Estimating Genetic and Environmental Influences on Depressive Symptoms in Adolescence: Differing Effects on Higher and Lower Levels of Symptoms

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We estimate the relative effect sizes of genetic and environmental influences on both higher and lower levels of depressive symptoms with attention to persistence over a 1-year period in the genetically informative subsample of adolescents participating in the National Longitudinal Study of Adolescent Health (Add Health). Shared environmental effects were significant for persistent higher levels of depressive symptoms but not nonpersistent symptoms. Genetic effects were significant for both persistent and nonpersistent lower levels of depressive symptoms. Nongenetic factors that promote similarity between siblings for high levels of depressive symptoms are important and should be considered in both etiological and applied research. Genetic contributions to lack of susceptibility to depression should be considered in biological models of depression suppression.

There is increasing recognition that depressive symptoms in adolescence may be of developmental and clinical significance. In addition to reflecting emotional and cognitive distress, such symptoms have been tied to various adverse outcomes, including psychosocial impairment and the utilization of mental health services (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Subthreshold depressive symptoms in adolescence are also predictive of major depressive disorder and suicidal behavior in adulthood (Fergusson, Horwood, Ridder, & Beautrais, 2005; Pine, Cohen, Cohen, & Brook, 1999), thus potentially reflecting earlier manifestations of liability to this disorder. Furthermore, it has been shown that adolescents who endorse high levels of depressive symptoms demonstrate high rates of persistence 1 year later, implying the need for long-term assessment and clinical evaluation (Rushton, Forcier, & Schectman, 2002). Thus, high levels of depressive symptoms in adolescence warrant attention both in terms of etiological mechanisms and resultant implications for preventive and interventive strategies.

Much attention has been given to deciphering the relative contributions of genes and environment on depressive symptoms in adolescence as a window into potential etiological mechanisms (Deater-Deckard, Reiss, Hetherington, & Plomin, 1997; Eley, 1997; Rende, Plomin, Reiss, & Hetherington, 1993; Rice, Harold, & Thapar, 2002). Two complementary strategies have been used. First, traditional behavioral genetic analysis has been used to decompose the variance in depressive symptoms into the three basic components of heritability (additive genetic variance; A), shared or common environment (C), and unique or nonshared environment (E). Across all studies, heritability estimates have been significant and of moderate effect size, typically accounting for about 30% of the variance. Shared environmental effects have been nonsignificant and of small enough magnitude to be dropped from a best-fitting model. The majority of the variance has been attributable to nonshared environmental effects, which incorporates measurement error and residual variance along with potential unique environmental effects not shared by family members.

A second strategy has focused specifically on higher levels of depressive symptoms (such as those exceeding typical cutoffs on depression inventories) using an analytic strategy referred to as DeFries–Fulker...
(DF) analysis (DeFries & Fulker, 1985, 1988; Rende, 1999; Rende & Slomkowski, 2005). The DF method is unique in that it provides a computational model to assess the magnitude of A, C, and E for “extreme” scores on a continuum to complement the more traditional focus on these parameters based on the full distribution of scores. In particular the DF method was designed as an analytic technique suitable for skewed distributions in which the clinical interest is often in those cases at the tail, as is typically found for measures of psychopathology. The important aspect of the DF method is that the estimation of the ACE parameters is based on both an empirical cutoff in the distribution (to identify probands) and the quantitative score of the cosiblings. It is based conceptually on the cosibling regression toward the population mean (or most typically mean of the sample) stratified by genetic relatedness (see Rende, 1999; further details are provided in the Methods section). The DF approach differs from traditional behavioral genetic analysis of the full continuum of scores, which assumes normality and thus could mask findings specific to the extremes. It also differs importantly from traditional “liability” models of disorder, which rely on categorical representation of probands and siblings and hence do not directly measure the ACE components using the observable quantitative scores (Rende, 1999).

