Published in final edited form as:

J Early Adolesc. 2011 December ; 31(6): 782–816. doi:10.1177/0272431610376248.

A Longitudinal Study of Cognitive Risks for Depressive Symptoms in Children and Young Adolescents

David A. Cole¹, Farrah M. Jacquez¹, Beth LaGrange¹, Ashley Q. Pineda¹, Alanna E. Truss¹, Amy S. Weitlauf¹, Carlos Tilghman-Osborne¹, Julia Felton¹, Judy Garber¹, Danielle H. Dallaire¹, Jeff A. Ciesla¹, Melissa A. Maxwell¹, and Lynette Dufton¹

¹Vanderbilt University, Nashville, TN

Abstract

Youths with high ($N = 52$) or low cognitive vulnerability ($N = 48$) for depression were selected from a larger sample ($N = 515$) of students (7-10 years old), based on their attributional style (AS), negative cognitions (NC), and/or self-competence (SC). Long-term effects of cognitive vulnerabilities on depressive symptoms were examined in a 3-year, three-wave, multiinformant, longitudinal design. Three findings emerged. First, some empirical overlap exists among these three types of cognitive diatheses, especially between NC and SC. Second, the combination of AS, NC, and SC had a significant (but diminishing) relationship to depressive symptoms at 6, 18, and 30 months, primarily due to NC and SC, not AS. Third, interactions between cognitive risk and life events were not significant, suggesting an additive type of diathesis-stress model for depression in young adolescents.

Keywords

children; depression; attributional style; negative cognitions; competence

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Corresponding Author: David A. Cole. PhD, Psychology & Human Development, Vanderbilt University Peabody, 0552 GPC, 230 Appleton Place, Nashville, TN 37203-5721 david.cole@vanderbilt.edu.

Declaration of Conflicting Interests

The author(s) declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

¹The entire protocol for the subsample was both expensive and time consuming. Our funding provided resources only for the in-lab testing and clinical interviewing of the individuals who were of highest and lowest risk.

²Consequently, we conducted two sets of analyses one with and one without controls for race/ethnicity and income. For ethnicity, we created two new dummy-coded variables, representing whether the child was White, African American, or Other. These and family income served as global covariates. The pattern of significance/nonsignificance remained completely unchanged, and the magnitude of every effect changed by an amount of .01 or less. The results reported in the text therefore represent the analyses without these covariates. We also conducted analyses with and without controls for child gender as a global covariate. Again the results were essentially unchanged.

³Recentering time means that the intercept shifts from wave to wave. Thus the prediction of intercepts refers to the prediction of the dependent variable at different waves, depending on the centering of time, as in Table 7.

⁴We also tested for age as a possible moderator. A two-group model (older vs. younger) fit the data well, but cross-group comparisons of all model parameters revealed no significant differences. We caution against the overinterpretation of these (null) results, however, in that the age range in any given wave of the study was relatively narrow.

⁵We speculate that this phenomenon is the result of the within-wave distribution of depression scores coupled with classic regression to the mean, a phenomenon not unlike the pattern described by Twenge and Nolen-Hoeksema (2002).

Several cognitive theories of depression have been developed for or applied to young adolescents. Among these are Abramson, Seligman, and Teasdale's (1978) attributional style (AS) model, Beck's (1963) cognitive model, and Cole's (1991) competence model. The kinds of cognitive risk featured in these models contain both unique and overlapping features. Nevertheless, longitudinal studies of depressive symptoms in young adolescents have not yet examined these types of risk simultaneously. The goal of the current study was to examine the conjoint and unique contributions of AS, depressive cognitions, and self-perceived competence (SC) in conveying risk for the emergence of depressive symptoms in young adolescents.

These three types of cognitive risk have specific definitions and derive from distinct theories. First, the reformulated helplessness theory (Abramson et al., 1978) asserts that stressful life events are more likely to induce depression in individuals with a specific type of causal AS. This style is the tendency to attribute negative life events to internal, stable, and global causes. Second, Beck's model posits that preexisting negative cognitive schemas adversely affect perceptions of self, world, and future and foster dysfunctional automatic thoughts (Beck, 1963, 1964; Clark & Beck, 1999). Finally, Cole's competence model (1991) suggests that the most critical cognitive risk in childhood and early adolescence is the individual's sense of personal incompetence in multiple domains, a viewpoint constructed out of negative feedback from significant others (e.g., Cole, Martin, & Powers, 1997).

