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Gene-Environment Interplay Between Peer Rejection and Depressive Behavior in Children

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Abstract

1. Background. Genetic risk for depressive behavior may increase the likelihood of exposure to environmental stressors (gene–environment correlation, rGE). By the same token, exposure to environmental stressors may moderate the effect of genes on depressive behavior (gene–environment interaction, GxE). Relating these processes to a peer-related stressor in childhood, the present study examined (1) whether genetic risk for depressive behavior in children is related to higher levels of rejection by the peer group (rGE) and (2) whether peer rejection moderates the effect of genetic factors on children's depressive behavior (GxE). **3. Methods.** The sample comprised 336 twin pairs (MZ pairs = 196, same-sex DZ pairs = 140) assessed in kindergarten (mean age 72.7 months). Peer acceptance/rejection was measured via peer nominations. Depressive behavior was measured through teacher ratings. **4**. **Results.** Consistent with rGE, a moderate overlap of genetic effects was found between peer rejection and depressive behavior. In line with GxE, genetic effects on depressive behavior varied across levels of peer acceptance/rejection. **5. Conclusions.** An increased genetic disposition for depressive behavior is related to a higher risk of peer rejection (rGE). However, genes play a lesser role in explaining individual differences in depressive behavior in rejected children than in accepted children (GxE). **6. Keywords**. Gene–environment interaction, gene–environment correlation, depressive behavior, peer rejection, childhood. **7. Abbreviations**. MZ: Monozygotic; DZ: Dizygotic; rGE: gene–environment correlation; GxE: gene–environment interaction; -2LL: -2LogLikelihood; RMSEA: root mean square error approximation.

Gene-Environment Interplay Between Peer Rejection and Depressive Behavior in Children

Between 10 and 20% of school-aged children show symptoms of depressive behavior (Achenbach, 1991) and 1 to 3% suffer from clinical levels of depression (Anderson, Williams, McGee, & Silva, 1987). Evidence from genetically informed quantitative research such as twin studies suggests that depressive symptoms in children are at least in part explained by genetic factors, with the remaining variance mostly explained by environmental factors unique to each child (Happonen et al., 2002; Rice, Harold, & Thapar, 2002). However, estimations of simple effects ignore possible gene-environment correlations or gene-environment interactions in the etiology of psychopathological behavior (Rutter & Silberg, 2002). A gene–environment correlation (rGE) may be caused by three processes (Rutter, Moffitt, & Caspi, 2006). *Passive* rGE arises when parents' genes influence the social environment they provide. *Active* rGE occurs when individuals shape or select their environment based on their own genetic dispositions. *Evocative* rGE refers to a process whereby individuals' genetic dispositions to a behavior evoke specific reactions from the environment. A gene–environment interaction (GxE) with respect to psychopathological behavior may arise through three generic mechanisms (Shanahan & Hofer, 2005). A *diathesis-stress* process occurs when an environmental stressor triggers or exacerbates genetic predisposition for a specific trait or phenotype. In a *compensation* process, the presence of a positive environment prevents or reduces the expression of a genetic disposition. A *control* or suppression process arises when restrictive environmental conditions reduce the role of genetic influences such that many individuals exhibit the same phenotype, irrespective of their genetic disposition.

Based mostly on older children and adolescent samples, genetically informed quantitative studies provide evidence for both rGE and GxE with respect to depressive symptoms in youth. Thus, in line with rGE, features of the social environment such as negative life events or problematic parent-child relationships have been found to be influenced by genetic factors that also contribute to depressive symptoms (Lau, Rijsdijk, & Eley, 2006; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996; Rice, Harold, & Thapar, 2003; Silberg et al., 1999). In support of GxE, negative life events have been found to moderate the contribution of genetic factors to depressive symptoms among adolescents (Eaves, Silberg, & Erkanli, 2003; Silberg, Rutter, Neale, & Eaves, 2001). These latter findings also concord with molecular studies showing that environmental variables such as a history of maltreatment or family adversity moderate the effect of specific measured genes on depressive symptoms in children and adolescents (Eley et al., 2004; Kaufman et al., 2006).

Although gene-environment correlation and gene-environment interaction have often been investigated independently, the two processes can co-occur. Indeed, the same environmental risk factor may be involved in both rGE and GxE on depressive symptoms in youth. Estimating both rGE and GxE simultaneously, Eaves and colleagues (2003) showed that genetic vulnerability to depressive symptoms increased adolescent girls' risk of experiencing negative life events (rGE). By the same token, exposure to negative life events exacerbated the effect of genes on depressive symptoms (GxE). Similar results for the link between negative life events and adolescent boys' and girls' depressive symptoms were reported in a study by Lau and Eley (2008). These authors also found that genetic factors influencing depressive symptoms were correlated with another environmental risk factor — maternal punitive

discipline —, indicating rGE. Additionally, in line GxE, maternal punitive discipline augmented genetic influences on depressive symptoms.

Peer Rejection: Gene-Environment Linkages with Depressive Behavior Symptoms?

