Gene–Environment Processes Linking Peer Victimization and Physical Health Problems: A Longitudinal Twin Study

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Objective This study examined whether (a) a genetic disposition for physical health problems increases the risk of peer victimization and (b) peer victimization interacts with genetic vulnerability in explaining physical health problems. **Methods** Participants were 167 monozygotic and 119 dizyogtic twin pairs. Physical symptoms were assessed in early childhood and early adolescence. Peer victimization was assessed in middle childhood. **Results** Genetic vulnerability for physical health problems in early childhood was unrelated to later peer victimization, but genetic vulnerability for physical health problems during early adolescence increased the risk of victimization. Victimization did not interact with genetic factors in predicting physical symptoms. Environmental, not genetic, factors had the greatest influence on the development of physical symptoms in victims. **Conclusion** Genetic vulnerability for physical health problems in early adolescence increases the risk of peer victimization. Whether victims suffer a further increase in physical symptoms depends on the presence of protective environmental factors.

Key words gene–environment correlation; gene–environment interaction; peer victimization; physical health problems; twins.

Peer victimization, defined as being the victim of physical or relational forms of aggression and harassment by peers, is a major public health concern. Around 75% of youth report being victimized by peers at least once over the course of a year (Glover, Gough, Johnson, & Cartwright, 2000) and 20–30% of youth are chronically victimized (Storch & Ledley, 2005). Victimized youth are two times more likely to present with physical health problems such as respiratory infections (colds, chronic coughs, or bronchitis), psycho-physiological disorders (asthma, stomach ulcers, chronic headaches), or skin disorders (hives, eczema, or psoriasis) (Gini & Pozzoli, 2009). Peer victimization is also associated with more frequent visits to the school nurse for somatic complaints, illness, and injury (Vernberg, Nelson, Fonagy, & Twemlow, 2011). It has been suggested that stress due to repeated peer victimization weakens the immune system, leaving youngsters vulnerable to infection and physical illness (Rigby, 1998). In line with this notion, alterations in neuroendocrine functioning explain part of the association between peer victimization and physical health problems (Knack, Jensen-Campbell, & Baum, 2011).

The physical health problems that are linked to peer victimization (e.g., allergic diseases, asthma, chronic

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headaches) show considerable genetic influence (Holloway, Yang, & Holgate, 2010; Ligthart, Nyholt, Penninx, & Boomsma, 2010; Thomsen, Van Der Sluis, Kyvik, Skytthe, & Backer, 2010). As proposed by Shostak (2003), environmental stressors and genetic vulnerability factors may not act independently but rather interact with each other to produce ill physical health. Shanahan and Hofer (2005) have described two forms *of gene–environment interaction* (G × E) that may occur in this context. Specifically, in line with a *diathesis-stress* process of G × E, an environmental stressor such as peer victimization may trigger or exacerbate the effect of genetic vulnerabilities, such that health problems are mainly observed in genetically vulnerable youth. For instance, findings from two molecular genetic studies suggest that the deleterious effect of peer victimization on depression symptoms is especially pronounced in early adolescents carrying two 5-HTTLPR short alleles, which increases their vulnerability to developing depression (Benjet, Thompson, & Gotlib, 2010; Sugden et al., 2010). Alternatively, one may observe a *suppression* process of G × E. This occurs when a stressful environmental experience reduces the role of genetic factors in explaining interindividual differences in health problems, such that many victims exhibit health problems, irrespective of their genetic vulnerabilities. Such a *suppression* process of G × E may be especially likely in younger victimized children, who often have not yet developed the social and emotional skills to cope with peer harassment (Smith, Shu, & Madsen, 2001). Support for this notion comes from findings that genetic vulnerability explained a large part of depression symptoms during kindergarten only in popular children, who presumably face little peer-related stress. In contrast, children who were rejected by their peers showed depressive symptoms regardless of their genetic disposition (Brendgen et al., 2009). It is still unknown, however, whether G × E can also be found in the link of peer victimization and physical health problems and whether such an interaction is in line with a trigger or a suppression process of G × E.

In addition to interacting with peer victimization in producing ill health, a genetic vulnerability for physical health problems may also increase youngsters' risk of being harassed by peers. Environmental experiences are not necessarily independent of genetic factors because individuals can evoke their environment as a function of heritable traits (Scarr & McCartney, 1983). In the process, genetic and environmental features become correlated. Such *gene–environment correlation* (rGE) may be seen as a special case of ''child effects'' on peer victimization (Kendler & Eaves, 1986). There is evidence of an increasing genetic contribution to peer victimization during the elementary school period, but it is unclear whether genetic factors related to physical health problems are at play in this context (Brendgen, 2012). Non-genetically informed studies investigating physical health problems as a risk factor of peer victimization have produced mixed results. Short-term longitudinal studies found no association between generalized physical health problems and peer victimization six months later (Fekkes, Pijpers, Fredriks, Vogels, & Verloove-Vanhorick, 2006; Nishina, Juvonen, & Witkow, 2005). Still, a different picture may emerge for youth with a more consistent, genetically based disposition for ill health. Thus, children with chronic physical health conditions such as epilepsy, eczema, or Tourette syndrome are more likely than other children to be the target of peer victimization (Sentenac et al., 2012). Although many of these conditions may have existed before children's first contact with peers, the cross-sectional nature of these latter studies makes it difficult to determine the directionality of effects. One way to address this issue is to investigate genetic susceptibility for physical health problems as a potential risk factor of peer victimization, as would be indicated by the presence of rGE.

