

The Timing of Parent and Child Depression: A Hopelessness Theory Perspective

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This study examined whether children's inferential styles moderate the association between the onset of depressive symptoms in children and their parents. To provide a powerful test of our hypotheses, we utilized a high-risk sample (parents with a history of major depressive episodes and their children) and a multiwave longitudinal design. During the initial assessment, 140 children (ages 6 to 14) completed measures assessing depressogenic inferential styles. Parents and children also completed measures assessing current level of depressive symptoms. Following the initial assessment, children and parents were contacted every 6 weeks for the next year to complete measures assessing depressive symptoms. The results of hierarchical linear modeling analyses indicated that children who exhibited depressogenic inferential styles reported greater elevations in depressive symptoms following elevations in their parent's level of depressive symptoms than did children who did not exhibit such styles. The strength of this association was greater in girls.

There is a wide consensus in the psychological literature that depression runs in families (Goodman & Gotlib, 2002; Hammen, 1991). Previous research has shown that children of parents with a history of major depressive episodes are four to six times more likely than other children to develop major depression (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Hammen, Burge, Burney, & Adrian, 1990; Weissman, Warner, Wichramaratne, Moreau, & Olfson, 1997). Furthermore, previous research has reported a significant temporal association between mother and child diagnoses (e.g., Hammen, Burge, & Adrian, 1991). At the same time, relatively little research has examined potential factors that moderate this association. Clearly not all high-risk children develop depressive symptoms following the onset of parental symptoms. Thus, it is possible that some children possess certain characteristics that make them more vulnerable than other children to the deleterious impact of parental depression.

One framework from which the temporal association between parent and child depression can be exam-

ined is from the perspective of the hopelessness theory (Abramson, Metalsky, & Alloy, 1989). The hopelessness theory posits three depressogenic inferential styles that serve as vulnerability factors to depression: (a) the tendency to attribute negative events to global and stable causes, (b) the tendency to perceive negative events as having many disastrous consequences, and (c) the tendency to view the self as flawed or deficient following negative events. Although research with adults has conceptualized the relation among these three styles using an additive approach (e.g., Alloy et al., 2000), research with younger samples has demonstrated that a weakest link approach (e.g., a child is as vulnerable to depression as his or her most depressogenic inferential style makes him or her; Abela & Payne, 2003; Abela & Sarin, 2002) better captures children and early adolescents' degree of vulnerability. Such a developmental difference is likely attributable to age-related differences in the degree to which the three inferential styles are associated with one another. In adults, inferential styles about the self, consequences, and causes are empirically indistinguishable (e.g., across studies, the average correlation between each possible pairing of the three inferential styles ranges from $r = .62$ to $r = .85$; Abela, 2002; Abela, Aydin, & Auerbach, in press; Abela & Seligman, 2000; Metalsky & Joiner, 1992). In children and early adolescents, however, the three inferential styles are relatively independent of one another (e.g., across studies, the average correlation between each possible

This research was supported, in part, by a Young Investigator Award from the National Alliance for Research on Schizophrenia and Depression (NARSAD) awarded to John R. Z. Abela. We thank Martin E. P. Seligman and David C. Zuroff for serving as mentors for the NARSAD grant.

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pairing of the three inferential styles ranges from $r = .23$ to $r = .36$; Abela, 2001; Abela & Payne, 2003; Abela & Sarin, 2002). Such a difference in patterns of interrelatedness suggests that the three depressogenic inferential styles featured in the hopelessness theory may consolidate into a broader “depressogenic cognitive style” during adolescence.

For children who possess depressogenic inferential styles, the onset of parental depression may serve as a significant stressor that increases the likelihood that they make depressogenic inferences. Parental depression may serve as a stressor for a multitude of reasons. For example, children of currently depressed parents are more likely than other children to be exposed to (a) parental irritability, aggression, dysphoria, and withdrawal (Cohn & Campbell, 1992; Cummings, Zahn-Waxler, & Radke-Yarrow, 1981); (b) dysfunctional parenting practices, such as inconsistent, lax, and ineffective child management (Fendrich, Warner, & Weissman, 1990); and (c) marital conflict (Beach, Smith, & Fincham, 1994). The hopelessness theory would predict that if a child makes depressogenic inferences for such stressors (e.g., “my parents are fighting and therefore may get divorced” or “My mom is quiet because she is angry at me”), he or she would be at increased risk for developing hopelessness. Once hopelessness develops, according to the theory, depression is inevitable as the theory views hopelessness as a sufficient cause of depression. It is important to emphasize that the hopelessness theory is a diathesis-stress theory. In other words, individuals who possess depressogenic inferential styles are only more likely than other individuals to exhibit depressive symptoms following negative events.

