. Depressive symptoms and genetic risk for anxiety were associated with more anxiety symptoms, respectively (r = .36; r = .12). A larger number of reinforced anti-bullying classroom policies was associated with a higher level of teacher's perceived self-efficacy in handling bullying situations (r = .35).

According to our hypotheses, the additive and combined effect of genetic risk for anxiety and peer victimization should predict twin's anxiety symptoms. Moreover, these associations should be moderated by anti-bullying classroom policies and teachers perceived self-efficacy. To test these hypotheses, multilevel regressions using generalized estimation equations (GEE) to account for the interdependence of twin data (i.e., two twins nested in a pair) were performed with the Statistical Package for the Social Sciences (SPSS) v. 22 software (IBM Corp. 2013). One set of consecutive models was 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 Quasi-likelihood under independence model criterion (QIC). While this fit index does not allow formal model comparisons, it can be used as a guideline for model selection, with lower values indicating a better overall model fit (Pan 2001). All variables except child sex were z-standardized prior to analyses to facilitate interpretation of effect sizes. Table 3 presents the results from the analyses.

The first model tested was an unconditional model, without including any predictors, which provided preliminary information about the model fit (QIC = 209.3). Added in the second model, depressive symptoms and genetic risk for anxiety were positively associated with self-reported anxiety symptoms, respectively (b = .35, SE = .13, $p \leq .01$; b = .28, SE = .07, $p < .001$), whereas child sex was not (b = .06, SE =.11, p = .60). Inclusion of these predictors resulted in a better model fit compared to the previous model (QIC = 197.7). The three variables added in the third model, contributed to a further

overall improvement of model fit (QIC = 177.1). However, only higher levels of peer victimization were significantly related to self-reported anxiety symptoms (b = .31, SE =.07, p < .001). No significant association emerged between anxiety symptoms and teachers' perceived self-efficacy (b = -.04, SE = .06, p =.54) or anti-bullying classroom policies (b = -.02, SE = .07, p =.77). In the fourth model, four two-way interaction terms were added to test the moderating effect of child sex: "genetic risk for anxiety * sex", "peer victimization* sex", "teachers' self-efficacy * sex", and "anti-bullying classroom policies * sex". Because these two-way interactions with child sex emerged as non-significant and seemed to lead to a relatively worse overall model fit (QIC = 183.7), they were removed from further analyses.

In the fifth model, three two-way interaction terms were included to test whether the effect of genetic risk for anxiety on actual anxiety symptoms was moderated by peer victimization, teacher efficacy, or classroom policies. Inclusion of these interactions resulted in a better model fit (QIC = 175.0) compared to the previous model without interactions. Indeed, there was a significant interaction between genetic risk for anxiety and peer victimization, albeit with a statistical trend (b = .12, SE = .07, p < .10) as well as a significant interaction between genetic risk for anxiety and anti-bullying classroom policies (b = -.15, SE = .05, p < .001). However, no interaction emerged between genetic risk and the teachers' self-efficacy. In the sixth model, two additional two-way interactions terms were added to test the potential moderating effects of teacher self-efficacy or classroom policies on the link between peer victimization and anxiety. Overall relative model fit did not change much (QIC = 177.6) but none of these two-way interactions were significant.

In the next models (7a and 7b), two three-way interaction terms were tested separately to reduce problems of multicollinearity due to multiple intercorrelated interaction terms. Both models showed relatively comparable overall model fit as the preceding model (QIC = 178.1 for model 7a and QIC = 177.9 for model 7b). In model 7a, the three-way interaction effect: "genetic risk for anxiety * teachers' self-efficacy * peer victimization" was significant (b = -.10, SE = .05, p < .05). To illustrate this

interaction effect (see Figure 1), we examined the association between genetic risk for anxiety and actual anxiety symptoms for four sample cases that represented different levels of peer victimization and teachers' perceived self-efficacy, i.e., when either one or both moderators were at high levels (1SD above the mean) or at low levels (1SD below the mean). Not surprisingly, when the level of peer victimization was low, genetic risk for anxiety was not associated with actual anxiety symptoms regardless of whether the teacher's self-efficacy to handle bullying incidences was high (*b* = .19, S*E* = .10, *ns*) or low (*b* = -.09, S*E* =.10, *ns*). In contrast, when the level of peer victimization was high but the teacher's perceived self-efficacy to handle bullying incidences was low, genetic risk for anxiety was significantly related to actual anxiety symptoms (*b* = .37, S*E* = .16, $p < .05$). However, when peer victimization was high but the teacher's perceived self-efficacy to handle bullying incidences was also high, genetic risk for anxiety was no longer predictive of actual anxiety symptoms (*b* = .24, S*E* = .13, *ns*).