To date, the substantive issue has been to determine if the effect sizes of the ACE parameters for the extremes differ from those using the full distribution of scores. Such a finding would imply that etiological investigations need to focus on mechanisms specific to producing high levels of symptoms that are not detectable when decomposing the variance of the entire skewed distribution. The studies applying DF analysis to depressive symptoms have suggested that genetic influences on higher levels of symptoms (those that exceed a traditional cutoff) are of similar magnitude to those estimated to affect the full distribution of scores (Deater-Deckard et al., 1997; Eley, 1997; Rende et al., 1993; Rice et al., 2002). In contrast, these studies reported that shared environmental factors have a detectable impact on extreme scores that is statistically larger than the effect size in the traditional model. This finding has been especially important as evidence for shared environmental factors has been elusive in behavioral genetic studies (Dick & Rose, 2002). The demonstration of shared environmental effects at the extremes may revamp the basic conclusion from behavioral genetics research that shared environmental effects are negligible and implies that social factors have a similar impact on siblings’ risk for depression in adolescence (Rende, 2004; Rende & Waldman, in press). Importantly, these studies have all been conducted with representative samples of adolescent twins in both the United States (Deater-Deckard et al., 1997; Rende et al., 1993) and the United Kingdom (Eley, 1997; Rice et al., 2002) suggesting good generalizability of findings to adolescent cohorts.

In this article we attempt to extend our understanding of the etiology of the extremes of adolescent depressive symptoms by utilizing a genetically informative sample derived from the National Longitudinal Study of Adolescent Health (Add Health; Resnick et al., 1997). This sample is advantageous because it is larger than any of the samples used in prior DF studies of depressive symptoms, reflects a nationally representative collection of U.S. adolescents, incorporates multiple levels of genetic relatedness, and includes two waves of assessment (1 year apart). We address two fundamental questions with the available data from these two first waves of the Add Health study. First we explore the impact of persistence of higher levels of depressive symptoms on the estimates derived from the DF model. Understanding the etiology of persistently higher levels of depressive symptoms would have particular relevance for prevention and intervention, especially as none of the studies to date using the DF methodology have analyzed data from multiple time points. Second, we explore the new issue of the etiology of lower levels of depressive symptoms in adolescence, as there may also be unique influences that act as a protective factor for those individuals who experience few or none depressive symptoms. There is evidence that low levels of psychological distress in adults are attributable to genetic influences but not shared environmental effects as determined using DF analysis (Rijndijk et al., 2003). However, to date the DF method has not been applied to lower levels of depressive symptoms in adolescent populations.

Method

Participants

The data were from Add Health, an ongoing study designed to assess the health status of adolescents and explore the causes of adolescent health-related behaviors (see Resnick et al., 1997). The study population includes a representative sample of all public and private high schools in the United States systematically identified to represent regional, urban, and racial strata. Participants consisted of 20,747 adolescents who were drawn to be representative of a national school survey of 90,118 adolescents using the sampling procedure noted previously. Participants completed in-home interviews administered between April and December of 1995; a follow-up interview was conducted 1 year later. Adolescents were stratified by gender and grade (7 through 12) from 80 high schools and 52 feeder middle schools. These high schools were chosen from an original sample of all United States high schools that included an 11th grade and enrolled
30 or more students. To ensure that they were nationally representative, the 80 high schools were systematically selected from the original sampling frame, with stratification by region, urbanicity, ethnicity, school type, and school size. Approximately 73% of the sample is Caucasian.

Within this larger sample, a subsample of families contributed more than one adolescent to the study. Specifically, a genetically informative subsample of sibling pairs was ascertained, reflecting a range of genetic relatedness from 100% (monozygotic [MZ] twins) to 0% (biologically unrelated siblings [US]). Only two siblings per family were included in our analyses. Determination of twin zygosity was based primarily on twins’ and parents’ reports of how physically similar the twins were to one another (Jacobson & Rowe, 1999). For 89 same-sex twins for whom zygosity remained uncertain, DNA genetic markers were used to decide zygosity. MZ pairs were identical for 11 genetic markers; dizygotic (DZ) pairs differed by one or more markers. The other types of siblings were categorized according to information about the household structure in the home interview and included full siblings (FS), HS, and US.

These analyses specifically used data from 1,978 sibling pairs (i.e., 3,956 individuals; average age = 15.5 years, $SD = 1.71$). By sibling type, the sample sizes were MZ, 232 pairs (115 male–male and 117 female–female); DZ, 371 pairs (102 male–male, 97 female–female, 172 mixed sex); FS, 841 pairs (250 male–male, 249 female–female, 342 mixed sex); HS, 295 pairs (71 male–male, 81 female–female, 143 mixed sex); US, 239 pairs (66 male–male, 56 female–female, 117 mixed sex).