Although early research examining the attributional vulnerability hypothesis of the hopelessness theory in children and early adolescents obtained mixed support (e.g., Abela, 2001; Conley, Haines, Hilt, & Metalsky, 2001; Dixon & Ahrens, 1992; Hilsman & Garber, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1992; Robinson, Garber, & Hilsman, 1995; Turner & Cole, 1994), recent research examining the cognitive vulnerability hypothesis of the theory in children and early adolescents using a weakest link approach has obtained more consistent support for the theory (e.g., Abela & Payne, 2003; Abela & Sarin, 2002). In the first study to examine the weakest link hypothesis, Abela and Sarin had a community sample of 79 seventh-grade children (63 girls and 16 boys) complete measures assessing depressogenic inferential styles and depressive symptoms. Ten weeks later, children completed measures assessing depressive symptoms and negative events. When examined individually, none of the three depressogenic inferential styles featured in the hopelessness theory interacted with negative events to predict increases in depressive symptoms. At the same time, in line with the weakest link

hypothesis, children’s weakest links interacted with negative events to predict increases in depressive symptoms. Similarly, using a community sample of 130 third-grade children (63 girls and 67 boys) and 184 seventh-grade children (78 girls and 106 boys), Abela and Payne reported that children’s weakest links interacted with negative events to predict increases in depressive symptoms over a 6-week interval. Given the promising results obtained in research examining the weakest link hypothesis in child and early adolescent samples, research testing the diathesis-stress component of the hopelessness theory in youth is likely to benefit from conceptualizing cognitive vulnerability to depression utilizing a weakest link approach.

The goal of this study was to examine whether children possessing a depressogenic weakest link report greater increases in depressive symptoms following increases in their parents’ levels of depressive symptoms than children not possessing a depressogenic weakest link. To provide a powerful test of this hypothesis, we utilized a sample of parents with a history of major depressive episodes and their children. Given that a past history of major depressive episodes is one of the best predictors of future depressive episodes (e.g., Belsher & Costello, 1988), the use of a such a sample maximized the number of parents who experienced elevations in depressive symptoms during the course of the study. Further, given that children of parents with a history of major depressive episodes are four to six times more likely than other children to develop depressive episodes (Beardslee et al., 1993; Hammen et al., 1990; Weissman et al., 1997), the use of such a sample also maximized the number of children who experienced elevations in depressive symptoms during the course of the study.

To provide a powerful test of this hypothesis, we also utilized a multiwave longitudinal design in which children and parents’ levels of depressive symptoms were assessed at multiple time points over a 1-year follow-up interval. The use of such a design allowed us to take an idiographic, as opposed to a nomothetic, approach toward examining our hypothesis. The vast majority of previous research examining the diathesis-stress component of the hopelessness theory in youth has relied on two time-point designs in which (a) inferential styles and depressive symptoms are assessed during an initial assessment and (b) depressive symptoms and negative events are assessed during a follow-up assessment (Abela, 2001; Abela & Payne, 2003; Abela & Sarin, 2002; Conley et al., 2001; Dixon & Ahrens, 1992; Hammen, Adrian, & Hiroto, 1988; Hilsman & Garber, 1995; Robinson et al., 1995; Turner & Cole, 1994). Such a design necessitates the use of a nomothetic approach toward operationalizing high levels of stress. In other words, children are considered to be experiencing a high level of stress when their level of stress is higher than the sample’s average level of

stress. In contrast, the use of a multiwave longitudinal design, in which depressive symptoms and negative events are assessed repeatedly throughout the follow-up interval, allows for an idiographic approach toward operationalizing high levels of stress. In other words, children are considered to be experiencing a high level of stress when their level of stress is higher than their own average level of stress. This distinction is central to testing the diathesis-stress component of the hopelessness theory given that the theory posits that increases in levels of stress rather than absolute levels of stress will be associated with increases in depressive symptoms in cognitively vulnerable youth (for a more detailed discussion see Abela et al., in press).

The procedure involved an initial laboratory-based assessment in which children completed measures assessing inferential styles about the self, consequences, and causes. In addition, children and parents completed measures assessing depressive symptoms. The procedure also involved a series of follow-up assessments, every 6 weeks for the next year, in which children and parents completed measures assessing depressive symptoms. We hypothesized that a depressogenic weakest link would be associated with greater elevations in children's level of depressive symptoms following elevations in their parent's level of depressive symptoms.

Some previous studies examining the diathesis-stress component of the hopelessness theory in youth have found stronger support for the theory in girls than in boys (e.g., Abela, 2001, 2005). Further, Nolen-Hoeksema and Girgus (1994) have proposed that one factor that may account for the emergence of gender differences in depression during adolescence is that girls have higher levels of certain vulnerability factors prior to adolescence that increase the likelihood that they develop depression. Therefore, we examined whether the strength of the association between a depressogenic weakest link and elevations in children's depressive symptoms following elevations in their parents' levels of depressive symptoms varied as a function of children's gender. Similarly, as some researchers have hypothesized that depressogenic inferential styles only emerge as vulnerability factors to depressive symptoms during the transition from childhood to early adolescence when children acquire the ability to engage in abstract reasoning and formal operational thought (Nolen-Hoeksema et al., 1992; Turner & Cole, 1994), we examined whether the strength of any relations varied as a function of children's age.