In model 7b, the three-way interaction term: "genetic risk * anti-bullying classroom policies * peer victimization" was also found to be significant (*b* = - .15, S*E* = .06, $p < .05$). To illustrate this interaction effect, the same procedure as previously described was used (see Figure 2). When the level of peer victimization was low, genetic risk for anxiety was not associated with actual anxiety symptoms, regardless of whether anti-bullying classroom policies were regularly (*b* = .07, S*E* = .11, *ns*) or rarely enforced (*b* = .01, S*E* = .13, *ns*). In contrast, when the level of peer victimization was high and anti-bullying classroom policies were rarely enforced, genetic risk for anxiety was significantly related to actual anxiety symptoms (*ß* = .59, *SE* =.12, $p < .001$). However, when the level of peer victimization was high but anti-bullying classroom policies were regularly enforced, genetic risk for anxiety was no longer predictive of actual anxiety symptoms (*b* = .05, S*E* = .13, *ns*). Notably, all interactions emerged while controlling for the overlap or association between genetic risk for anxiety and peer victimization (i.e., rGE), as both of these variables were already included as unique predictors in steps 2 and 3 of the model.

Discussion

The first objective of the present study was to examine GxE in the link between peer victimization and anxiety symptoms in youth, while controlling for depressive symptoms and accounting for potential rGE. The second objective was to examine the moderating role of classroom anti-bullying policies and teachers' perceived self-efficacy in handling bullying situations in regard to the effects of peer victimization and genetic vulnerability on anxiety symptoms. The third objective was to examine the potential moderating effect of child sex on the previous associations.

Gene-Environment Interaction between Anxiety and Peer Victimization

Genetically vulnerable victims displayed anxiety symptoms to a greater extent than others, indicating a diathesis-process of GxE. These results are in line with previous genetically informed studies suggesting that individuals who are genetically vulnerable for depression are more likely than others to be depressed when they are victimized (Sugden et al. 2010; Benjet et al. 2010). The fact that anxiety and depression share common genetic influences (Middeldorp et al. 2005) may explain the similar GxE linking peer victimization with these two internalizing problems. Moreover, findings from a neuroimaging study show that, compared to individuals with low trait anxiety, individuals with high trait anxiety display not only greater amygdala reactivity to fear stimuli but also less ventral prefrontal cortical recruitment to downregulate their fear responses (Indovina et al. 2011). Our results suggest that a stressful experience such as frequent peer victimization seems to be an important environmental trigger for the expression of these dispositional vulnerabilities. Notably, these GxE were observed even when accounting for rGE. Indeed, genetic risk for anxiety was significantly associated with a higher level of peer victimization, which could be due to the fact that genetically vulnerable children may be considered as easy targets and thus fall prey to bullying more frequently than other children (Brendgen et al., 2014; Reijntjes et al., 2010). Importantly, however, the present findings suggest that protective factors of the school context moderate the link between genetic vulnerability for anxiety, peer victimization and actual anxiety symptoms.

The Moderating Role of Teacher's Perceived Self-Efficacy and Anti-Bullying Classroom Policies

A genetic disposition for anxiety predicted actual anxiety symptoms only for those youngsters who were highly victimized and whose teacher had low perceived self-efficacy in dealing with bullying. For victimized youngsters with teachers who perceive themselves as effective – and similar to youth who were not victimized – genetic risk for anxiety was not associated with actual anxiety symptoms. These results add to other studies on peer victimization suggesting that teacher's perceived efficacy seems to play a role in reducing bullying. For instance, Veenstra and colleagues (2014) found that classrooms with teachers that were not perceived by their students as competent in reducing bullying displayed a higher level of peer victimization. However, the results of the present study suggest that efficacious teachers might not only decrease bullying but also buffer genetically vulnerable individuals from the negative consequences of peer victimization.

Teachers who perceived themselves as competent in handling bullying situations might be more likely than others to endorse and openly express disapproving attitudes toward bullying and to use effective anti-bullying strategies such as increased supervision (Saarento et al. 2013; Troop-Gordon and Ladd 2013). Hence, genetically vulnerable youngsters with proactive and protective teachers that disapprove bullying might feel secure in their classroom, and therefore less anxious, even when they are victimized. They may feel that their teachers will intervene when needed and help them during difficult social situations. Moreover, competent teachers might promote or model helpful response strategies such as non-aggressive assertion (Troop-Gordon and Ladd 2013). These strategies may help vulnerable youngsters to deal not only with concrete bullying incidences but also with their own anxiety and fears. Hence, teachers who perceive themselves as competent in handling bullying situations seem to play a significant role in preventing the expression of a genetic disposition for anxiety. However, the specific mechanisms linking teacher's involvement, peer victimization and anxiety should be investigated in future studies.