Materials

Depressive symptoms were recorded using 20 items that corresponded with the 20-item Center for Epidemiologic Studies–Depression Scale (CES–D; Radloff, 1977) in both Wave 1 and Wave 2. The CES–D has been used to characterize depressive symptoms in adolescents with acceptable reliability and validity (Garison, Addy, Jackson, McKeown, & Waller, 1991), although the evidence for the sensitivity and specificity in studies of adolescent populations has been mixed (Rushton et al., 2002). As described by Rushton et al., the wording pertaining to two symptoms (“restless sleep” and “crying spells”) was slightly modified in the Add Health survey. Each of the 20 items was scored from 0 (never) to 3 (daily) on the basis of frequency of depressive symptoms reported during the past week.

Design and Procedures

Study interviewers read and recorded responses for items deemed to be minimally intrusive or sensitive, including those pertaining to depressive symptoms. Adolescent consent or assent was obtained in the home with cooperation by both the adolescent and a parent or guardian.

Scoring

**Depressive symptoms.** A total CES–D score was computed by summing all 20 items producing a possible range of 0 (no depressive symptoms) to 60. Principal components analysis yielded a single factor solution, with good internal consistency at both Wave 1 (Cronbach’s $\alpha = .86$) and Wave 2 (Cronbach’s $\alpha = .87$). Correlation between Wave 1 and Wave 2 depression scores was .57.

We utilized a cutoff of 16 to represent higher levels of depressive symptoms at each time point as is traditionally done with the CES–D. Rushton et al. (2002) have suggested that this cutoff reflects mild depressive symptoms, with a cutoff of 24 reflecting more severe depressive symptoms. However, given our interest in persistence of symptoms, we chose to utilize the “mild” cutoff of 16 to ensure appropriate power for the DF analyses, which have adequate power to detect small effect sizes for tails that represent about 10% of the continuum (Rende et al., 1993). Using the cutoff of 16 at each time point, we categorized participants as persistent if they met this cutoff at both Wave 1 and 2 and nonpersistent if they met this cutoff for only one wave. About 8% of the sample (70% female) were high persistent cases and 19% of the sample (55% female) were high nonpersistent cases. For low scores, we followed Rijnsdijk et al. (2003) and selected a cutoff score of 2 or less at each wave. About 10% of the sample (36% female) were low persistent and 17% of the sample (47% female) were low nonpersistent cases. After accounting for pairs in which both members met a cutoff, the final number of pairs analyzed were as follows: 293 high persistent probands and their cosiblings (32 MZ, 42 DZ, 118 FS, 54 HS, and 47 US), 646 high nonpersistent probands and their cosiblings (83 MZ, 119 DZ, 245 FS, 122 HS, and 77 US), 335 low persistent probands and their cosiblings (49 MZ, 73 DZ, 154 FS, 32 HS, and 27 US), and 614 low nonpersistent probands and their cosiblings (66 MZ, 123 DZ, 276 FS, 83 HS, and 66 US). Gender distributions of the cosiblings were 55% female for high persistent probands, 53% female for high nonpersistent probands, 43% female for low persistent probands, 51% female for nonpersistent low probands.

**Genetic relatedness.** Sibling pairs were categorized based on their level of genetic relatedness (R). MZ twins were assigned a genetic relatedness correlation of 1.0, reflecting their 100% shared genotype. US raised in the same family (e.g., step-siblings, children from successive adoptions, an adopted child and a bio-
Results

Analytic Strategy

Multiple levels of genetic relatedness are incorporated into DF regression models that can be used to generate estimates of heritable ($h^2$) and shared environmental ($c^2$) influences (Cherny, DeFries, & Fulker, 1992; DeFries & Fulker, 1985, 1988; Rende et al., 1993; Rende & Slomkowski, 2005; Rodgers & McGue, 1994). Analyses of the extremes require two steps (DeFries & Fulker, 1985, 1988; Rende et al., 1993; Rende & Slomkowski, 2005). First, probands are identified as those individuals exceeding a preselected cutoff score (as described previously for our various groupings). Once identified, the regression model is modified to predict cosibling depressive symptoms (DCS) as a function of proband depressive symptoms (DP) and genetic relatedness:

$$DCS = b_0 + b_1DP + b_2R + e.$$ 

As shown by DeFries and Fulker (1985, 1988), $b_1DP$ represents an estimate of shared environmental influence specific to extreme group membership, and $b_2R$ represents an estimate of heritability specific to extreme group membership. Conceptually, the model analyzes regression toward the mean in the cosibling’s score as a function of genetic relatedness (see Rende, 1999). Heritability is implied by the extent to which such regression is conditional on genetic relatedness, and shared environmental effects are suggested by the extent to which there is only partial regression toward the mean (i.e., higher than average cosibling scores) not conditional on genetic relatedness.