Importantly, rGE and G × E often co-occur, and testing one without the other can lead to biased estimates, as the same environmental risk factor may be involved in both rGE and G × E (Purcell, 2002). Thus, Lau and Eley (2008) found that genetic factors influencing depression increased adolescents' risk of experiencing negative life events (rGE). In turn, negative life events exacerbated the effect of genetic factors on depression (G × E). It remains to be seen whether both rGE and G × E can also be observed in the link between peer victimization and physical health problems in youth.

The Present Study

The goal of this study was to examine potential rGE and G × E linking high exposure to victimization by peers during childhood with physical health problems. To this end, we used a longitudinal quantitative genetic design based on a sample of monozygotic (MZ) and same-sex dizygotic (DZ) twin pairs reared together (see description of the underlying rationale below). Peer victimization was assessed during elementary school, that is, the period when peer victimization tends to be highest (Smith, Madsen, & Moody, 1999). Physical health problems were assessed during early childhood and in early adolescence. By separating assessments of early childhood health problems from assessments of peer victimization in school, we could examine whether a genetic vulnerability that is already prevalent early in life is associated with an increased

risk of peer victimization later on. In line with rGE, we expected that children with a genetic disposition for physical health problems would be more at risk of being victimized by their peers. In particular, a genetic disposition for physical health problems already early in life should be associated with an increased risk of peer victimization later on. If such rGE with genetic vulnerability for early physical health problems were found, it would help screen young children at risk of being victimized by peers to take preventive measures at an early stage. However, because physical health complaints such as digestive tract problems and respiratory infections are fairly common in young children, it was also possible that genetic vulnerabilities for physical health problems that occur *after* early childhood (i.e., health problems that are observed in early adolescence) contribute to the risk of peer victimization. In line with G × E, we expected that peer victimization interacts with genetic vulnerability in explaining later physical health problems while controlling for earlier physical health problems. In this regard, either a trigger process or a suppression process was possible, both theoretically and based on the previously mentioned G × E linking peer victimization with mental health.

Method

Sample

Participants of the present 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. 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. 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 <$30,000. These demographic characteristics were highly comparable with those of a sample of single births representative of the urban centers in the province of Quebec, initiated by the Quebec Ministry of Health and Social Services (Jetté & Des Groseilliers, 2000).

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. As is common practice in twin studies (e.g., Burt & Klump, 2013; Hopper, Foley, White, & Pollaers, 2013; Magnusson et al., 2013), zygosity was determined based on physical resemblance questionnaires at 18 months and again at age 9 (Goldsmith, 1991; Spitz et al., 1996) when genetic material was insufficient or unavailable owing to parental refusal (43% of cases). The comparison of zygosity based on genotyping with zygosity based on physical resemblance in a subsample of 237 same-sex pairs revealed a 94% correspondence rate, which is extremely similar to rates obtained in other studies (Magnusson et al., 2013; Spitz et al., 1996).

The sample was followed with distinct assessments at ages 0.5, 1.5, 2.5, 4, 5, 6, 7, 9, 10, 12, and 13 years. To be included in the analyses for the present study, participants needed to have at least one valid measurement time point for (a) early childhood physical health problems, (b) childhood peer victimization, and (c) early adolescent physical health problems, respectively. These conditions were met for 286 out of the initial 448 twin pairs. The final study sample of 286 twin pairs (167 MZ pairs, 119 same-sex DZ pairs) did not differ from excluded participants in regard to physical health problems in early childhood, mothers' or fathers' education, or parents' age at the birth of the twins. However, family income was higher in the final study sample. There were 16% of missing data points in the 286 twin pairs of the final sample. However, the vast majority (84%) of these 286 twin pairs had at least three of the five data points for physical health in early childhood, at least three of the five data points for peer victimization in middle childhood, and at least one of the two data points for physical health in early adolescence. Because all participants in the final study sample had at least one valid data point for each of the three study variables as per our selection criterion, and because each overall scale was calculated as the mean across all available data points (see description below), the analyses contained no missings.

Measures

Physical health problems in early childhood were assessed when the children were 0.5, 1.5, 2.5, 4, and 5 years old via mother ratings. Each time, 12 items assessed the presence of a range of different physical health problems such as asthma, allergies, bronchitis, nose–throat infection, digestive tract or urinary health problems, heart palpitations, kidney problems. For each item, the response scale ranged from 0 (no) to 1 (yes). The respective item scores were summed, separately for each assessment time, to form an overall symptom count scale (1-year stability of physical health problems varied from $r = .37$–.43). The four yearly symptom count scores were then averaged to represent a

child's overall level and chronicity of physical health problems during early childhood to be used in the analyses [MZ mean = .57, standard deviation (*SD*) = .53, minimum = 0.00, maximum = 4.25; DZ mean = .55, *SD* = .63, minimum = 0.00, maximum = 4.67].

Physical health problems in early adolescence were assessed when the children were 12 and 13 years old via self-reports on 10 items similar to those of other widely used self-reported physical health complaints scales (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974; Pennebaker, 1982). The items assessed a range of general physical health problems that may have occurred since the beginning of the school year and that resembled those assessed via mother reports in early childhood. Symptoms included frequent digestive tract problems (nausea, stomachaches), migraines, vertigo, asthma attacks, nose–throat infections, chest pains, heart palpitations, and loss of consciousness. For each item, the response scale ranged from 0 (never), 1 (rarely), 2 (sometimes), to 3 (often). The respective item scores were averaged, separately for each assessment time (stability $r = .56$), and the age 12 and age 13 scales were then averaged to represent the overall level of physical health problems in early adolescence to be used in the analyses (MZ mean = .57, *SD* = .34, minimum = 0.00, maximum = 1.96; DZ mean = .58, *SD* = .37, minimum = 0.00, maximum = 2.33).