Negative experiences with peers – especially rejection from the peer group – may also play an important role in the development of depressive behavior symptoms in children. Peer rejection often entails overt expressions of dislike including physical and nonphysical forms of harassment (McDougall, Hymel, Vaillancourt, & Mercer, 2001), which has been related to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis system (Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003). Dysregulation of the HPA axis, in turn, can disrupt the biosynthesis of the serotonin system, which is considered a key factor in the development of depression (Porter, Gallagher, Watson, & Young, 2004). Accordingly, a large body of research has linked peer rejection to both concurrent and subsequent depressive behavior symptoms in children (McDougall et al., 2001). Despite its importance, however, peer rejection has not yet been examined in the context of gene-environment interplay on depressive behavior symptoms.

Findings from single-birth studies suggest that peer rejection might be implicated in both rGE and GxE with respect to depressive behavior symptoms. In support of a possible rGE, depressed children's social behavior has been found to elicit rejection from peers (Baker, Milich, & Manolis, 1996; Rudolph, Hammen, & Burge, 1994). Evidence also hints at a possible GxE. Specifically, peer rejection has been related to an escalating trajectory of depressive symptoms during early adolescence, but only for youth with a highly reactive temperament (Brendgen, Wanner, Morin, & Vitaro, 2005). Temperamental features, including reactivity, are partly heritable (Kagan, 1997) and may reflect genetic vulnerability to stress-related depressive symptoms. These findings suggest that, in line with a diathesis-stress mechanism of GxE, peer

rejection may exacerbate genetically mediated depressive behavior in youth. The present study aimed to test rGE and GxE in the link between peer rejection and children's depressive behavior symptoms in a sample of six-year old twins.

Method

Sample

The 336 twin pairs (MZ males = 93, MZ females = 103, DZ males = 72, DZ females = 68) participating in this study were part of a population-based sample of 448 twin pairs from Montreal, Canada, who were recruited at birth between November 1995 and July 1998. Zygosity was assessed at 18 months based on physical resemblance via the Zygosity Questionnaire for Young Twins (Goldsmith, 1991). A DNA sample was evaluated with respect to 8-10 highly polymorphous genetic markers. The comparison of zygosity based on the similarity of these genetic markers with zygosity based on physical resemblance revealed a 94% correspondence rate, which is similar to rates obtained in older twin samples (Forget-Dubois et al., 2003). Eighty-seven percent of the families were of European descent, 3% were of African descent, 3% were of Asian descent, and 1% were Native North Americans. The remaining families did not provide ethnicity information.

The demographic characteristics of the twin families were comparable to those of a sample of single births representative of the urban centers in the province of Quebec (SantéQuébec, Jetté, Desrosiers, & Tremblay, 1998). At the time of their child(ren)'s birth, 95% of parents lived together; 44% of the twins were the first born children; 66% of mothers and 60% of fathers were between 25 and 34 years old; 17% of mothers and 14% of fathers had not finished high school; 28% of mothers and 27% of fathers held a university degree; 83% of the parents held an

employment; 10% of the families received social welfare or unemployment insurance; 30% of the families had an annual income of less than $30,000.

The sample was followed at 5, 18, 30, 48, and 60 months focusing on a variety of child-related and family-related characteristics. Another data collection was completed at age six years (*mean* = 72.7 months, *SD* = 3.6) to assess social adaptation in kindergarten. These data were used for the present paper. Of the 448 pairs in the initial sample, 25% were lost over the years, which resulted in the final N of 336 twin pairs for the age 6 data collection. Twins remaining in the study at 6 years of age did not differ from those lost since the first wave of data collection in regard to zygosity, parenting behavior, parent-rated child temperament at 5 months of age, or any socio-demographic measures. However, fathers in the remaining sample had a slightly higher level of education.

Measures

Peer acceptance/rejection was assessed via sociometric procedures with the twins' classmates. Booklets of photographs of all children in a class were handed out to each child in the class. The children were asked to circle the photos of three classmates they most liked to play with (positive nominations) and of three children they least liked to play with (negative nominations). Following widely used criteria for assessing peer acceptance and rejection (Coie, Dodge, & Coppotelli, 1982), the total number of received positive nominations was calculated for each participant and z-standardized within classroom to create a total Liked-Most-score. Similarly, the total number of received negative nominations was calculated for each participant and z-standardized within classroom to create a total Liked-Least-score. The Liked-Most-score was then subtracted from the Liked-Least-score to create a uni-dimensional, continuous scale of Peer Acceptance versus Rejection, which was again z-standardized within classroom. High levels

on this scale indicate acceptance whereas low levels indicate rejection. The nomination-based peer acceptance/rejection score is moderately to highly stable over a one-year period, is highly correlated with rating-based measures of peer rejection and popularity, and predicts a vast array of adjustment problems, including depression symptoms, delinquency, and school dropout (McDougall et al., 2001).