Longitudinal tests of these models in early adolescent populations have been somewhat infrequent, and the results have been mixed. The reformulated helplessness theory (Abramson et al., 1978) has received the most empirical attention. A recent meta-analysis by Lakdawalla, Hankin, and Mermelstein (2007) focused on Abramson, Metalsky, and Alloy's (1989) hopelessness model of depression; however, most of the reviewed studies also afforded tests of the reformulated helplessness model. Approximately half of these tests found significant longitudinal effects of an Attributional style $\times$ Stressful life events interaction after controlling for prior levels of depressive symptoms (e.g., Hilsman & Garber, 1995). On average, the effect size was relatively small. Some evidence suggested that the effect is moderated by age. In keeping with developmental theory, the effects were somewhat larger in early adolescence than in childhood (see also Cole et al., 2008; Cole & Turner, 1993; Gibb & Coles, 2005). Most of these results were qualified, however, by the preponderant use of relatively weak measures of AS, especially at younger ages.

Beck's (1963) model has been examined extensively in adult populations; however, longitudinal tests of the diathesis-stress interaction in young adolescent populations have been relatively rare. Our review revealed only two such studies (the same two identified in Lakdawalla et al.'s 2007 review). First, Lewinsohn, Joiner, and Rohde's (2001) examination of high school students in the Oregon Adolescent Depression Project found a marginally significant longitudinal effect for a Dysfunctional attitudes $\times$ Stressful events interaction in the 1-year prediction of depression. After controlling for prior depression and a number of other covariates, the effect size for this interaction was quite small. Second, in a large sample of seventh graders, Abela and Sullivan (2003) found a small but significant longitudinal effect for the Dysfunctional attitudes $\times$ Stressful life events interaction on depressive symptoms over a 6-week interval. Qualifying this result, however, is the fact that

it was only significant for youth with high self-esteem and high social support. Overall, very few longitudinal studies have tested Beck's model in younger populations, and the results are not strong among those that did.

Cole's (1991) model has generally been supported by longitudinal research. In several large-scale longitudinal studies, Cole and colleagues provided evidence that self- (and others') perceptions of incompetence in multiple domains are associated with subsequent increases in depressive symptoms and negative affect (Cole et al., 1997; Cole, Jacquez, & Maschman, 2001; Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; Cole, Martin, Powers, & Truglio, 1996; Cole, Peeke, Dolezal, Murray, & Canzoniero, 1999; Hoffman, Cole, Martin, Tram, & Seroczynski, 2000; Tram & Cole, 2000). Testing other aspects of Cole's model, Seroczynski, Cole, and Maxwell (1997) found that significant others' positive evaluations in multiple domains have a cumulative negative relationship to depressive symptoms, and that negative evaluations have a cumulative positive relationship to depressive symptoms. Moreover, positive evaluations in one domain somewhat compensate for negative evaluations in another domain, and negative evaluations in one domain offset positive evaluations in another domain. Similar results have emerged from other labs (e.g., Brendgen, Vitaro, Turgeon, Poulin, & Wanner, 2004; Crockett, Moilanen, Raffaelli, & Randall, 2006; Ohannessian, Lerner, Lerner, & von Eye, 1999; Reinherz et al., 1989; Roeser, Eccles, & Sameroff, 1998). In a few studies, however, the model was either partially supported (Kistner, David-Ferdon, Lopez, & Dunkel, 2007; Mitchell, Murdock, & Berz, 2004) or not supported at all (McGrath & Repetti, 2002).

Two things are conspicuous for their absence from these literatures. One is any attempt to examine the combined effects of these three putative cognitive diatheses. It is possible that different individuals develop somewhat different kinds of cognitive vulnerabilities. Abela and Sarin (2002) raised a similar point in their weakest link hypothesis, although their work focuses primarily on the hopelessness model of depression (Abela & McGirr, 2007; Abela, McGirr, & Skitch, 2007). Along similar lines, however, the conjoint examination of AS, negative cognitions (NC), and self-perceived (in)competence could yield stronger results than studies that examine only one diathesis or another. The second is any attempt to compare these models to one another. Without such comparisons, knowing which type of cognition is more characteristic of depression during childhood and early adolescence is difficult. Also unclear is the degree to which these three constructs truly represent distinct cognitive diatheses. Conjoint analyses will reveal the degree to which they are empirically redundant of one another.