Method

Participants

Participants were recruited through ads placed in local newspapers as well as through posters placed

throughout the greater Montreal area (additional details are provided in Abela, Hankin, et al., 2005; Abela, Skitch, Auerbach, & Adams, 2005). Two hundred fifty people responded to these ads. Respondents were invited to participate in a telephone interview during which a diagnostician administered the affective disorders affective disorders module of the Structured Clinical Interview for the *DSM-IV* (SCID-I; First, Gibbon, Spitzer, & Williams, 1995). One hundred thirty-three parents met criteria for either a current or past major depressive episode and were invited to participate in the study. Eighty-six parents, with 140 children in the appropriate age range, decided to participate.

The final sample consisted of 140 children (69 boys and 71 girls) and one of their parents (88 mothers and 14 fathers). Thirty-eight sibling pairs were included in the final sample. Children's ages ranged from 6 to 14 with a mean age of 9.8 ($SD = 2.37$). Parents' ages ranged from 27 to 53 with a mean age of 40.37 ($SD = 6.38$). The sample was 84.3% Caucasian. The mother tongue of participants included English (68.7%), French (9.8%), Spanish (2.9%), and other languages (18.6%). At the same time, all of the participants were fluent in English. Of the parents, 14.7% were single, 43.1% were married, 9.8% were separated, and 27.5% were divorced. The median family income ranged from \$30,000 to \$45,000. The highest level of education completed by the parents was an elementary school diploma for 7.8%, a high school diploma for 14.7%, a community college diploma for 39.3%, a bachelor's degree for 22.5%, and a graduate degree for 15.7%.

Procedure

Phase 1 of the study involved an initial laboratory assessment. Two research assistants met with one parent-child pair at a time. Parents completed a consent form and a demographics form. Children were told that their participation was voluntary and they could choose not to participate. All children decided to participate. During the initial assessment, a research assistant verbally administered the Children's Depression Inventory (CDI; Kovacs, 1981), the Children's Attributional Style Questionnaire (CASQ; Seligman et al., 1984), and the Children's Cognitive Style Questionnaire (CCSQ; Abela, 2001) to the child. Parents completed the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Phase 2 of the study involved a series of eight telephone follow-up assessments. Assessments occurred every 6 weeks during the year following the initial assessment. At each follow-up, a research assistant verbally administered the CDI to the child and the BDI to the parent. One hundred thirty-three children and their participating parent completed the Phase 2 assessments. The average number of follow-up assessments completed by participants was 4.79 ($SD = 2.13$).

The number of follow-up assessments completed was not significantly associated with the following Phase 1 variables: parental depressive symptoms ($r = .11$, *ns*), children's depressive symptoms ($r = .09$, *ns*), children's age ($r = -.17$, *ns*), children's gender ($r = -.01$, *ns*), or children's weakest link scores ($r = .06$, *ns*). The 7 children who did not complete any Phase 2 assessments did not significantly differ from the 133 children who completed assessments on any Phase 1 child or parent variables.¹

Phase 3 of the study occurred 1 year after Phase 1 and involved a final laboratory assessment during which a research assistant verbally administered the CASQ and CCSQ to the child. At the end of the assessment, participants were fully debriefed. Parents and children were compensated \$180 for time lost and expenses incurred while participating in the study. One hundred six children and their participating parent completed the Phase 3 follow-up assessment. The 34 children who did not complete the Phase 3 follow-up assessment did not significantly differ from the 106 children who completed this assessment on any Phase 1 child or parent variables.²

Measures

SCID-I (First et al., 1995). The SCID-I is a semi-structured clinical interview designed to arrive at current and lifetime *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) diagnoses. This study employed the affective disorders module and the psychotic screen. Diagnostic interviewers completed an intensive training program for administering the SCID-I and for assigning *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.) diagnoses. The training program consisted of attending approximately 40 hr of didactic instruction, listening to audiotaped interviews, conducting practice interviews, and passing regular exams (85% or above). The principal investigator held weekly supervision sessions. The principal investigator also reviewed interviewers' notes and tapes to confirm the presence or absence of a diagnosis. Discrepancies were resolved through consensus meetings and best estimate procedures. The SCID-I yields reliable diagnoses of depressive disorders (Zanarini et al., 2000) and is frequently used in clinical studies of depression in adults. The percentage of interrater agreement in terms of presence of a diagnosis between the principal investigator and diagnosticians was 97%. For the 3% cases in which the principal investigator did not agree with the diagnostician's diagnosis, the parent was not invited to participate in the study.

BDI (Beck et al., 1961). The BDI is a 21-item self-report measure that assesses severity of depressive symptoms within the past 2 weeks. Scores on each item range from 0 to 3, with higher scores indicating more severe symptoms. Total scores range from 0 to 63. Comparisons of the BDI with psychiatric rating of depression in clinical populations have shown it to have good concurrent validity (Beck et al., 1961). Furthermore, the BDI has been extensively employed across a variety of populations and has been found to have high internal consistency and moderate to high test-retest reliability (Beck, Steer, & Garbin, 1988).