Depressive behavior was rated by teachers. Teacher-rated depressive behavior correlates reasonably well (r = .49) with child self-reported depressive symptoms if the teacher is familiar with the child (Ines & Sacco, 1992). Four items of the Emotional Disorder Scale of the Ontario Child Health Study (Offord, Boyle, & Racine, 1989) were used: 'Seems unhappy or sad', 'Is not as happy as other children', 'Lacks energy, seems tired', 'Has difficulties enjoying him-or herself'. Ratings for each item ranged from 0 'does not apply', and 1 'applies sometimes' to 2 'applies often'. For each child, individual item scores were added to compute scale scores. Internal consistency was somewhat low but acceptable (Nunally & Bernstein, 1994), Cronbach's alpha = 0.74, *mean* = 2.00, *SD* = 1.73.

Procedure

Active written consent from the parents of all children in the classroom was obtained. Data collection took place in the spring to ensure that the children and the teachers knew each other. The sociometric procedure took 45 minutes. At the same time, teachers completed the behavior questionnaires in a separate room. The instruments were approved by the Institutional Review Board and the school board administrators.

Results

Preliminary Analyses

Differences in peer rejection and depressive behavior between boys and girls and between MZ and DZ twins were examined with nested χ^2-difference tests. Boys were more rejected by their peers, $\chi^2(1) = 7.66$, $p = .01$, whereas girls showed more depressive behavior symptoms, $\chi^2(1) = 6.02$, $p = .01$. However, a comparison of the multivariate correlation matrices revealed no difference between boys and girls ($\chi^2(6) = 8.32$, $p = .22$, for MZ twins; $\chi^2(6) = 8.94$, $p = .18$, for DZ twins). Data were therefore pooled across male and female MZ pairs and across male and female DZ pairs to maximize statistical power (for a similar approach, see Arseneault et al., 2003; Van den Oord, Boomsma, & Verhulst, 2000). To control for the mean differences between boys and girls, variable scores were z-standardized within gender prior to the pooling of the data. Supporting previous findings (Pulkkinen, Vaalamo, Hietala, Kaprio, & Rose, 2003), further analyses revealed that MZ twins were more popular among peers than DZ twins, $\chi^2(1) = 18.04$, $p = .00$, but did not differ with respect to depressive behavior, $\chi^2(1) = 0.05$, $p = .82$. The mean difference in peer acceptance/rejection between MZ and DZ twins was modeled in the genetic analyses. Table 1 depicts the within-pair correlations as well as the means and standard deviations of peer acceptance/rejection and depressive behavior. The phenotypic correlation between peer acceptance/rejection and depressive behavior was $r = -.23$ (Confidence Interval (CI) = -.30;-.15).

Table 1

Main Analyses

Using the Mplus software package (Muthén & Muthén, 1998-2004), two sets of analyses were performed. In the first set, two univariate models were fitted to the data to estimate the relative contribution of genetic and environmental factors to peer acceptance/rejection and depressive behavior, respectively. By comparing within-pair correlations for MZ twins (who are genetically identical) and DZ twins (who on average share only half of their genes), sources of variability of a measured variable (phenotype) can be estimated in terms of latent additive genetic (A), latent shared environmental (C), and latent nonshared environmental (E) factors (Neale & Cardon, 1992). Within-twin pair correlations of the latent genetic factors (A) are fixed to 1.0 for MZ twins and to 0.5 for DZ twins. Within-twin pair correlations of the latent shared environmental factors (C) are fixed to 1.0 for both MZ and DZ twins, and within-twin pair correlations of the latent nonshared environmental factors (E) are fixed to 0.0 for both MZ and DZ twins. The estimated coefficients a, c, and e, which are fixed to be equal across the two members of a twin pair and across MZ and DZ twins, are the factor loadings that provide information about the relative contribution of the latent factors A, C, and E to the total variance V_T of each phenotype, with $V_T = a^2 + c^2 + e^2$, with measurement error included in e^2.

In the next set of analyses, we first examined whether a gene-environment correlation (rGE) could be observed in the link between peer acceptance/rejection and depressive behavior. For this purpose, a bivariate Cholesky model (Figure 1) was specified where the covariance structure of peer acceptance/rejection and depressive behavior was partitioned into (1) "common" latent factors A_C , C_C , and E_C that influence both phenotypes, and (2) "unique" latent factors A_U , C_U , and E_U that are specific to depressive behavior. Coefficients a_P, c_P and e_P represent the factor loadings of peer acceptance/rejection (P) on the common latent factors. Coefficients a_C, c_C and e_C

represent the factor loadings of depressive behavior on the common latent factors. Coefficients a_U, c_U and e_U represent the factor loadings of depressive behavior on the unique latent factors. The relative contribution of the "common" latent genetic factor A_C to the variance of depressive behavior ($a_c^2/(a_c^2 + a_u^2)$) indicates the overlap or correlation between genetic dispositions to depressive behavior and peer acceptance/rejection (rGE).