Consideration of developmental theory and research gives rise to several hypotheses. First, recent evidence suggests that AS does not become particularly "style-like" or predictive of depressive symptoms until early adolescence (Cole et al., 2008). Developmental research further suggests that children may not understand the internality, stability, and globality of attributions in the same way as do early adolescents (Folmer et al., 2008; Nicholls, 1978; Nicholls & Miller, 1983; Nicholls, Patashnick, & Mettetal, 1986). Nevertheless, researchers continue to apply the AS model of depression to children and young adolescents, without apparent regard for the level of cognitive development needed to have this kind of AS. Consequently, during the transition into adolescence that is the focus of the current study,

we do not expect AS to be particularly correlated with other kinds of depressogenic cognitions or particularly predictive of depressive symptoms. Second, the content of Beck's cognitive triad is not particularly triadic in youth. LaGrange et al. (2008) demonstrated that child and adolescent thoughts about self, world, and future are better conceptualized as generalized collections of negative versus positive thoughts. Given the egocentrism and self-focus of youth (Elkind, 1978), we suspect that positive and negative views of "self" are at the heart of these dimensions. As such, we hypothesize that Beck's measure of such cognitions will predict the emergence of depressive symptoms. Third, given that the construction of self is such a key developmental task during this age range (Harter, 1988; Marsh, 1990), we hypothesize that SC represents the most developmentally relevant constellation of self-cognitions. Furthermore, we hypothesize that SC will be strongly predictive of depressive symptoms, potentially supplanting a substantial part of Beck's more generalized NC in youths in this age range.

The purpose of the current study was to examine simultaneously the predictive utility of AS, NC, and SC in relation to depressive symptoms in late childhood and early adolescence. In accordance with our cross-sectional hypotheses, we began by examining the correlation between these types of cognitions. To test our prospective hypotheses, we tested both the main effects of these cognitive diatheses and their interactions with negative life events as predictors of depressive symptoms over time. As part of this effort, we also examined changes in the relationship of these cognitive diatheses to depressive symptoms as a function of age. The current sample consists of a relatively high proportion of minority, low-socioeconomic status (SES) youths. A few studies have suggested that at least Beck's automatic thoughts or cognitive triad may not be as reflective of depressive symptoms among African American youths, compared to Latino or White youths (Cardemil, Reivich, Beevers, Seligman, & James, 2007; Greening, Stoppelbein, Dhossche, & Martin, 2005). However, Dallaire et al. (2008) found that AS and low levels of SC predicted depressive symptoms in a child and adolescent sample that was predominantly African American, even after controlling for parental education and negative parenting behaviors. In light of these findings but without strong hypotheses, we examine race/ethnic differences, primarily to judge the generalizability of our results across ethnicities.

Method

Overview

This study is a part of a larger longitudinal investigation of the developmental origins of depressive cognitions in youth (see Cole et al., 2008). At the beginning of the larger study, we obtained measures of the kinds of cognitive risk implicated by Abramson et al.'s (1978), Beck's (1963), and Cole's (1991) theories of depression. This larger screening sample consisted of 515 public elementary school students, ages 7 through 10 at the beginning of the study ($M = 8.5$, $SD = 1.2$). Based on the cognitive risk data from the screening sample, we obtained high-risk (HR) and low-risk (LR) subsamples of youth. Stated generally (and described in more detail in the Measures and Procedures sections below), HR participants were those who scored high on at least one of the three cognitive risk dimensions and scored low on none of these dimensions. Conversely, LR participants were those who scored low

on at least one of the three cognitive risk dimensions and scored high on none of these dimensions. Separate active parental consents were obtained for inclusion in the larger (screening) study and the smaller HR/LR follow-up study, which is the primary focus of this investigation. After screening, the HR/LR youths and a parent or guardian participated in three waves of data collection. Waves 1, 2, and 3 occurred 6, 18, and 30 months after the initial screening (with 1-year lags between adjacent waves).

Participants

Screening sample participants—For our screening sample, we recruited 515 students from 5 elementary and 2 middle schools in a major southeastern metropolitan area. Overall, the screening sample was ethnically diverse: 69% African American, 28% White, 1% Hispanic, 1% multiethnic, and 1% Other. The sample was 45% male and 55% female. The participants had an average of 2 siblings (range = 0-13). Approximately 24.5% had at least one parent who was currently or previously divorced. The families were of relatively low SES with an average family income between US\$20,000 and US\$30,000 per year (range = US\$0-US\$84,000). The majority of families reported receiving some type of public assistance in the past year, including welfare (39%), public housing (36%), and food stamps (57%). Participants in this sample lived in neighborhoods in which 27.3% of the families fell below the 2004 poverty threshold established by the U.S. Census Bureau.