Peer victimization was assessed at ages 6, 7, 9, 10, and 12 via teacher ratings of three items: ''was called names by other children,'' ''was hit or pushed by other children,'' ''was made fun of by other children.'' Equivalent teacher ratings in elementary school have been related to child self-ratings as well as to mother ratings of peer victimization in a population-based sample of children (Barker et al., 2008). For each item, the response scale ranged from 0 (never), 1 (sometimes), to 2 (often). The respective item sores were averaged, separately for each assessment time (1-year stability of peer victimization varied from $r = .21$ to .41). Growth curve analyses using Robust Hubert-White-Sandwich estimators for confidence intervals (CIs) to account for data interdependence due to twinning revealed that peer victimization followed a flat trajectory with no significant mean change (slope mean = −.01, $p = .12$) and no significant variance around the level of change (slope variance = .002, $p = .07$) over the course of elementary school. The five yearly scores were therefore averaged to represent a child's overall level and chronicity of peer victimization experiences across elementary school to be used in the analyses (MZ mean = .34, *SD* = .29, minimum = 0.00, maximum = 1.13; DZ mean = .44, *SD* = .31, minimum = 0.00, maximum = 1.26).

Procedure

Active written consent from the child's parents as well as verbal assent of all children was obtained for each wave of data collection. Starting in kindergarten (age 6 years), data collections took place in the spring to ensure that the teachers knew the children well. All instruments and study procedures were approved by the Ethics Committee of the Ste- Justine Hospital Research Center and the school administrators.

Analytical Rationale

Univariate Genetic Models

The twin design makes it possible to assess the relative role of latent (i.e., unmeasured) genetic factors and latent environmental factors associated with a measured phenotype (i.e., behavior or other variable measured for each twin separately) (Neale & Cardon, 1992). Specifically, by comparing within-pair correlations for MZ twins, who share 100% of their genes, and same-sex DZ twins, who share ~50% of their genes, sources of variability of a phenotype can be estimated in terms of additive genetic factors (A), shared environmental factors (C), which affect twins within a pair in a similar way (e.g., family socioeconomic status, parental education, school or neighborhood characteristics), and nonshared environmental factors (E), which uniquely affect each twin in a pair (e.g., differential treatment by parents, exposure to different peers or teachers). Any measurement error is also captured in the E variance component. The relative effect of additive genetic factors is approximately twice the MZ and same-sex DZ correlation difference. For instance, if the MZ intra-pair correlation is $r = .80$ and the DZ intra-pair correlation is $r = .50$, the percent of variance of the measured phenotype explained by genetic factors can be estimated as 2 (.80–.50) = .60 (or 60%). The relative effect of shared environmental factors can be estimated by subtracting the MZ correlation from twice the DZ correlation, that is, (2*.50) − .80 = .20 (or 20%) in our example. Nonshared environmental effects are approximated by the extent to which the MZ correlation is <1, that is, 1 − .80 = .20 (or 20%) in our example.

Structural equation modeling (SEM) using a maximum likelihood fit function enables a more precise estimation of the genetic and environmental parameters that also includes CIs and hence the statistical significance of the estimated parameters (Neale & Cardon, 1992). To this end, a two-group model is fitted to the data where (1) the latent genetic correlations between the two twins in a pair are fixed to 1.0 for MZ twins and to 0.5 for DZ twins, (2) the latent shared environmental correlations between

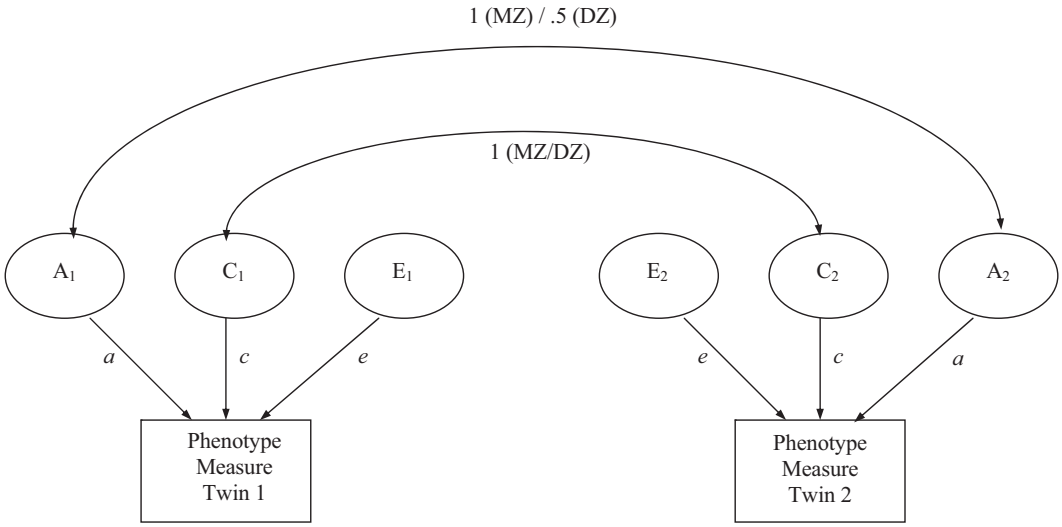

Figure 1. Univariate ACE model. A = additive genetic factors; C = shared environmental factors, which affect twins within a pair in a similar way, E = nonshared environmental factors, which uniquely affect each twin in a pair. Parameters a, c, and e denote effects of the respective factor on the measured variable.

twins in a pair are fixed to 1.0 for both MZ twins and DZ twins, and (3) the nonshared environmental correlations between twins in a pair are fixed to zero for both MZ twins and DZ twins (see Figure 1). The estimated coefficients a, c, and e, which are fixed to be equal across the two twins in a pair and across MZ and DZ twins, provide information about the relative contribution of the latent factors A, C, and E to the total variance of each phenotype P, with the variance of $P = a^2 + c^2 + e^2$.