Teachers are also the ones implementing and enforcing anti-bullying classroom policies such as PTA meetings, classroom rules and follow-ups after bullying incidents. The results of the present study showed that, in classrooms where anti-bullying policies were regularly enforced, genetic risk for anxiety was not associated with actual anxiety symptoms in victimized youth. In contrast, in classrooms where anti-bullying policies were rarely enforced, genetic risk for anxiety was significantly related to actual anxiety symptoms for victimized youngsters. The latter results are also in line with previous studies showing that appropriate anti-bullying policies seem to decrease the level of peer victimization and its negative consequences. For instance, recent results from the anti-bullying program KIVa suggest that program components such as classroom lessons that promote positive social interactions, self-confidence and interpersonal self-efficacy decreased the level of students' social anxiety (Williford et al. 2012). Moreover, Fekkes and colleagues (2006) showed that teacher training and the development of written anti-bullying school policies reduced peer victimization as well as depression in intervention schools. However, when schools diminished their anti-bullying efforts, there were no differences between the intervention and the control schools in regard to the level of peer victimization and its related consequences. Hence, the implementation and the enforcement of appropriate anti-bullying policies seem to reduce the negative consequences of peer victimization such as anxiety and depression. The results of the present study suggest, however, that anti-bullying classroom policies may be particularly helpful for individuals who are genetically vulnerable for anxiety, as these vulnerable youngsters might feel safer in classrooms with clear rules and strict follow-ups after bullying incidences.

Strengths, Limitations and Conclusions

The present study has a number of positive features. First, a multi-source approach was employed to reduce potential bias due to shared source variance. Twins reported about their own level of depression, anxiety and victimization, whereas teachers reported about their own level of self-efficacy and their classroom anti-bullying policies. Second, this study used a genetically informed

design to assess GxE between peer victimization and anxiety. Although genetically informed studies cannot provide conclusive proof of causation, such studies can nevertheless help examine causal hypotheses that are difficult to test unequivocally without the use of experimental designs (Moffitt 2005) or where experimental designs are not feasible due to ethical or other considerations. Indeed, by disentangling genetic from environmental sources of interindividual variation, even cross-sectional data from sibling designs such as those based on twins "provide robust quasi-experimental tests of causal environmental hypotheses" (Lahey and D'Onofrio 2010). In the context of the present study, this design helped investigate to what extent a genetic vulnerability for anxiety interacts with peer victimization and classroom rules or teacher characteristics to predict anxiety while also controlling for any predictive effect of a genetic vulnerability for anxiety on the risk of experiencing peer victimization. Third, by adding the examination of protective school context factors to its genetically informed design, this study also has important practical implications.

The present study also has several limitations that need to be considered when interpreting the results. First, the study was based on cross-sectional correlational data. Although this limitation is partly offset through the use of a genetically informed design, an experimental design in the context of a prevention program - especially when combined with genetically informative data - would provide the ideal framework to test the causal hypotheses of the present study. Second, it should be mentioned that the important number of missing data may have affected to some extent the findings of the present study. Indeed, one hundred and sixteen teachers refused to complete the study questionnaire due to labor disputes and teachers' perceived self-efficacy in dealing with bullying and anti-bullying classroom policies may have been more strongly enforced in classrooms where teachers agreed to participate in the study. The resulting reduced variance and lack of power may have led to an underestimation of associations. A related limitation concerns the cut-off used to compute the genetic risk scale for anxiety (0.5 SD above the mean), which may also have contributed to an underestimation of effect sizes, especially for youth at risk for clinical anxiety levels. Indeed, Reynolds and Richmond

(1978) suggested that scores within one standard deviation of the mean may indicate the normal range of variability for anxiety. While analyses performed using 1SD above the sample mean as a cut-off yielded very similar results, the value of 0.5 SD above the sample mean was chosen to maximize statistical power. It is important to note however that both triple interactions were significant despite the limited statistical power. Results should therefore be replicated in longitudinal studies with a larger sample size. Finally, it may have been useful to measure the teachers' self-efficacy to handle bullying incidences as perceived by their students as there might be an important discrepancy between teachers' and students' perceptions. Moreover, students' collective perception of their teacher's self-efficacy and attitudes seems to be an important predictor of classroom levels of bullying and peer victimization (Veenstra et al. 2014; Saarento et al. 2014).