Repeated residential relocation was common for our participants. Some students had as many as six different home addresses and attended as many as five different schools over the course of a single academic year. School principals reported that only 30% to 40% of the students finished an academic year in the school where they started. Tracking participants over time required repeated mailings and phone calls to parents and extended family members. We examined school records, conducted web searches, and consulted city and school databases to track participants. Although we collected 92% of the screening data on site at the participating schools, we also provided cab fare for families to come to the university when they had moved to nonparticipating school districts.

HR/LR subsample of participants—The subsample consisted of 100 students: 52 HR and 48 LR.¹ For selection details, see Measures and Procedures sections below. When selecting HR participants into the subsample, we recruited all who met our criteria. When selecting LR participants, we invited a sample that had the same mean age, sex ratio, and ethnic breakdown as the HR sample. In total, 10% of those invited did not participate (see Procedures), resulting in some differences between the HR and LR groups on ethnicity and income. Our HR group had slightly more African Americans and had a median family income US\$5,000 lower than our LR group (see Table 1).²

Measures

During the screening process, we obtained self-reports of all three cognitive diatheses for depression. For each of the next 3 years, we obtained measures of depressive symptoms by youth self-report, parent report, and clinical interview. We also obtained yearly measures of negative life events by youth self-report and parent report.

Cognitive diatheses—We measured three cognitive diatheses for depression (AS, NC, and SC) using four measures. We obtained information on participants' attributions using the Children's Attributional Style Interview (CASI; Conley, Haines, Hilt, & Metalsky, 2001). The CASI is a semistructured interview of AS in youth. The current study used only the eight negative scenarios. These scenarios described four social/interpersonal events (e.g., "You're lining up for lunch and you get pushed") and four academic/achievement events (e.g., "You do a math worksheet, but you get a lot wrong"). Respondents provide a causal explanation for each hypothetical event and then rate (on 7-point Likert-type scales) the degree to which this cause is internal, stable, and global. Interviewers used large visual aids, depicting different degrees of internality, stability, or globality. Unlike the original CASI, we added a 1 (*low*) to 7 (*high*) numeric scale to these visual aids to facilitate scoring. Three scale scores (internality, stability, and globality) consisted of the sum of the corresponding questions over the eight scenarios. Scores on each scale could range from 8 to 56, with higher scores representing higher levels of the construct. Conley et al. (2001) reported internal consistency for the CASI subscales ranging from .78 to .83 for elementary school students. Dallaire et al. (2008) reported similar psychometric characteristics in a predominantly African American sample. In the current study, Cronbach's alpha for the CASI was .82.

To measure participants' NC, we used two measures, the Children's Automatic Thoughts Questionnaire (CATQ; Hollon & Kendall, 1980) and the Cognitive Triad Interview for Children (CTI-C; Kaslow, Stark, Printz, Livingston, & Tsai, 1992). The CATQ is a 30-item questionnaire that assesses negative automatic thoughts. Respondents rate how often, in the past week, they have had specific negative thoughts, using 5-point scales (1 = *not at all* to 5 = *all the time*). Scores range from 30 to 150 with higher scores representing higher levels of negative automatic thoughts. This measure has good internal consistency in White and minority populations (Cronbach's alpha range from .94 to .96; Cardemil et al., 2007; Kazdin, 1990). In our sample, Cronbach's alpha for the CATQ was .93. The CTI-C is a 36-item self-report questionnaire assessing youths' views of themselves (e.g., "I am a failure"), their world (e.g., "The world is a very mean place"), and their future (e.g., "Nothing is likely to work out for me"). Respondents indicate having had specific thoughts, using a *yes/maybe/no* format. Scores range from 0 to 72 with higher scores representing more negative views. The CTI-C has acceptable internal consistency with Cronbach's alphas ranging from .86 to .94 for the full scale (Kaslow et al., 1992; LaGrange et al., 2008). The measure has been validated for use with African American youths (Greening et al., 2005). In the current sample, Cronbach's alpha for the CTI-C was .94.

We evaluated five domains of self-concept with Harter's (1985) Self-Perception Profile for Children (SPPC).