Figure 1

The bivariate Cholesky model was then expanded to test whether genetic and environmental influences on depressive behavior are moderated by levels of peer acceptance/rejection. To this end, an interaction term was included on all pathways leading to depressive behavior, including those in common with peer acceptance/rejection and those unique to depressive behavior (Figure 2). A significant interaction between the "common" genetic factor A_C and Peer Acceptance/Rejection (represented by the term $\beta_{AC}P$) indicates that the *same* genetic factor is implicated in both rGE and GxE. A significant interaction between the "unique" genetic factor A_U and Peer Acceptance/Rejection (represented by the term $\beta_{AU}P$) indicates that a *different* genetic factor effect is implicated in GxE than in rGE. Interaction terms between the shared environmental factors C_C and C_U and Peer Acceptance/Rejection (represented by the terms $\beta_{CC}P$ and $\beta_{CU}P$) and between the nonshared environmental factors E_C and E_U and Peer Acceptance/Rejection (represented by the terms $\beta_{EC}P$ and $\beta_{EU}P$) were also added to this model. Inclusion of interactions with latent environmental factors is crucial to examine whether the moderating effect of the measured environment variable is specific to the latent genetic variance components, or whether it increases variation over the entire range of the phenotypic

distribution (Purcell, 2002). The total variance (V_T) of depressive behavior as a function of peer acceptance/rejection (P) can thus be expressed as

$$V_T \mid P = (a_C + \beta_{AC}P)^2 + (a_U + \beta_{AU}P)^2 + (c_C + \beta_{CC}P)^2 + (c_U + \beta_{CU}P)^2 + (e_C + \beta_{EC}P)^2 + (e_U + \beta_{EU}P)^2$$

Model fit for univariate and bivariate models was assessed based on the RMSEA and the -2LL and the χ^2 statistics. Low and nonsignificant χ^2 values and values of RMSEA below .08 indicate good fit and parsimony. All models fit the data well. Since χ^2 and RMSEA were not available for models including interaction terms, the bivariate models were compared using the –2LL difference test, which is equivalent to a nested χ^2-difference test (Purcell, 2002).

Figure 2

Univariate Model Results

As can be seen in Table 2, genetic factors accounted for 30.1% of the variance of peer acceptance/rejection, another 15.4% was explained by shared environmental sources, and the remaining 54.5% of the variance was due to nonshared environmental factors. Genetic factors explained 30.4% of the variance of depressive behavior, another (albeit nonsignificant) 3.2% was due to shared environmental sources, and the remaining 66.4% of the variance was accounted for by nonshared environmental factors. The finding that not only children's depressive behavior but also their level of acceptance versus rejection by the peer group has some genetic basis indicates the presence of a possible gene-environment correlation (rGE). Because the pattern of correlations did not suggest a dominance genetic effect D for the two study variables (Neale & Cardon, 1992), D effects were not estimated.

Table 2

Bivariate Model Results

A summary of the bivariate model tests without and with interaction terms is provided in Table 3. The results from Model 1 (without interaction terms) revealed that about 54% ($-.35^2/(-.35^2 + .32^2)$) of the genetic variance of children's depressed behavior was explained by genetic factors that also influenced peer acceptance/rejection. There was also a significant, albeit small, overlap of about 3% ($-.13^2/(-.13^2 + .78^2)$) in the nonshared environmental factors predicting depressed behavior and peer acceptance/rejection, but most nonshared environmental influences were specific to depressive behavior. Shared environmental effects on depressive behavior were non-significant and no significant overlap with peer acceptance/rejection was found.

Inclusion of the interaction terms in Model 2 significantly improved model fit, $\Delta\chi^2$ (6) = 14.852, p = .021. The results revealed a significant interaction of "Peer acceptance/rejection" with the latent genetic factor A_U that was unique to depressive behavior (β_{AU} = .21, CI = .01;.42) but not with the latent genetic factor A_C that was common to peer acceptance/rejection and depressive behavior (β_{AC} = .06; CI = -.23;.35). This latter finding suggests that the genetic influences on depressive behavior that are implicated in rGE with peer acceptance/rejection may not be the same as those that are implicated in GxE with peer acceptance/rejection. Peer acceptance/rejection also did not significantly interact with the latent shared environmental factors C_C and C_U (β_{CC} = -.02; CI = -.44;.41 and β_{CU} = .08; CI = -.43;.59) or with the latent nonshared environmental factor E_C (β_{EC} = .02; CI = -.13;.18). However, peer rejection significantly interacted with the latent nonshared environmental factor E_U that was unique to

depressive behavior (β_{EU} = -.17; CI = -.31;-.02). To identify the most parsimonious model that adequately explained the data without a significant loss in model fit, nonsignificant parameters were successively fixed to zero in additional analyses and the resulting nested model -2LL was compared with Model 2. The results from the best fitting model (Model 3) were basically identical to those from the full model (Model 2).

Table 3

Figure 3 plots the magnitude of the total variance of depressive behavior and of its components as a function of peer acceptance/rejection. As can be seen, total variance of depressive behavior is lower under conditions of peer rejection (peer acceptance/rejection below the mean of 0) than under conditions of peer popularity (peer acceptance/rejection above the mean of 0). This seems to be due to the fact that genetic influence on depressive behavior is reduced under conditions of peer rejection whereas it increases considerably under conditions of increasing peer popularity.