Using the Mplus software package, such a univariate model was fitted to the data separately for each of the three study variables. Model fit was assessed based on the χ^2-statistic, the Akaike information criterion (AIC), the Bayesian information criterion (BIC), the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). Low and nonsignificant χ^2 values, lower values of AIC and BIC, CFI values of $\geq .9$, and values of RMSEA $< .08$ indicate good model fit and parsimony. Using nested χ^2-difference tests, the full ACE model depicted in Figure 1 (i.e., which estimated all three parameters a, c, and e) was compared with a series of more parsimonious submodels: (1) an AE model, where the effect of C was fixed to zero, (2) a CE model, where the effect of A was fixed to zero and (3) an E model, where the effects of both A and E were fixed to zero. Models without the E effect, which also contains measurement error, cannot be estimated. Selection of the best fitting model was based on the previously mentioned model fit criteria, on the significance and estimated values of the a, c, and e parameters, as well as on the significance of the nested χ^2-difference tests of the submodels.

Multivariate Genetic Models

Using the results from the univariate models of variance decomposition as a basis, multivariate models can be specified to test sources of covariation between two or more measured variables, such as between physical health problems in early childhood and in early adolescence and peer victimization during elementary school. If one of the measured variables is a putative environmental variable, such as peer victimization, rGE and G × E can also be tested. To this end, a multivariate Cholesky model (see Figure 2) was specified where the covariance structure of peer victimization and physical health problems in early childhood and in early adolescence was partitioned into (1) a ''common'' genetic factor A_{HC} and a ''common'' nonshared environmental factor E_{HC} that not only influence physical health problems in early childhood (denoted by the subscript $_{HC}$) but also peer victimization in middle childhood and physical health problems in early adolescence, (2) a ''common'' genetic factor A_V and a ''common'' nonshared environmental factor E_V that influence peer victimization in middle childhood (denoted by the subscript $_V$) as well as physical health problems in early adolescence, (3) a ''unique'' genetic factor A_{HA} and a ''unique'' nonshared environmental factor E_{HA} that are specific to physical health problems in early adolescence (denoted by the subscript $_{HA}$), and (4) a ''unique'' shared environmental factor C_V specific to peer victimization. Because the results from the univariate models showed no shared environmental influences (C) for physical health problems either in early childhood or in early adolescence (see below), shared

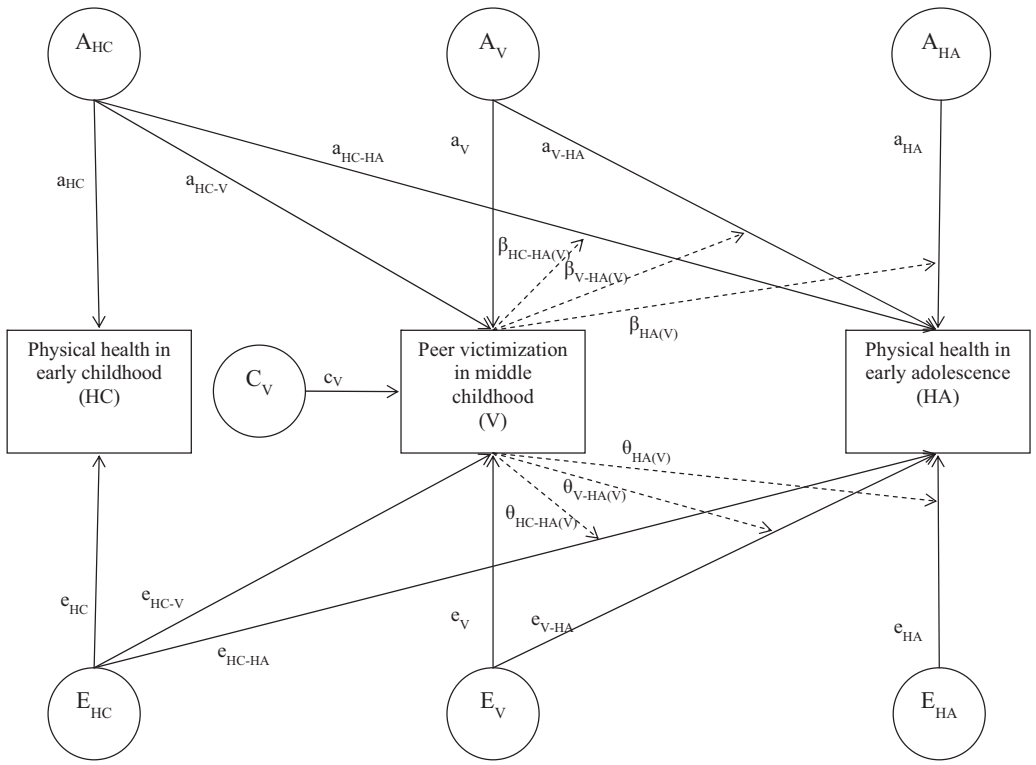

Figure 2. Multivariate Cholesky model of physical health problems in early childhood, peer victimization in middle childhood, and physical health problems in early adolescence. To reduce model complexity, the model is only depicted for one member of a twin pair. The covariance structure of peer victimization and physical health problems in early childhood and in early adolescence is partitioned into (1) a "common" genetic factor A_{HC} and a "common" nonshared environmental factor E_{HC} that not only influence early childhood physical health problems (denoted by the subscript $_{HC}$) but also peer victimization in middle childhood and physical health problems in early adolescence, (2) a "common" genetic factor A_V and a "common" nonshared environmental factor E_V that influence peer victimization in middle childhood (denoted by the subscript $_V$) as well as physical health problems in early adolescence, (3) a "unique" genetic factor A_{HA} and a "unique" nonshared environmental factor E_{HA} that are specific to physical health problems in early adolescence (denoted by the subscript $_{HA}$), and (4) a "unique" shared environmental factor C_V specific to peer victimization. Because the results from the univariate models showed no shared environmental influences (C) for physical health problems either in early childhood or in early adolescence (see text), shared environmental influences were not specified for the two physical health variables in the multivariate Cholesky model. Please see text for an explanation of the parameter acronyms that represent the effects of the latent factors on the three measured study variables.

environmental influences were not specified for the two physical health variables in the multivariate Cholesky model.