CDI (Kovacs, 1981). The CDI is a 27-item self-report questionnaire that assesses the cognitive, affective, and behavioral symptoms of depression. For each item, children are asked whether it describes how they have been thinking and feeling in the past week. Total scores range from 0 to 52. The CDI possesses excellent internal consistency, adequate test-retest reliability, and sensitivity in distinguishing children with major depressive disorders from nondepressed children (Saylor, Finch, Spirito, & Bennett, 1984; Smucker, Craighead, Craighead, & Green, 1986). We obtained alphas ranging from .79 to .87 ($M = .83$) across administrations indicating moderate to high internal consistency.

CASQ (Seligman et al., 1984). The CASQ contains 48 items. Each item is a hypothetical event that children are asked to imagine happened to them. As the hypotheses of this study only involved children's attributional styles for negative events, only the 24 negative event items were used. For each event, respondents were presented with two possible causes and asked to choose which cause best described the way they would think if the event happened to them. The two causes held constant two attributional dimensions (internal-external, global-specific, and stable-unstable) while varying the third. In line with the hopelessness theory (Abramson et al., 1989), we operationalized a depressogenic attributional style as the tendency to attribute negative events to global and stable causes. Thus, total scores were equivalent to the sum of all global and stable responses with scores ranging from 0 to 16 and higher scores indicating a more depressogenic attributional style.

Seligman et al. (1984) found CASQ scores to be fairly consistent over a 6-month interval ($r = .66$, $p < .001$) in a sample of children between the ages of 8 and 13. Cronbach's alpha for the negative events composite score ranged from .50 and .54 across administrations indicating moderate internal consistency. Regarding validity, higher levels of depressive symptoms were associated with depressogenic attributions for negative events. Furthermore, a pessimistic attributional style predicted increases in depressive symptoms during the 6-month follow-up period. Several other studies using

¹Details on these specific analyses are available from John R. Z. Abela.

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the CASQ have obtained similar findings (e.g., Hillsman & Garber, 1995; Panak & Garber, 1992). In this study, we obtained an alpha of .52 at the initial assessment and .54 at the 1-year follow-up assessment. Test-retest reliability over the 1-year follow-up interval was .27 ($p < .01$).

CCSQ (Abela, 2001). The CCSQ is a two-part questionnaire. Each part contains 12 items, each of which is a hypothetical negative event involving the child. As with the CASQ, children are instructed to imagine that the event happened to them and then to choose the response that would best describe the way they would think. Part 1 assesses the tendency to catastrophize the consequences of negative events. In part 1, for each item, the child is given the following four choices: (a) This won't cause other bad things to happen to me, (b) this might cause other bad things to happen to me, (c) this will cause other bad things to happen to me, and (d) this will cause many terrible things to happen to me. Each response is assigned a value from 0 to 3 with higher scores indicating a greater tendency to catastrophize the consequences of negative events. Total scores are equivalent to the sum of all responses with scores ranging from 0 to 36. Part 2 assesses the tendency to view oneself as flawed or deficient following negative events. In part 2, for each item, the child is given the following three choices: (a) This does not make me feel bad about myself, (b) this makes me feel a little bad about myself, and (c) this makes me feel very bad about myself. Each response is assigned a value of 0 to 2 with higher scores indicating a greater tendency to view oneself as flawed or deficient following negative events. Total scores are equivalent to the sum of all responses with scores ranging from 0 to 24.

In a study examining the reliability and validity of the CCSQ in third and seventh graders, Abela (2001) found scores on both subscales of the CCSQ to be moderately consistent over a 7-week interval ($r = .46-.63$, $p < .001$). Cronbach's alphas for the two subscales ranged from .64 and .81 across administrations indicating moderate internal consistency. Regarding validity, higher levels of depressive symptoms were associated with more depressogenic inferential styles about consequences and the self. Last, depressogenic inferential styles about consequences and the self each interacted with negative events to predict increases in depressive symptoms. In this study, we obtained an alpha of .78 at the initial assessment and .71 at the 1-year follow-up assessment for the inferential style about consequences subscale of the CCSQ. For the inferential style about the self subscale, we obtained an alpha of .78 at the initial assessment and .72 at the 1-year follow-up assessment. Test-retest reliability over the 1-year follow-up interval was .26 ($p < .01$) for the inferential style about consequences

subscale and .42 ($p < .001$) for the inferential style about the self subscale.

Results

Examining Nonindependence in Data

Given that 38 pairs of siblings participated in this study, we conducted preliminary analyses examining whether nonindependence in our data impacted our findings. To do so, we first ran all analyses including only (a) the 64 children who did not have a sibling participating in the study and (b) the first child from each sibling pair to complete the assessment ($n = 38$). Next, we ran all analyses including only (a) the 64 children who did not have a sibling participating in the study and (b) the second child from each sibling pair to complete the assessment ($n = 38$). Last, we ran all analyses including all 140 children.³ The direction and magnitude of effects were similar in all three sets of analyses, suggesting that the inclusion of siblings in this study did not have a significant impact on the pattern of findings obtained. Thus, we used the entire sample of children ($n = 140$) in the analyses presented.