If both siblings in a pair exceed the cutoff, one is randomly assigned as proband and the other as cosibling (e.g., Rende et al., 1993). Although other methods have been used in prior studies such as double-entry of extremes and adjustment of variance components based on that procedure, such corrections have been done in twin samples, without having the necessity to deal with the computational demands raised by the combined twin-step design. Also, it has been suggested (Eley, 1997) that the double-entry method may yield less than optimal values for the group shared environmental component. Individual raw scores are transformed, (Raw Score – Mean of Sample / Proband Mean – Mean of Sample) to yield directly interpretable parameters in the regression model (see DeFries & Fulker, 1985; Eley, 1997; Rende et al., 1993). Confidence intervals may be computed around the parameters for alternative models (e.g., parameters generated for persistent vs. nonpersistent symptoms) to compare the overlap; a lack of overlap is taken as evidence of a significant difference between these parameters (Rende et al., 1993). For all models run, we corrected for effects of age, age difference between siblings, gender, and gender composition of the sibling pair.

Mean Scores

Given that our cutoffs utilize both Wave 1 and Wave 2 data, we focus on an average CES–D score for probands and cosiblings as an indicator of depressive symptoms displayed during the two assessment periods. As a first descriptive step, we present the means (corrected for age and gender), stratified by sibling type, for the probands and cosiblings for persistent and nonpersistent high depressive symptoms (Table 1) and for persistent and nonpersistent low depressive symptoms (Table 2). One important comparison is to examine the means of cosiblings as compared to 9.07, the mean average CES–D score in this sample (based on all participants). Deviations from this mean (the expected value if there is no familial resemblance) indicate familiality, and differential deviations associated with genetic relatedness would indicate the possible importance of heritable factors as a source of familiality.

We consider first the means for cosiblings of probands with persistent high depressive symptoms

### Table 1. Means and Standard Deviations for Averaged Depression Scores for Probands With Higher Levels of Depressive Symptoms and Their Cosiblings Stratified by Proband Persistence

| Sibling Type | Persistent Proband | | | Persistent Cosibling | | | Nonpersistent Proband | | | Nonpersistent Cosibling |
|--------------|-------------------|---|---|----------------------|---|---|-----------------------|---|---|
|              | $M$               | $SD$ | $M$ | $SD$ | | $M$ | $SD$ | | $M$ | $SD$ | $M$ | $SD$ |
| MZ           | 23.51             | 3.98 | 14.12 | 6.21 | | 14.70 | 2.56 | 12.07 | 6.69 |
| DZ           | 21.39             | 4.19 | 12.24 | 5.49 | | 15.44 | 2.87 | 12.24 | 5.49 |
| FS           | 22.35             | 4.87 | 12.06 | 7.21 | | 14.92 | 2.87 | 11.24 | 6.34 |
| HS           | 23.17             | 5.38 | 13.50 | 7.81 | | 15.47 | 3.09 | 10.61 | 6.55 |
| US           | 22.43             | 4.93 | 11.04 | 9.24 | | 16.17 | 3.76 | 10.18 | 5.69 |

Note: MZ = monozygotic twins; DZ = dizygotic twins; FS = full biological siblings; HS = half-siblings; US = biologically unrelated siblings.
The evidence for a dose–response relation between genetic relatedness and cosibling mean is not especially strong, as the cosibling mean for HS is greater than the cosibling means of the DZ twins and FS. Furthermore, as the differences in cosibling mean across sibling type appear to be a small effect size (as referenced by the standard deviations) and the cosibling mean for all groups is greater than the mean score in the sample, the possibility of shared environmental influence is raised. For the siblings of nonpersistent high probands (Table 1), there is stronger evidence for genetic influence (a dose–response relation between genetic relatedness and cosibling mean). Again, all cosibling means are elevated compared to the sample mean, suggesting some form of familial resemblance.