Coefficients a_{HC} and e_{HC} indicate the effect of genetic and nonshared environmental factors on early childhood physical health problems. Coefficients $a_{HC\text{-}HA}$ and $e_{HC\text{-}HA}$ indicate to what extent genetic or nonshared environmental factors that influence physical health problems in early childhood also contribute to such problems in early adolescence. These coefficients provide a control for the stability of physical health problems over time while at the same time indicating the source (i.e., genetic or environmental) of the stability. Coefficients $a_{HC\text{-}V}$ and $e_{HC\text{-}V}$ indicate to what extent genetic or nonshared environmental factors associated with early childhood physical health problems also influence peer victimization in elementary school, with a significant coefficient $a_{HC\text{-}V}$ reflecting rGE (Study Objective 1). Coefficients $a_{V,}$ c_V, and e_V indicate to what extent peer victimization is affected by genetic, shared environmental, and nonshared environmental factors that are not associated with physical health problems during early childhood. Coefficients $a_{V\text{-}HA}$ and $e_{V\text{-}HA}$ indicate to what extent genetic or nonshared environmental factors associated with peer victimization are also associated with newly emerging physical health problems assessed in early adolescence. A significant coefficient $a_{V\text{-}HA}$ thus also reflects a significant rGE (Study Objective 1). Finally, coefficients a_{HA} and e_{HA} indicate to what extent early adolescent physical health problems are affected by genetic and nonshared environmental factors that are not associated with physical health problems during early childhood or with peer victimization. These latter coefficients need to be included to avoid biased estimates of rGE.

To test G × E in the prediction of early adolescent physical health problems (Study Objective 2), we included several interaction terms: (1) an interaction term between peer victimization and the ''common'' genetic factor A_{HC}, represented by the term $\beta_{HC\text{-}HA(V)}$, (2) an interaction term between peer victimization and the ''common'' genetic factor A_V, represented by the term $\beta_{V\text{-}HA(V)}$, and (3) an interaction term between peer victimization and the ''unique'' genetic factor A_{HA}, represented by the term $\beta_{HA(V)}$. If any of these three interaction terms is significant, this would indicate that genetic effects on physical health problems in early adolescence vary depending on the level of peer victimization, that is, indicating G × E. However, as noted by Purcell (2002), it is important to ascertain whether any observed moderating effect of peer victimization is truly specific to the latent genetic effects on physical health problems in early adolescence. To avoid potentially biased estimates of G × E, we therefore also added (4) an interaction term between peer victimization and the nonshared environmental factor E_{HC}, represented by the term $\theta_{HC\text{-}HA(V)}$, (5) an interaction term between peer victimization and the nonshared environmental factor E_V, represented by the term $\theta_{V\text{-}HA(V)}$, and (6) an interaction term between peer victimization and the nonshared environmental factor E_{HA}, represented by the term $\theta_{HA(V)}$. These three interaction terms tested whether peer victimization moderated the nonshared environmental effects on physical health problems in early adolescence (i.e., environment–environment interaction).

Of note, χ^2, RMSEA, and comparative fit indices are not available for a multivariate model that includes interaction terms. To test whether the inclusion of interaction terms significantly improves model fit, the Cholesky model with interaction terms depicted in Figure 2 was compared with a Cholesky model without interaction terms using the Likelihood Ratio test (i.e., based on the 2LogLikelihood difference between the two models), which is equivalent to a nested χ^2-difference test (Purcell, 2002).

Results

Preliminary Analyses

SEM-based means structure analyses revealed that boys were more victimized by peers than girls, $\chi^2(1) = 16.7$, $p = .00$. However, girls and boys did not differ in regard to physical health problems in early childhood, $\chi^2(1) = 1.48$, $p = .22$, or in early adolescence, $\chi^2(1) = 1.91$, $p = .17$. Moreover, there was no significant difference between boys and girls with respect to the variance-covariance structure of the study variables, $\chi^2(12) = 19.6$, $p = .08$ for MZ pairs and $\chi^2(12) = 18.8$, $p = .09$ for DZ pairs. Data were therefore pooled combining male and female MZ pairs and combining male and female DZ pairs, respectively, to maximize statistical power. To account for the sex-related mean difference in peer victimization, variables were standardized within sex groups before data pooling. No significant mean differences in the study variables were found between MZ pairs and DZ pairs, $\chi^2(3) = 1.24$, $p = .74$. Bivariate correlations revealed that physical health problems in early childhood and physical health problems in early adolescence were significantly correlated, $r = .13$, $p = .00$, suggesting some continuity of physical health problems over time. For the tests of potential rGE (but not for tests of potential G × E), it was also important that physical health problems are correlated with peer victimization. No significant bivariate correlation emerged between physical health problems in early childhood and peer victimization, $r = .06$, $p = .18$. However, physical health problems in early adolescence and peer victimization were significantly correlated, $r = .21$, $p = .00$, which opened the possibility that the subsequent multivariate model tests (described below) may reveal a significant rGE in the link of these two variables. All these correlations were the same even when controlling for family income.