Descriptive Data

Means, standard deviations, and intercorrelations for all Phase 1 measures are presented in Table 1. Means and standard deviations for BDI and CDI scores across the eight follow-up assessments are presented in Table 2. As each child completed the CDI at multiple follow-up assessments, each child has his or her own mean level of depressive symptoms (i.e., his or her average CDI score during the follow-up interval) as well as his or her own degree of variation in depressive symptoms during the follow-up interval (i.e., his or her standard deviation on the CDI across administrations). Within-subject means on the CDI ranged from 0.00 to 29.00 ($\mu = 7.78$; $SD = 5.40$) whereas within-subject standard deviations ranged from 0.00 to 12.73 ($\mu = 3.05$; $SD = 2.13$). With respect to the parents, within-subject means on the BDI ranged from 1.33 to 40.17 ($\mu = 16.30$; $SD = 8.33$) whereas within-subject standard deviations ranged from 0.00 to 19.40 ($\mu = 6.47$; $SD = 3.75$).

To compute children's weakest link scores, we standardized scores on the generality subscale (i.e., sum of global and stable responses) of the CASQ and on the consequences and self subscales of the CCSQ. Each child's weakest link score was equal to the highest of his or her three standardized scores. At Phase 1, the difference between the standardized scores for the chil-

³Details on these specific analyses are available from John R. Z. Abela.

Table 1. Means, Standard Deviations, and Intercorrelations Between Children’s Gender, Age, and Phase 1 Measures

	1	2	3	4	5	6	7	8
1. CDI	—							
2. BDI	.23**	—						
3. CASQ	.45***	.30***	—					
4. CCSQ–CONS	.34***	.16	.43***	—				
5. CCSQ–SELF	.36***	.17	.28***	.60***	—			
6. WEAKLINK	.46***	.25**	.76***	.72***	.68***	—		
7. AGE	.07	.02	–.11	.01	–.07	–.09	—	
8. GENDER	–.06	.07	–.11	–.01	.20*	.03	–.11	—
<i>M</i>	10.08	19.17	4.31	12.56	9.99	0.71	9.80	0.51
<i>SD</i>	6.74	12.41	2.38	5.80	4.50	0.93	2.37	0.50

Note: CDI = Children’s Depression Inventory; BDI = Beck Depression Inventory; CASQ = Children’s Attributional Style Questionnaire, Generality subscale; CCSQ–CONSEQUENCES = Children’s Cognitive Style Questionnaire, Inferential Style about Consequences subscale; CCSQ–SELF = Children’s Cognitive Style Questionnaire, Inferential Style about the Self subscale; WEAKLINK = Children’s Most Depressogenic Inferential Style (e.g., highest of their standardized CASQ, CCSQ–CONSEQUENCES, and CCSQ–SELF scores). *df* = 139 for all Phase 1 correlations.

p* ≤ .05. *p* ≤ .01. ****p* ≤ .001.

Table 2. Means and Standard Deviations of Parental and Child Depressive Symptoms Over Eight Follow-Up Assessments

	1	2	3	4	5	6	7	8
BDI								
<i>M</i>	17.65	15.93	17.58	14.64	15.63	14.28	17.55	15.74
<i>SD</i>	11.27	10.38	9.74	9.46	11.29	9.20	11.62	9.49
CDI								
<i>M</i>	7.97	8.24	7.50	7.62	7.43	7.14	7.72	7.85
<i>SD</i>	6.00	6.98	5.48	6.01	6.04	5.74	6.14	6.21

Note: BDI = Beck Depression Inventory; CDI = Children’s Depression Inventory.

dren’s most depressogenic and least depressogenic inferential styles ranged from 0.10 to 3.30 ($\mu = 1.32$, $SD = 0.70$) with 27.9% of children exhibiting a difference greater than 1.75. Of the children who scored in the top quartile on at least one of the three measures assessing depressogenic inferential styles, 35.7% also scored in the bottom quartile on at least one of the other measures. Similar results were obtained at the 1-year follow-up assessment.⁴ Test–retest reliability for weakest link scores over the 1-year follow-up interval was .38 ($p < .001$).

The Diathesis-Stress Hypothesis

To test our hypothesis that children possessing a depressogenic weakest link would report greater elevations in depressive symptoms following elevations in parental levels of depressive symptoms than children not possessing a depressogenic weakest link, we utilized multilevel modeling. Analyses were carried out using the SAS (version 8.1) MIXED procedure and maximum likelihood estimation. Our dependent variable was within-subject fluctuations in CDI scores during the follow-up interval (FU_CDI). As FU_CDI is a

within-subject variable, CDI scores were centered at each participant’s mean such that FU_CDI reflects upward or downward fluctuations in a child’s level of depressive symptoms compared to his or her mean level of depressive symptoms during the follow-up interval. Our primary predictors of within-subject fluctuations in CDI scores were children’s age, gender, Phase 1 weakest link scores (WEAKLINK), and fluctuations in parental BDI scores during the follow-up interval (FU_BDI). As age and weakest link scores are between-subject predictors, these variables were standardized prior to analyses. As FU_BDI is a within-subject predictor, BDI scores were centered at each parent’s mean prior to analyses such that FU_BDI reflects upward or downward fluctuations in a parent’s level of depressive symptoms compared to his or her mean level of depressive symptoms during the follow-up interval.