The pattern of cosibling means given the presence of a proband with persistently low scores strongly suggests a dose–response relation between genetic relatedness and cosibling mean (Table 2). In particular, the effect size of difference between contiguous groups of genetic relatedness (e.g., MZ cosibling vs. DZ and FS cosibling) implies a large heritability. Although the pattern of cosibling means for the nonpersistent low condition are also consistent with genetic influence (Table 2), the smaller differences between the means for each group based on genetic relatedness suggests a lower magnitude of genetic effects (and raises the possibility of shared environmental effects).

DF Models

The DF model formalizes the observations made previously concerning genetic relatedness and cosibling means. It provides a simultaneous estimate of the genetic and shared environmental influences on each proband grouping by examining the regression of the cosibling mean as a function of genetic relatedness, after controlling for age and sex. The estimates of additive genetic influence and shared environment on each proband group, along with the corresponding confidence intervals, are presented in Table 3.

First we consider the parameters for high levels of depressive symptoms. Shared environmental influences were significant for persistent high depressive symptoms, but not nonpersistent symptoms. The confidence intervals did not overlap, suggesting a statistically significant difference between these estimates given the sample size. Although the heritability estimate of nonpersistent high depression was moderate (.28), it was not significant and did not differ significantly from the lower heritability estimate (.08) of persistent high depression.

For low depression scores, shared environmental effects were nonsignificant for both persistent and nonpersistent cases. The confidence intervals did overlap, suggesting no significant difference between these estimates. Heritability estimates for both persistent and nonpersistent low depression were significant and large. The confidence intervals did not overlap, suggesting that the heritability of persistent low depression was significantly higher than the heritability for nonpersistent low depression.

Discussion

This report adds to a prior literature documenting shared environmental effects on higher levels of depressive symptoms in adolescence (Deater-Deckard et al., 1997; Eley, 1997; Rende et al., 1993; Rice et al., 2002). This study provided data on two waves of data and demonstrated that significant shared environmental...

### Table 2. Means and Standard Deviations for Averaged Depression Scores for Probands With Lower Levels of Depressive Symptoms and Their Cosiblings Stratified by Proband Persistence

<table>
<thead>
<tr>
<th>Sibling Type</th>
<th>Persistent Proband</th>
<th>Cosibling</th>
<th>Nonpersistent Proband</th>
<th>Cosibling</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>MZ</td>
<td>1.25</td>
<td>0.65</td>
<td>3.78</td>
<td>3.64</td>
</tr>
<tr>
<td>DZ</td>
<td>1.38</td>
<td>0.59</td>
<td>6.10</td>
<td>6.00</td>
</tr>
<tr>
<td>FS</td>
<td>1.33</td>
<td>0.61</td>
<td>6.04</td>
<td>4.28</td>
</tr>
<tr>
<td>HS</td>
<td>1.31</td>
<td>0.70</td>
<td>7.45</td>
<td>4.78</td>
</tr>
<tr>
<td>US</td>
<td>1.33</td>
<td>0.69</td>
<td>11.46</td>
<td>7.41</td>
</tr>
</tbody>
</table>

Note: MZ = monozygotic twins; DZ = dizygotic twins; FS = full biological siblings; HS = half-siblings; US = biologically unrelated siblings.

### Table 3. Estimates With 95% Confidence Intervals of Heritability and Shared Environment for Higher and Lower Levels of Depressive Symptoms Stratified by Persistence

<table>
<thead>
<tr>
<th></th>
<th>Heritability</th>
<th>Shared Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
<td>95% CI</td>
</tr>
<tr>
<td>High Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent</td>
<td>.08</td>
<td>.02–.14</td>
</tr>
<tr>
<td>Nonpersistent</td>
<td>.28</td>
<td>.12–.44</td>
</tr>
<tr>
<td>Low Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent</td>
<td>.85</td>
<td>.70–.99**</td>
</tr>
<tr>
<td>Nonpersistent</td>
<td>.50</td>
<td>.34–.66**</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01.
ental effects were found only for persistent higher symptoms. This is critical from a clinical perspective as it would be suspected that adolescents with persistent symptoms may suffer more consequences and may be at elevated risk for future depressive episodes, including those that might meet diagnostic criteria. We note here that the mean level of the probands with persistent higher levels of depression (ranging from 21.39–23.51) broaches the more extreme cutoff (24) proposed by Rushton et al. (2002) as a more sensitive screen for adolescent depression (at each wave, the mean depression score for this group exceeded 22). Although the predictive validity of the CES–D has been questioned (see Rushton et al., 2002), a number of studies suggest that adolescents with subthreshold levels of depressive symptoms are at risk for developing major depressive disorder (Fergusson et al., 2005; Pine et al., 1999; Rushton et al., 2002). This study implicates shared environmental factors as a potential critical etiological mechanism for such persistent subthreshold levels of symptoms or mild depression (Rushton et al., 2002).