Figure 3

Discussion

This study examined gene-environment correlation (rGE) and gene-environment interaction (GxE) in the link between peer acceptance/rejection and children's depressive behavior. Univariate analyses confirmed previous findings of significant genetic effects on depressive behavior in children (Happonen et al., 2002; Scourfield et al., 2003), with the remaining variance

mostly explained by nonshared environmental factors. In line with rGE, a significant portion of the genetic factors influencing depressive behavior also predicted peer acceptance/rejection. This result corroborates previous findings that children with a genetic risk for depressive behavior are exposed to more negative social environmental experiences (Lau & Eley, 2008) and our study suggests that these negative experiences also include peer rejection. Peers' negative evaluations might be explained by the fact that depressed children's peer interactions are characterized by more conflict and less collaboration than interactions of nondepressed children (Altmann & Gotlib, 1988; Rudolph et al., 1994). Controlling for rGE, results also provided evidence that peer acceptance/rejection moderated the genetic and environmental effects on depressive behavior (GxE). Specifically, individual differences in depressive behavior were less explained by genetic factors under conditions of peer rejection, but they were under increasing genetic influence the more peer popularity increased. Interindividual variability of depressive behavior was also lower and the mean level of depressive behavior was higher under conditions of peer rejection than under conditions of popularity. These findings are inconsistent with a diathesis-stress or a compensation process and point instead to a suppression mechanism underlying GxE.

Peer rejection is a rather stable phenomenon, which is often expressed through social exclusion or physical and nonphysical forms of harassment (McDougall et al., 2001). Experiences of social rejection have been shown to activate the same areas of the brain that register physical pain (Eisenberger, Lieberman, & Williams, 2003). Since children generally attend kindergarten full time with the same peer group, they may have relatively few opportunities to escape this hurtful social context. In addition, many young children have not yet acquired the social interactional skills and coping strategies necessary to deal effectively with peer rejection. They more often report having cried or run away when being harassed than older children, which is

unlikely to remedy the situation and may accentuate their negative peer status (Smith, Shu, & Madsen, 2001). As such, peer rejection may constitute an important source of stress that may foster depressive behavior symptoms even in children who do not necessarily have a genetic propensity for such behavior. Interestingly, a similar environmental suppression of genetic effects was reported for the link between family adversity and stress-reactivity in infancy (Ouellet-Morin et al., 2008). A different pattern may be found in older children and adolescents, however. Since older youth generally have acquired more social and coping skills to deflect negative peer experiences, there may be greater variability in depressive behavior under conditions of peer rejection – and genetic propensity may explain a considerable portion of this variability. Hence, a GxE pattern consistent with a diathesis-stress process may be observed in the link between peer rejection and depressive behavior for older youth.

Strengths, Limitations, and Conclusions

This study is the first to assess gene-environment interplay in the link between a peer-related environmental stressor and children's depressive behavior symptoms. The use of different reporting sources eliminated shared source variance problems. The use of peer reports, which are rarely employed in twin studies, provided a more direct and less biased assessment of peer rejection than either self-ratings or mother and teacher reports of children's peer difficulties (Bukowski, Hoza, & Newcomb, 1994). Despite these strengths, our study also has several limitations. One limitation is the small sample size, which likely limited the statistical power to detect certain effects. Thus, the lack of shared environmental effects on depressive behavior, the lack of environmental moderation of common genetic effects, as well as the lack of sex moderation in the present sample may be attributable to low power. The high costs associated with classroom-based peer ratings rendered the assessment of a larger sample difficult, however.

Moreover, attrition analysis suggested that the final sample was not overly biased. The results may also not generalize beyond the assessed age, as the processes underlying GxE between peer rejection and depressive behavior may differ for older youth. Additionally, due to the somewhat low internal consistency of the teacher-rated depressive behavior measure, the phenotypic association may have been underestimated. Analyses thus need to be replicated in future studies, preferably using a multi-method multi-informant approach and testing for potential sex differences. Finally, it should be noted that the present findings of GxE, which pertain to changes in the variance components of depressive behavior as a function of a peer acceptance/rejection, are not directly comparable to more classical GxE analyses in molecular genetic studies, which assess the interactive contribution of genetic and environmental risk to changes in mean levels of a given phenotype (Button et al., 2007).

Despite these limitations, the present study provides important insights into the link between peer rejection and depressive behavior in young children. Specifically, while genetic propensity to depressive behavior might increase the risk of rejection by peers, peer rejection may foster depressive behavior even in children without a genetic disposition to such behavior. These results emphasize the importance of teaching social interactional skills that promote positive peer relations in all children to help prevent the development of depressive behavior at a young age.

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Table 1

Descriptive Statistics

	Peer rejection	***Depressive behavior***
MZ (n = 196)		
Mean	.01	-.01
SD	.98	1.00
r	.45	.34
DZ (n = 140)		
Mean	-.15	.01
SD	.99	.99
r	.31	.18

Note. SD = Standard deviation; r = Within-pair correlation.