Within-twin pair correlations were considerably higher in MZ than DZ pairs for physical health problems in early childhood (MZ $r = .75$; DZ $r = .45$) and in early adolescence (MZ $r = .40$; DZ $r = .18$) as well as for peer victimization (MZ $r = .51$; DZ $r = .35$). This suggests significant genetic effects on all study variables but small, if any, shared environmental effects. However, the fact that MZ within-pair correlations were well below 1.0 suggests sizable nonshared environmental effects.

Univariate Models

As can be seen in Table I, in the best fitting univariate model (AE) for physical health problems in early childhood, genetic factors accounted for 75% of the variance, whereas the remaining 25% were explained by nonshared environmental sources. In the best fitting model (AE) for physical health problems in early adolescence, genetic factors accounted for 41% of the variance and the remaining 59% were explained by nonshared environmental sources. In the best fitting model (ACE) for peer victimization, genetic factors explained 32% and nonshared environmental factors explained 49% of the variance. Shared environment explained another 19%, but did not reach statistical significance.

Table I. *Univariate Model Results*

Model	%a^2	%c^2	%e^2	AIC	BIC	CFI	RMSEA	χ^2 (*df*)	*p*
Physical health problems in early childhood									
ACE	75.2 (61.0, 84.0)	.0 (.0, .0)	24.8 (15.4, 38.1)	1,464.4	1,479.0	.97	.07	1.12 (2)	.57
AE	**75.2 (61.9, 84.5)**	**–**	**24.8 (15.5, 38.1)**	**1,462.4**	**1,473.4**	**.98**	**.06**	**1.12 (3)**	**.77**
CE	–	58.7 (41.1, 73.1)	41.3 (26.8, 58.9)	1,497.3	1,508.3	.76	.20	36.0 (3)	.00
E	–	–	100.0 (–, –)	1,616.1	1,623.4	.00	.37	156.8 (4)	.00
Physical health problems in early adolescence									
ACE	41.2 (23.1, 56.7)	.0 (.0, .0)	58.8 (43.1, 74.2)	1,595.4	1,612.0	1.00	.00	1.63 (2)	.44
AE	**41.2 (26.0, 56.7)**	**–**	**58.8 (45.3, 73.9)**	**1,593.4**	**1,604.4**	**1.00**	**.00**	**1.63 (3)**	**.65**
CE	–	30.3 (18.8, 43.0)	69.7 (58.9, 83.1)	1,599.3	1,610.3	.91	.05	7.51 (3)	.06
E	–	–	100.0 (–, –)	1,624.8	1,632.1	.08	.16	35.0 (4)	.00
Peer victimization									
ACE	**31.9 (.1, 59.3)**	**18.6 (.0, 46.7)**	**49.5 (37.2, 61.6)**	**1,564.8**	**1,579.4**	**1.00**	**.00**	**.02 (2)**	**.99**
AE	51.9 (40.5, 63.2)	–	48.1 (36.8, 59.5)	1,564.0	1,575.0	1.00	.00	1.22 (3)	.75
CE	–	43.9 (32.4, 53.2)	56.1 (46.7, 67.6)	1,566.0	1,576.9	.99	.02	3.20 (3)	.36
E	–	–	100.0 (–, –)	1,625.1	1,632.4	.05	.23	64.3 (4)	.00

Note. ACE = full ACE model where the A, C, and E effects are estimated; AE = submodel where A and E effects are estimated but the C effect is fixed to zero; CE = submodel where the C and E effects are estimated but the A effect is fixed to zero; E = submodel where the E effect is estimated (which also contains measurement error) but both A and C effects are fixed to zero; %a^2 = percent of variance explained by genetic influences; %c^2 = percent of variance explained by shared environmental influences; %e^2 = percent of variance explained by nonshared environmental influences; AIC = Aikaike information criterion; BIC = Bayesian information criterion; CFI = comparative fit index; RMSEA = root means square error of approximation; *df* = degrees of freedom.
Best fitting models are in bold.

Table II. *Multivariate Cholesky Model Results*

Parameter	Estimate	LL	Number of parameters	AIC	BIC
a_{HC}	.86 (.68, 1.04)	−2,270.5	22	4,585.0	4,665.4
$a_{HC\text{-}V}$	.07 (−.06, .19)				
$a_{HC\text{-}HA}$	.19 (.08, .29)				
$\beta_{HC\text{-}HA(V)}$	−.04 (−.17, .09)				
a_V	.58 (.21, .96)				
$a_{V\text{-}HA}$	.21 (.01, .43)				
$\beta_{V\text{-}HA(V)}$	.09 (−.29, .47)				
a_{HA}	.56 (.37, .74)				
$\beta_{HA(V)}$	.03 (−.18, .23)				
c_V	.40 (−.08, .88)				
e_{HC}	.50 (.41, .58)				
$e_{HC\text{-}V}$	−.01 (−.12, .10)				
$e_{HC\text{-}HA}$	−.05 (−.19, .09)				
$\theta_{HC\text{-}HA(V)}$	.02 (−.16, .20)				
e_V	.70 (.61, .79)				
$e_{V\text{-}HA}$	.08 (−.04, .20)				
$\theta_{V\text{-}HA(V)}$	−.02 (−.28, .23)				
e_{HA}	.75 (.65, .85)				
$\theta_{HA(V)}$	.12 (.01, .23)				

Note. AIC = Aikaike information criterion; BIC = Bayesian information criterion; LL = Log-likelihood.
χ^2-based fit statistics, including root mean square error of approximation, are not available for models that include interaction terms.
Confidence intervals are in parentheses.
Please see text for explanation of the parameter acronyms.