For all analyses presented, preliminary models were also tested examining whether Phase 1 CDI scores served as a moderator of any relations. Such analyses test the assumption of homogeneity of covariance, which examines whether findings obtained apply equally across varying levels of initial symptom severity (i.e., onset and exacerbation of symptoms; Joiner, 1994). No significant interactions involving Phase 1 CDI scores were found. Consequently, for the sake of

⁴Details on these specific analyses are available from John R. Z. Abela.

simplicity, results are presented only for models including children's age, gender, and weakest link scores and fluctuations in parental BDI scores.

When fitting hierarchical linear models, one must specify appropriate mean and covariance structures. It is important to note that mean and covariance structures are not independent of one another. Rather, an appropriate covariance structure is essential to obtain valid inferences for the parameters in the mean structure. Overparametrization of the covariance structure can lead to inefficient estimation and poor assessment of standard errors (Altham, 1984). On the other hand, too much restriction of the covariance structure can lead to invalid inferences when the assumed structure does not hold (Altham, 1984). Diggle, Liang, and Zeger (1994) recommended that one use a "saturated" model for the mean structure while searching for an appropriate covariance structure.

We were interested in examining the effects of children's age, gender, and weakest link scores and fluctuations in parental levels of depressive symptoms on children's CDI scores during the follow-up interval. Consequently, we chose a mean structure that included children's gender, children's age, children's weakest link scores, fluctuations in parents' BDI scores during the follow-up interval, and all two-, three-, and four-way interactions. Four additional effects were also included in this initial mean structure. First, to control for individual differences in baseline levels of depressive symptoms, children's Phase 1 CDI scores were included in the model. Second, to account for the possible correlation in response variables between siblings from the same family, random effects for children nested within families were included in the model. Last, given that fluctuations in parental BDI scores is a within-subject predictor whose effect is expected to vary from participant to participant, a random effect for slope was included in the model.

Using this saturated mean structure, we searched for the most appropriate covariance structure to use in our analyses. Commonly used covariance structures in studies in which multiple responses are obtained from the same individual over time (and consequently within-subject residuals over time are likely to be correlated) include compound symmetry, first-order autoregressive, heterogeneous autoregressive, and banded Toeplitz. To select one of these covariance structures for our analyses, we fitted models utilizing each structure and chose the "best" fit based on Akaike information criterion and Schwarz Bayesian criterion. In all cases, the best fit was a heterogeneous autoregressive structure. Such a covariance structure indicates two general patterns in CDI scores during the follow-up interval (for further explanation see Littell, Pendergast, & Natarajan, 2000). First, as the interval between any two follow-up assessments increases, the degree of intercorrelation between children's CDI scores at these

two time points decreases (i.e., CDI scores obtained at follow-up 4 are more strongly associated with CDI scores obtained at follow-ups 3 and 5 than with CDI scores obtained at follow-ups 2 and 6). Second, the variance in CDI scores across administrations is not constant (i.e., variance in CDI scores is greater at some follow-up assessments than at others).

After choosing the appropriate covariance structure, we next examined the random-effects component of our model. Nonsignificant random-effect parameters were deleted from the model prior to examining the fixed-effects component. With respect to random effects, the random effect for children was significant ($p < .01$) and thus was retained in the model. Although the random effect for families was not significant, it was retained in the model to account for any non-independence of data due to inclusion of siblings in the study. Last, the random effect for slope was not significant and consequently was deleted from the model prior to examining the fixed effects.

As mentioned previously, a saturated model was initially tested. The four-way interaction Gender \times Age \times WEAKLINK \times FU_BDI was not significant ($\beta = -.015$, $SE = .052$, $F(1, 493) = .09$, ns). Analyses were therefore repeated excluding the four-way interaction. Three of the three-way interactions, Gender \times Age \times WEAKLINK, $\beta = 0.26$, $SE = .779$, $F(1, 120) = .11$, ns ; Gender \times Age \times FU_BDI, $\beta = -.013$, $SE = .046$, $F(1, 494) = .08$, ns ; and Age \times WEAKLINK \times FU_BDI, $\beta = .05$, $SE = .028$, $F(1, 494) = 3.34$, ns , were not significant. Analyses were therefore repeated excluding these nonsignificant three-way interactions.