Table 2

Univariate Models

	A	C	E	%A^2	%C^2	%E^2	RMSEA	-2LL (np)	χ^2	df	p
Peer rejection	.54 (.25;.83)	.39 (.06;.71)	.73 (.65;.80)	30.1	15.4	54.5	.00	2067.41 (5)	.034	1	.854
Depressive behavior	.55 (.22;.88)	.18 (-.62;.98)	.81 (.72;.89)	30.4	3.2	66.4	.02	2110.05 (4)	.174	2	.917

Note. Confidence Intervals are in parentheses. Np = number of parameters.

Table 3

Bivariate Models

	A	β_A	C	β_C	E	β_E	RMSEA	-2LL (np)	χ^2	df	p
Model 1 (without interactions)							.03	3756.02 (12)	7.35	4	.118
Peer rejection	.67 (.37;.86)	-	.67 (.37;.86)	-	.68 (.60;.75)	-					
Depressive behavior											
Common effects	-.35 (-.73;-.02)	-	.27 (-.45;.99)	-	-.13 (-.25;-.01)	-					
Unique effects	.32 (.28;.91)	-	.21 (-.03;1.45)	-	.78 (.70;.86)	-					
Model 2 (with interactions)							-	3741.18 (18)	-	-	-
Peer rejection											
	.62 (.42;.82)	-	.33 (.04; .61)	-	.68 (.61; .75)	-					
Depressive behavior											
Common effects	-.30 (-.52;-.09)	.06 (-.23;.35)	.23 (-.11;.57)	-.02 (-.44;.41)	-.14 (-.25;-.03)	.02 (-.13;.18)					
Unique effects	.33 (.03;.63)	.21 (.01;.42)	-.27 (-.64;.11)	.08 (-.43;.59)	.75 (.68;.83)	-.17 (-.31;-.02)					
Model 3 (best fitting model with interactions)							-	3743.68 (13)	-	-	-
Peer rejection											
	.64 (.43;.85)	-	.29 (.11; .69)	-	.68 (.60; .75)	-					
Depressive behavior											
	-.35	-	.36	-	-.13	-					

Common effects	(-.58;-.12)		(.00;.72)		(-.23;-.02)	
	.26	.22	-	-	.76	-.19
Unique effects	(.13;.66)	(.01;.43)			(.69;.83)	(-.32;-.06)

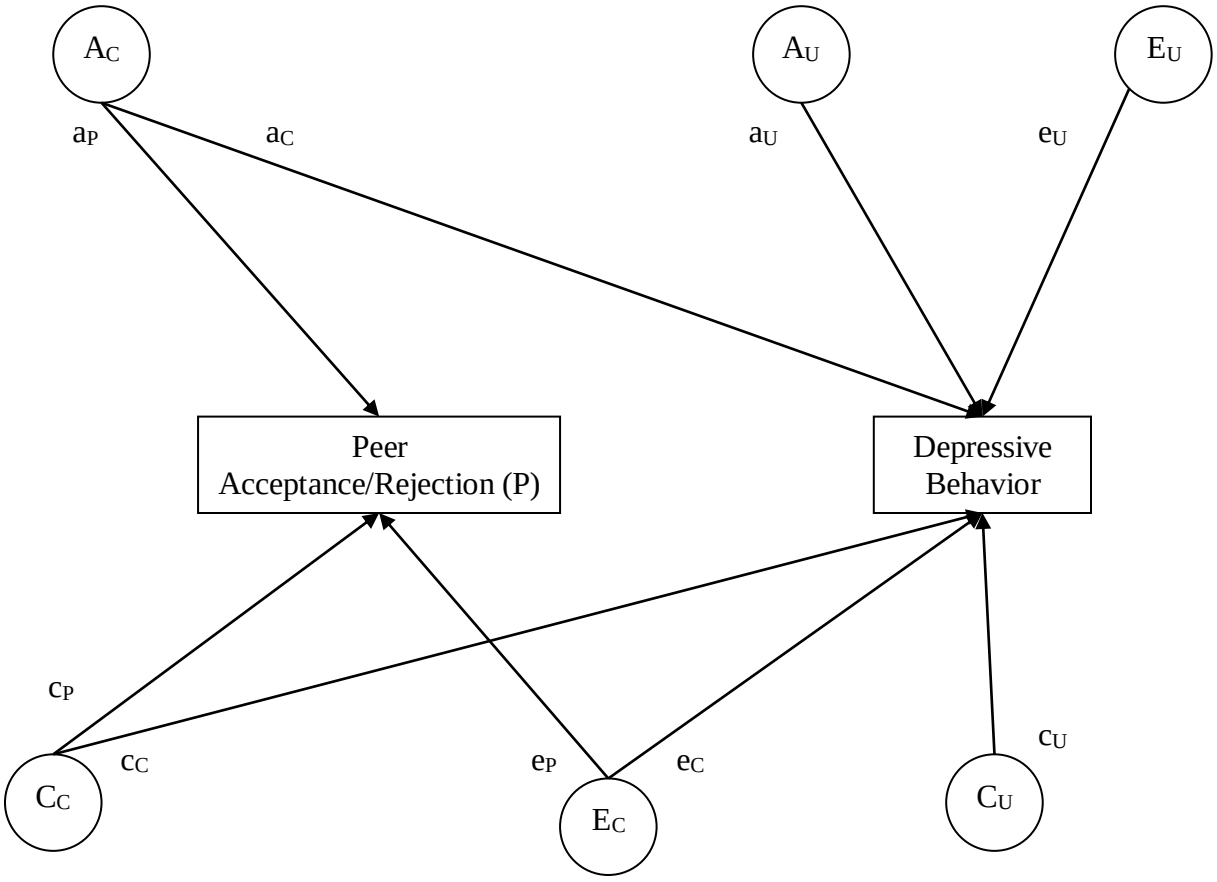
Note. Model 1 versus Model 2: Δ-2LL(6) = 14.852, p = .021; Model 1 versus Model 3: Δ-2LL(1) = 12.352, p = .001; Model 2 versus Model 3: Δ-2LL(5) = 2.5, p = .777.