Despite these limitations, the present study offers new insights into the complex interaction between children's genetic disposition for anxiety and peer victimization, as well as the protective factors of the school environment. Indeed, genetically vulnerable youngsters that were in a safe classroom environment did not display greater anxiety symptoms even when they were highly victimized. Anti-bullying prevention and intervention programs should therefore continue to promote teachers' involvement, as well as the implementation and the enforcement of quality anti-bullying classroom policies, in order to diminish peer victimization and its related consequences such as anxiety symptoms.

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Table 1

Number of Boys and Girls by Genetic Risk Status

Genetic Risk Status	Boys (n = 88)	Girls (n = 120)	Total
Highest Risk (MZ)	16	17	33
High Risk (DZ)	11	22	33
Low Risk (DZ)	29	28	57
Lowest risk (MZ)	32	53	85

Table 2

Bivariate Correlations Among Study Variables

Variable	1	2	3	4	5	6	7
1. Sex	-						
2. Depressive symptoms	-.01	-					
3. Genetic risk for anxiety	.05	.07	-				
4. Peer victimization	.22**	.23**	.15*	-			
5. Teachers self-efficacy	-.02	-.07	.00	.04	-		
6. Classroom policies	.10	-.09	.02	.05	.35**	-	
7. Anxiety symptoms	-.02	.36**	.12^{t}	.38**	-.05	-.06	-

Note. N = 208 twins. Sex is coded 0 = boys, 1 = girls. $^{t}p < .10$; * $p < .05$; **$p < .01$.

Table 3

Multilevel Regression Analyses Assessing the Predictive Links of Genetic Risk, Classroom Policies, Teachers' Self-Efficacy and Peer Victimization with Twins' Anxiety Symptoms

Parameter	QIC	b	SE
Model 1	209.3		
Unconditional model			
Model 2	197.7		
Depression		.35**	.13
Sex		.06	.11
Genetic risk		.28***	.07
Model 3	177.1		
Peer victimization		.31***	.07
Teachers' self-efficacy		-.04	.06
Classroom policies		-.02	.07
Model 4	183.7		
Sex * Genetic risk		.04	.14
Sex * Peer victimization		.17	.12
Sex * Teachers' self-efficacy		.13	.14
Sex * Classroom policies		-.08	.15
Model 5	175.0		
Genetic risk * Peer victimization		.12^{t}	.07
Genetic risk * Teachers' self-efficacy		.07	.07
Genetic risk * Classroom policies		-.15*	.05
Model 6	177.6		
Teachers' self-efficacy * Peer victimization		-.03	.04
Classroom policies * Peer victimization		.00	.06
Model 7a	178.1		
Teachers' self-efficacy * Peer victimization* Genetic risk		-.10*	.05
Model 7b	177.9		
Classroom policies * Peer victimization* Genetic risk		-.15*	.06

Note. N = 208. QIC = Quasi Likelihood under Independence Model Criterion. QIC can be used to informally compare different models, with smaller QIC values indicating a "better" fit to the data. SE = Standard Error.

^{t} $p \leq .10$; * $p < .05$; ** $p \leq .01$; *** $p \leq .001$.