Results with respect to the fixed-effects component of the final model are presented in Table 3. Of primary importance, a significant three-way, cross-level interaction emerged between gender, weakest link scores, and fluctuations in parental depressive symptoms during the follow-up interval. As recommended by Holmbeck (2002), to examine the form of the Gender \times WEAKLINK \times FU_BDI interaction, the model summarized in Table 2 was used to calculate predicted CDI scores for both boys and girls either possessing or not possessing a depressogenic weakest link (plus or minus $1.5 \times$ between-subject standard deviation) whose parents were experiencing low or high levels of depressive symptoms in comparison to their own average level of depressive symptoms (minus or plus $1.5 \times$ mean within-subject standard deviation). The results are presented in Figure 1. As both FU_CDI and FU_BDI are within-subject variables centered at each participant's mean, slopes are interpreted as the increase in a child's CDI score that would be expected given that his or her parent scored 1 point higher on the BDI.

Analyses were conducted for each Gender \times WEAKLINK condition examining whether the slope of the relation between parental depressive symptoms

Table 3. Age, Gender, Depressogenic Weakest Link, and Parental BDI Scores Predicting Depressive Symptoms

Predictor	<i>b</i>	<i>SE</i>	<i>F</i>	<i>df</i>
Phase 1 CDI	3.59	0.40	80.66****	1,121
AGE	0.33	0.49	0.47	1,121
GENDER	0.79	0.66	1.44	1,121
WEAKLINK	-0.51	0.51	0.98	1,121
FU_BDI	0.07	0.03	4.76*	1,496
AGE × WEAKLINK	-0.35	0.38	0.87	1,121
AGE × FU_BDI	0.06	0.02	7.92**	1,495
WEAKLINK × FU_BDI	0.03	0.02	1.40	1,496
GENDER × WEAKLINK	0.22	0.70	0.11	1,121
GENDER × FU_BDI	0.01	0.04	0.00	1,496
AGE × GENDER	0.35	0.72	0.24	1,121
GENDER × WEAKLINK × FU_BDI	0.08	0.04	3.74*	1,496

Note: Phase 1 CDI = Phase 1 Children’s Depression Inventory; WEAKLINK = weakest Link scores; FU_BDI = fluctuations in parents’ Beck Depression Inventory scores.

p* ≤ .05. *p* ≤ .01. *** *p* ≤ .001. *****p* ≤ .0001.

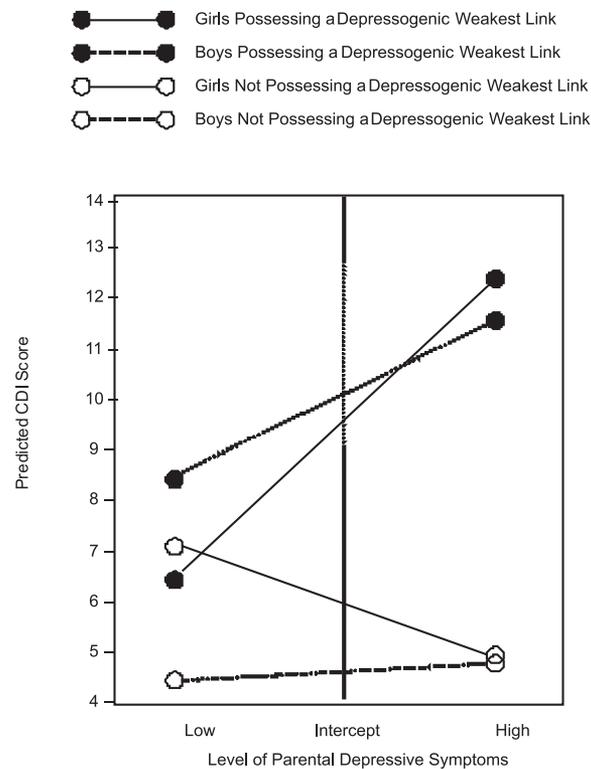


Figure 1. Predicted slope of the relation between parental depressive symptoms and depressive symptoms for boys and girls possessing depressogenic or nondepressogenic weakest links.

and children’s depressive symptoms significantly differed from zero. Analyses indicated that girls possessing a depressogenic weakest link reported higher levels of depressive symptoms when their parent was experiencing high but not low levels of depressive symptoms in comparison to their own average level of depressive symptoms, $t(495) = 4.39, p < 0.001$. Also, boys possessing a depressogenic weakest link reported higher levels of depressive symptoms when their parent was experiencing high but not low levels of depressive symptoms in comparison to their own average level of

depressive symptoms, $t(495) = 2.45, p < 0.05$. Level of depressive symptoms did not vary as a function of level of parental depressive symptoms for either (a) girls not possessing a depressogenic weakest link, $t(495) = -1.83, ns$, or (b) boys not possessing a depressogenic weakest link, $t(495) = 0.71, ns$. Follow-up analyses indicated that the relation between parental and child depressive symptoms was significantly greater in girls possessing a depressogenic weakest link (slope = 0.27) than in boys possessing a depressogenic weakest link (slope = 0.11), $t(495) = 2.11, p < .05$.