Figure Captions

Figure 1. Bivariate model (for one member of a twin pair) of peer acceptance/rejection and depressive behavior.

Figure 2. Bivariate model (for one member of a twin pair) incorporating interactions between peer acceptance/rejection and latent genetic and environmental effects on depressive behavior.

Figure 3. Total variance and genetic, shared environmental, and nonshared environmental variance components of depressive behavior as a function of peer acceptance/rejection.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Peer Acceptance/Rejection (P)
Depressive Behavior
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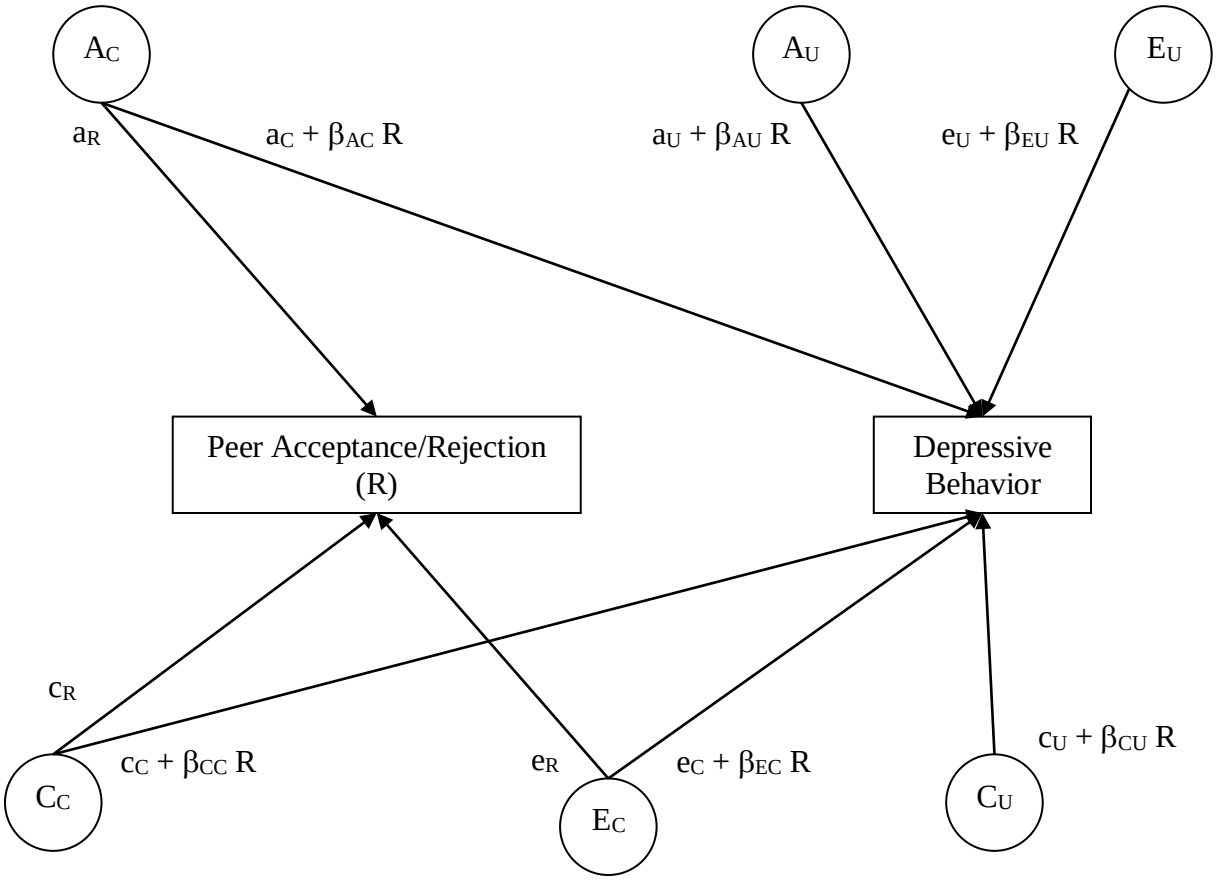
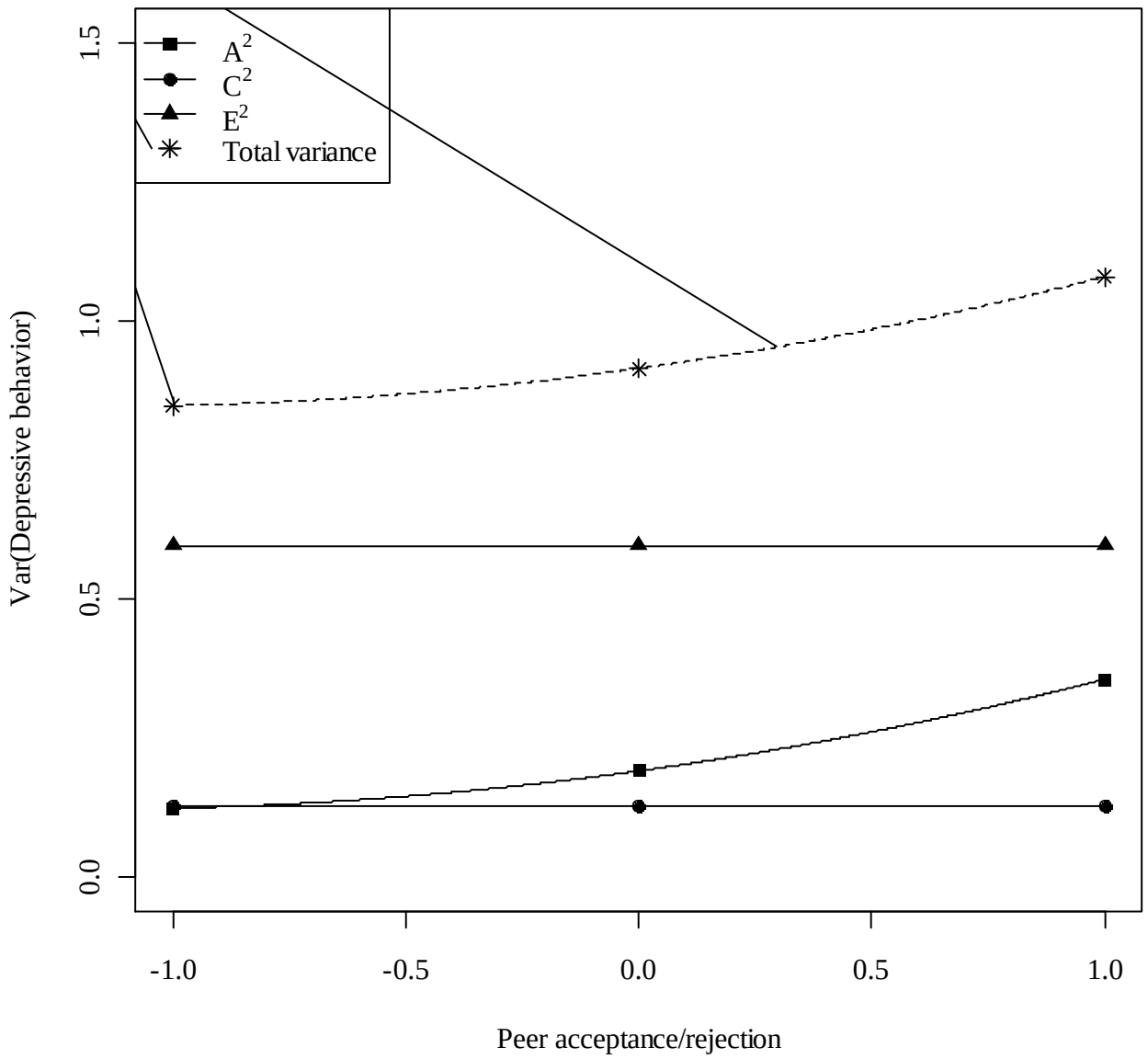

A_C
A_U
E_U
a_R
$a_C + \beta_{AC} R$
$a_U + \beta_{AU} R$
$e_U + \beta_{EU} R$
Peer Acceptance/Rejection (R)
Depressive Behavior
c_R
$c_C + \beta_{CC} R$
e_R
$e_C + \beta_{EC} R$
$c_U + \beta_{CU} R$
C_C
E_C
C_U


A2
C2
E2
Total variance
Var(Depressive behavior)
0.0
0.5
1.0
1.5
-1.0
-0.5
0.0
0.5
1.0
Peer acceptance/rejection

- Depressive behavior symptoms in children appear to be at least in part explained by genetic factors.
- A child's genetic risk for depressive behavior may increase the likelihood of being rejected by peers.
- By the same token, the experience of peer rejection may constitute an important source of stress that may foster depressive behavior symptoms even in children who do not necessarily have a genetic propensity for such behavior.
- These results emphasize the importance of teaching social interactional skills that promote positive peer relations in all children to help prevent the development of depressive behavior at a young age.

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