Main Analyses: Multivariate Model

Inclusion of interaction terms in the multivariate Cholesky model significantly improved model fit compared with a multivariate model without interaction terms, $2\Delta LL(6) = 27.8$, $p = .00$. The results from the multivariate model with interaction terms are presented in Table II. As can be seen, genetic factors contributing to physical health problems in early childhood also explained a small but significant portion of physical health problems in early adolescence [$a_{HC\text{-}HA} = .19$; CI = .08–.29]. Nevertheless, most genetic influences on early adolescent health problems seemed to appear after the early childhood period. Moreover, a significant portion of the newly emerging genetic influence on physical health problems in early adolescence was also associated with peer victimization ($a_{V\text{-}HA} = .21$; CI = .01–.43), indicating a significant rGE = .38 (CI = .07–.70). In contrast, there was no significant overlap (i.e., no rGE) between the genetic factors related to physical health problems during early childhood and peer victimization in primary school. Contrary to our expectations of G × E, there was no significant interaction of peer victimization with any of the latent genetic factors predicting physical health problems in early adolescence. However, there was a significant interaction of peer victimization with the latent nonshared environmental factor E_{HA} predicting physical health problems in early adolescence

($\theta_{HA(V)} = .12$, CI = .01–.23). This finding indicates that environmental influences on physical health problems in early adolescence vary significantly depending on the child's level of peer victimization. This interaction is illustrated in Figure 3. As can be seen, the total variance of physical health problems in early adolescence was higher in children who were highly victimized during middle childhood. This increase in variance is mostly due to the fact that the influence of environmental factors—relative to genetic factors—on physical health problems was much stronger in highly victimized children.

Discussion

This study examined potential gene–environment processes underlying the association between peer victimization and physical health problems. Similar to other studies (Holloway et al., 2010; Ligthart et al., 2010; Thomsen et al., 2010), univariate analyses showed that genetic effects account for a significant proportion of the variance of physical health problems both in early childhood and in early adolescence, with the remaining variance mostly explained by nonshared environmental factors. Despite the use of different informants (i.e., mothers during early childhood and youths' self-reports during early adolescence), physical health problems showed some, albeit modest, stability over the course of development, which was entirely explained by genetic factors that were already present in early childhood. Moreover, multivariate analyses revealed evidence of rGE and of environmental interaction in the link between peer victimization and physical health problems.

Gene–Environment Correlation in the Link of Peer Victimization and Physical Health Problems

In line with rGE, genetic factors accounted for more than half of the interindividual variance in chronic peer victimization during middle childhood. This finding corroborates results from other studies that certain personal, partly heritable characteristics may partly explain why some children may be targeted by bullies (Ball et al., 2008; Brendgen et al., 2011). Our results also showed that genetic factors specifically related to physical health problems observed in early adolescence (i.e., susceptibility to health problems that occur *after* early childhood) were associated with peer victimization in elementary school. Many physical health symptoms such as digestive tract problems and nose–throat infections are fairly common in early childhood. Even children who are highly susceptible to poor health during the toddler and preschool years may therefore not necessarily be seen as weak and easy targets once they enter school. This situation may be different for children with poor health at later ages. Physical health

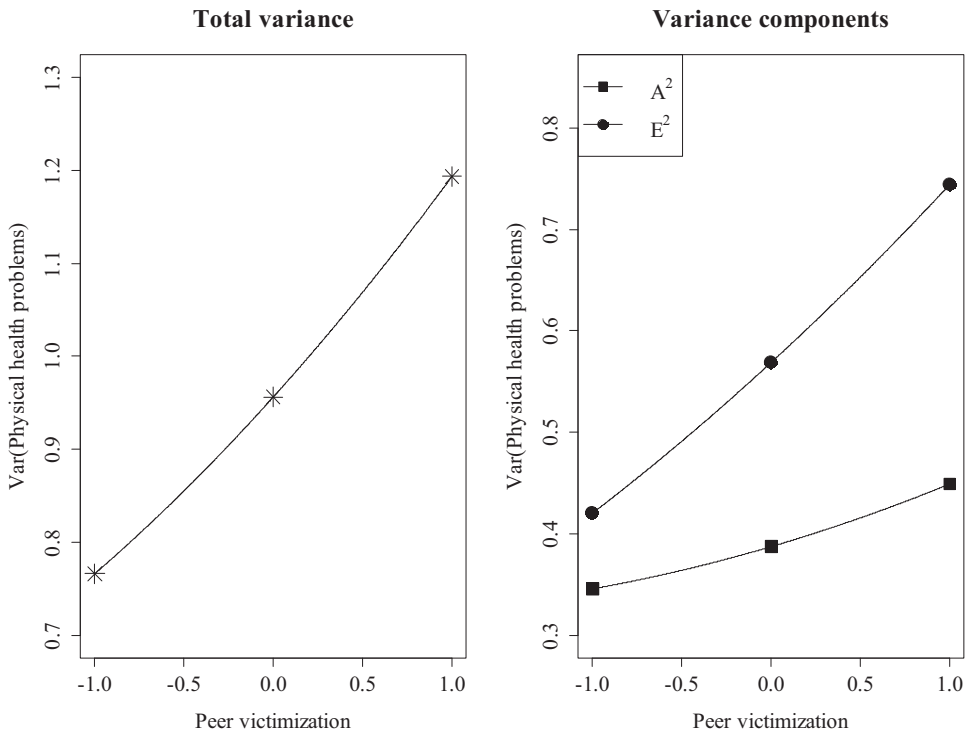

Figure 3. Plot of total variance as well as additive genetic (A^2) and nonshared environmental (E^2) variance components of physical health problems as a function of peer victimization.