Discussion

The results of this study provide support for our hypothesis that children possessing a depressogenic weakest link would report greater elevations in depressive symptoms following elevations in their parents’ levels of depressive symptoms than children not possessing a depressogenic weakest link. In addition, consistent with previous research examining the diathesis-stress component of the hopelessness theory in youth using a weakest link approach (Abela & Payne, 2003; Abela & Sarin, 2002), the strength of this association was not moderated by age. At the same time, gender differences emerged that warrant attention.

A depressogenic weakest link was associated with greater elevations in children’s depressive symptoms following elevations in parental depressive symptoms in both boys and girls. At the same time, the strength of this association was significantly greater in girls. Although research examining the diathesis-stress component of the hopelessness theory in children and early adolescents has failed to obtain consistent gender differences (e.g., Abela, 2001; Abela & Payne, 2003; Abela & Sarin, 2002; Conley et al., 2001; Hankin, Abramson, & Siler, 2001; see Hankin & Abela, 2005,

for a recent review), research examining the association between maternal depression and child depression has obtained relatively consistent gender differences with maternal depression having a greater impact on girls than boys. For example, several studies have reported that maternal depression is associated with depressive symptoms in adolescent girls but not adolescent boys (e.g., Davies & Windle, 1997; Hops, 1992, 1996; Thomas & Forehand, 1991). Similarly, Radke-Yarrow, Nottelman, Belmont, and Welsh (1993) reported a temporal association between negative affect in depressed mothers and their daughters but not in depressed mothers and their sons.

Sheeber, Davis, and Hops (2002) offered several possible explanations for increased risk for depression among daughters of affectively ill parents. For example, one explanation is that during adolescence, mother–daughter relationships contain higher levels of conflict than mother–son relationships (e.g., Steinberg, 1987, 1988). Consequently, daughters of depressed mothers may be exposed to higher levels of aversive interactions than are sons of depressed mothers. A second explanation is that socialization mechanisms may account for girls' greater degree of risk because girls are socialized to be more relationship and family oriented (e.g., Slavin & Rainer, 1990) and consequently may be more sensitive to parental conflict than boys. Future research is needed examining the mechanisms that account for girls' greater sensitivity to parental depression. It is important to emphasize, however, that although these results suggest that cognitively vulnerable girls are more sensitive to the deleterious impact of parental depressive symptoms than cognitively vulnerable boys, parental depressive symptoms increased the risk for elevations in depressive symptoms in both girls and boys. Nevertheless, this finding, combined with research showing that adolescent girls exhibit higher levels of cognitive vulnerability than adolescent boys (Hankin & Abramson, 2001), may provide some explanation as to why girls begin to exhibit higher depression rates than boys during adolescence (Hankin et al., 1998).

Several limitations of this study should be noted. First, self-report measures were used to assess depressive symptoms in both the children and their parents. Although the CDI and the BDI both possess high degrees of reliability and validity, it is difficult to make conclusions about clinically significant depression based on self-report questionnaires. Future studies should utilize semistructured diagnostic interviews to see if these findings extend to the onset of clinically significant depressive episodes. Second, this study utilized a high-risk design. Although such a design allowed for a powerful test of our hypotheses by maximizing the number of children and parents who exhibited elevations in depressive symptoms during the course of the study, results cannot be generalized to

low-risk samples. Third, the internal consistency of the study's measure of attributional style, the CASQ, was low. As low internal consistency can attenuate correlations between measures, future research should attempt to replicate these results using an alternate measure of children's attributional style that possesses stronger psychometric properties. Fourth, children's weakest link scores only showed moderate stability over the 1-year follow-up interval indicating substantial change occurred in many children's inferential styles. Future research is needed identifying the factors that predict both stability and change in inferential styles throughout development. Fifth, the majority of participants in this study were Caucasian and from middle to upper-middle socioeconomic background. Future research is needed to examine the generalizability of these findings to individuals from different cultural and socioeconomic groups. Sixth, although this study examined whether a depressogenic weakest link moderates the relation between elevations in children's depressive symptoms following elevations in parental symptoms, future research is needed examining the mechanisms and processes that mediate this relation. Potential mediators may include parental withdrawal and irritability (Cohn & Campbell, 1992), dysfunctional parenting practices (Fendrich et al., 1990), and marital conflict (Beach et al., 1994). Last, this study only examined the relation between a depressogenic weakest link, parental depressive symptoms, and child depressive symptoms. Thus, we were unable to identify whether the interaction of this cognitive vulnerability factor with parental depressive symptoms is specific to child depressive symptoms rather than broadly applicable to other disorders.

In sum, the results of this study suggest that the hopelessness theory may prove to be a useful framework for understanding individual differences in vulnerability to depressive symptoms in children of affectively ill parents. Identifying the factors that confer vulnerability to depression among high-risk youth is likely to be beneficial in guiding clinicians and researchers in designing effective treatment and prevention programs for such youth. Given the high level of risk for developing depression among such children, research in this area is desperately needed.

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Received February 7, 2005

Accepted December 15, 2005