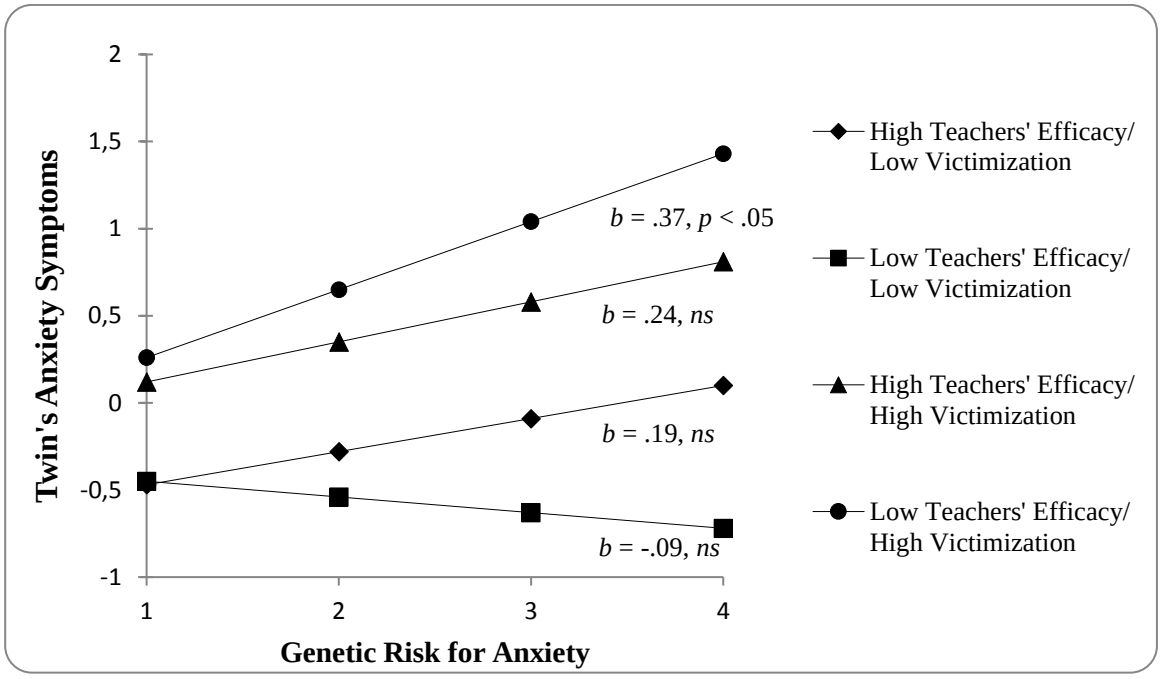

*Figure 1***.** Illustration of the triple interaction between teachers' perceived self-efficacy to handle bullying situations, twin's genetic risk for anxiety and level of peer victimization

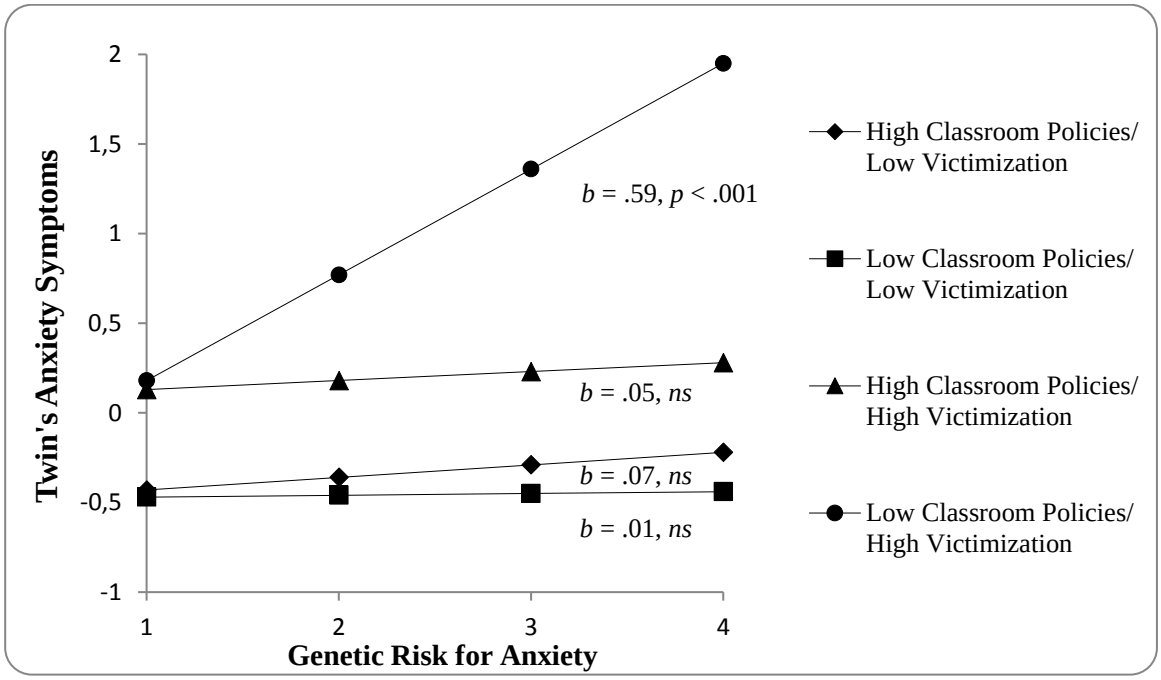

Figure 2. Illustration of the triple interaction between anti-bullying classroom policies, twin's genetic risk for anxiety and level of peer victimization