It is thus imperative that conclusions about the lack of shared environmental effects on developmental outcomes (see Dick & Rose, 2002; Rende, 2004; Rende & Waldman, in press) be tempered by a consideration of more specific symptom profiles, including level as well as persistence. Reliance on traditional behavioral genetic models of depressive symptoms in adolescence would imply that there are no nongenetic factors that contribute to familial resemblance for high depressive symptoms. The prior studies using DF analysis and our study suggest that this is not accurate for higher levels of depressive symptoms and especially persistent symptoms, one putative target of intervention and prevention efforts. As family-based interventions are well recognized as having potential to reduce levels of depression in youth (e.g., Beardslee, Gladstone, Wright, & Cooper, 2003), the results of our study suggest that such programs consider the possibility of having beneficial outcomes for multiple children in the family. Recording data on the effects on siblings of targeted youth may strengthen the support for family-based intervention and prevention models.

Our data on lower levels of depressive symptoms suggest a different etiological model for these extremes. Genetic influences are not only significant but estimated as a large effect size, whereas shared environmental effects are negligible. Prior work with an adult sample also concluded that genes rather than shared environment confer protection against depressive symptoms (Rijksdijk et al., 2003). Although genetic effects are typically conceptualized as risk factors for psychopathology, these results imply that they may also serve as protective factors as well (Rende & Waldman, in press). A focus on the biological characteristics of adolescents who report few or no depressive symptoms could provide a model for buffering of depression.

The results of our study should be interpreted in light of a number of limitations. The Add Health study did not include diagnostic assessments, precluding the possibility of extending the DF model to segregate by the presence or absence of major depressive disorder and age of onset. Most efficient would be longitudinal follow-up studies of genetically informative samples that would allow for the testing of more complex developmental models. Given the inherent limitations of power when conducting analyses of extreme scores in a population, we did not pursue in this article potential age and sex differences in shared environmental effects on higher levels of depressive symptoms, although we did provide statistical correction for these variables in our analytic models. We note here that these would be of theoretical importance but pragmatically would be difficult to explore even in a sample as large as the one utilized in this report. Again, this would be an ideal topic for future large-scale investigations on the etiological architecture of depressive symptoms in adolescence and the onset of major depressive disorder in adolescence and adulthood. It is interesting to note, however, that recent work suggests no sex differences in the etiology of depressive symptoms in adulthood (Agrawal, Jacobson, Gardner, Prescott, & Kendler, 2004). Other potential factors that could influence the relative mix of genetic and environmental influences on depressive symptoms, such as ethnicity and socio-economic status, should also be considered in future investigations.

As a final consideration, we turn to speculations concerning how genes and social environment come together as predictors of depressive symptoms, a prominent issue in behavioral genetic research (Caspi et al., 2003; Moffitt, Caspi, & Rutter, 2005). The DF methodology is rooted in an additive model that is not capable of detecting Gene × Environment interaction. The results of this study suggest that alternative models capable of testing for Gene × Environment effects on both high and low levels of depressive symptoms might be illuminating. For example, given the repeated demonstration of shared environmental influences on high depression scores, future studies should focus on sources of such influence. These should include the range of risk factors typically shown to affect adolescent depressive symptoms, including individual, family, school, and community variables (e.g., Bond, Toumbourou, Thomas, Catalano, & Patton, 2005). Once identified, these environmental features could be crossed with candidate genes to examine if there is evidence for Gene × Environment interaction. Similarly, those with persistent low depression scores would be hypothesized to show a lack of responsivity if exposed to environmental risks. Such work could expand our understanding of the underlying mechanisms responsible for persistently high and low depressive symptoms in adolescence.
References


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