problems often lead to low energy and withdrawal from social interactions with others, which can put especially older children at risk of becoming victimized by peers (Boivin, Hymel, & Hodges, 2001). It is also worth noting that physical health problems assessed in early childhood included chronic illnesses or infectious diseases, whereas physical health problems assessed in early adolescence included primarily somatic complaints and illness symptoms. Although more severely handicapped children did not participate in our study, these conceptual differences may also explain, at least to some extent, the lack of association between physical health problems in early childhood and later peer victimization. However, children with chronic or congenital physical health conditions have been found to be more often victimized than others in cross-sectional research (Sentenac et al., 2012). Thus, to the extent that the early childhood physical health measure represented more serious conditions than the later physical health measure, one should have expected a stronger association with peer victimization for the former than for the latter. Still, the overall extent of rGE linking peer victimization and physical health problems observed in early adolescence was relatively modest, indicating that other genetically influenced characteristics may also explain interindividual differences in peer victimization. Several studies have shown that a considerable part of genetic influence on peer victimization is mediated by a disposition for aggressive behavior (Ball et al., 2008; Brendgen et al., 2008, 2011).

Environment–Environment Interaction Linking Peer Victimization and Physical Health Problems

In contrast to the significant rGE, no significant G × E was observed. However, our results revealed a significant environment–environment interaction because peer victimization significantly moderated the effect of nonshared environmental factors on physical health problems in early adolescence. Specifically, the relative influence of nonshared environmental features on physical health problems in early adolescence was increased—and hence the relative influence of genetic factors decreased—when children were highly victimized by their peers during elementary school. Interestingly, this pattern resembles a suppression process of genetic influences. The stress of frequent harassment by peers may lead to somatic symptoms in many children and perhaps even weaken immune functioning in some. Whether victimized children develop physical symptoms may therefore not so much depend on their genetic susceptibility for physical health problems but rather on environmental factors. An environmental factor that may be especially important in this regard is the availability of support from their social environment—or lack thereof. Support from parents, teachers, or friends has been found to protect victimized youth against internalizing and externalizing problems (Malcolm, Jensen-Campbell, Rex-Lear, & Waldrip, 2006; Yeung & Leadbeater, 2010). Similar buffering effects may occur against the development of physical symptoms in victims.

Strengths, Limitations, and Conclusions

This study is the first to assess gene–environment processes in the link between peer victimization and physical health problems. The use of a longitudinal design spanning early childhood through early adolescence allowed us to examine during which developmental period a genetic vulnerability for physical health problems may put children at risk of being victimized by peers. The measurement of peer victimization over multiple time points during elementary school made it possible to investigate the link between chronic victimization and poor health. The use of distinct raters for the different study variables helped reduce shared source variance. Our study also has several limitations. First, physical health problems were measured based on subjective accounts of mothers or the youngsters themselves. The reliance on questionnaire-based self-reports is common practice in studies on peer victimization and physical health problems (Gini & Pozzoli, 2009), and it has been argued that the person's own voice is needed when the research focus is on well-being and symptoms (Riley, 2004). Parents' reports of use of health services by their children are relatively accurate (Riley, 2004) and both mother and child reports of the child's physical symptoms predict the child's subsequent health care use (Forrest, Riley, Vivier, Gordon, & Starfield, 2004). Second, given the normative nature of our sample, both physical health problems and peer victimization occurred rather infrequently. Because more severely handicapped children did not participate in our longitudinal study, the findings may not generalize to children with serious genetically induced physical impediments that are present in early childhood. Third, future studies should reexamine potential sex moderation. Girls and boys were examined together because no sex differences in the covariances of the study variables were found, allowing us to maximize statistical power. Moreover, although some studies observed somewhat stronger links between peer victimization and physical health problems for girls (Biebl, Dilalla, Davis, Lynch, & Shinn, 2011; Knack et al., 2011; Rigby, 1998), others did not (Fekkes et al., 2006; Nishina et al., 2005; Nixon, Linkie, Coleman, & Fitch, 2011; Vernberg et al., 2011). Finally, the limited number of items did not allow us to differentiate between physical victimization, such as hitting

and kicking, and relational victimization, such as public humiliation. However, the two forms of victimization often co-occur and both have been associated with physical health complaints in youth (Nixon et al., 2011).

These limitations notwithstanding, our study demonstrates that genetically informed research can provide important insights into the sometimes complex processes that link genetic and environmental risk factors with impaired physical health. The findings suggest that genetic vulnerability for physical health problems beyond the early childhood phase increases a child's risk of being victimized by peers. However, whether victimized children develop physical symptoms seems to depend not so much on genetic vulnerability but on the presence of environmental factors that can protect against the deleterious health consequences of peer victimization. The results emphasize the importance of involving pediatricians as well as other health care practitioners in the early identification and treatment of victims of peer aggression, as advocated by the American Academy of Pediatrics (Committee on Injury, Violence, & Prevention, Poison, 2009). However, many pediatricians do not seem to routinely ask questions about problematic peer experiences (Ozer et al., 2009). Appropriate training has been found to increase pediatric clinicians' self-efficacy to screen and counsel youth in regard to risky behaviors such as unprotected sex and drug use (Buckelew, Adams, Irwin, Gee, & Ozer, 2008) and a similar training effect may be observed in regard to screening for peer victimization. Treatment plans may then need to include interventions that also address peer victimization rather than just the associated health problems. For example, recent evidence provides support for the efficacy of brief computer-assisted therapist interventions with at-risk youth during emergency room visits for reducing self-reported peer victimization (Cunningham et al., 2012). As a consequence, such interventions may also be effective in decreasing victims' physical symptoms.

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