This self-report inventory contains 36 items measuring five domains of SC (scholastic competence, social acceptance, athletic competence, physical appearance, and behavioral conduct) and global self-worth. For each item, respondents select one of two statements to indicate whether they are more like someone who is good or someone who is not so good at a particular activity. Then they select statements indicating whether the selected statement is "sort of true" or "really true" about them. Responses are then converted to 4-point rating

scales with high scores reflecting greater SC. The SPPC has a highly interpretable factor structure, and all subscales have good internal consistency in predominantly White samples (Harter, 1985). In African American samples, domain-specific factors are less differentiated (Schumann et al., 1999; Thomson, & Zand, 2002). Therefore, we represented SC as the sum of the five domain-specific subscales. The scales have good internal consistency, with Cronbach's alphas ranging from .71 to .86 for students in Grades 3 through 7 (Harter, 1985) as well as good test-retest reliability (.70 to .87; Harter, 1982). In our sample, the reliability for the composite was .86.

Depressive symptoms—We obtained measures of depressive symptoms by clinical interview, self-report, and parent report. The clinical interview was the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Present and Lifetime (K-SADS-PL; Kaufman et al., 1997). This semistructured clinical interview was administered first to parents and then to the youths. The K-SADS-PL assesses current and past symptoms of many disorders including depression. Interviewers completed extensive training, were regularly supervised by a doctoral level psychologist, and were naïve to participants' risk status. Interviews were videotaped; of these, 20% were independently rerated by an experienced interviewer. Interrater kappas indicated acceptable interrater reliabilities for each symptom of depression averaging .87 for White and .89 for African American youths.

Level of depressive symptoms was also assessed using the Child Depression Rating Scale (CDRS-R; Poznanski, Freeman, & Mokros, 1984), a clinical interview modeled after the Hamilton Depression Rating Scale for adults (Hamilton, 1960). The CDRS-R assesses 17 symptom areas relevant to depression. These items are similar to the depression items on the K-SADS but pertain only to the last 2 weeks. We conducted the interviews first with the parent and then with the child/adolescent. All CDRS-R interviews were videotaped and 20% were rerated by an experienced rater. Interrater reliabilities were .90 for the CDRS-C and .94 for the CDRS-P. In a manner consistent with the procedure for scoring the K-SADS, we developed a parent-youth composite for the CDRS-R by scoring each item according to the informant that the interviewer indicated provided the more reliable information. We then summed these items. Cronbach's alphas for the composite were .75 for Whites and .79 for African Americans.

The self-report was the Children's Depression Inventory (CDI; Kovacs, 1985), a 27-item self-report measure that assesses cognitive, affective, and behavioral symptoms of depression in youth. Each item consists of three statements graded in order of increasing severity from 0 to 2. Respondents select one sentence from each group that best describes themselves for the past 2 weeks (e.g., "I am sad once in a while," "I am sad many times," or "I am sad all the time"). In nonclinic populations, the CDI has relatively high levels of internal consistency, test-retest reliability, predictive, convergent, discriminant, and construct validity (Cole & Jordan, 1995; Craighead, Smucker, Craighead, & Ilardi, 1998; Smucker, Craighead, Craighead, & Green, 1986; Timbremont, Braet, & Dreesen, 2004). Similar characteristics have been reported in African American samples (Cole, Martin, Peeke, Henderson, & Harwell, 1998). Cronbach's alpha for the CDI in the current study was .78.

The parent report was a version of the CDI reworded for use by parents about their child or adolescent's depressive symptoms (CDI-P; Wierzbicki, 1987). This method is commonly used (e.g., Garber, 1984; Tram & Cole, 2006), has a highly interpretable factor structure, and demonstrates good reliability (Cole, Hoffman, Tram, & Maxwell, 2000). In the current study, Cronbach's alpha was .83. Correlations between the parent and youth forms of the CDI were low (0.20 to 0.30). Examination of discrepancies item-by-item revealed two patterns: (a) youths sometimes reported symptoms that the parents had not noticed and (b) parents reported that their children had symptoms that the youths themselves denied. In a manner consistent with the aggregation of parent and child information in the K-SADS and in the CDRS-R, we examined parent and youth responses to each CDI item, taking the higher of the two for each item, and then summed these item scores. Alpha reliabilities for the composite ranged from .82 to .83.

The CDI composite and the CDRS-R composite correlated 0.56, 0.66, and 0.57 with each other in Waves 1, 2, and 3, respectively. We standardized each measure (using its grand mean and pooled *SD* across waves) and summed them within wave to create a composite index of depressive symptoms for each wave. We used this composite depression index as the dependent variable in the following analyses.

Negative life events—We used both a youth- and parent-report version of the Life Events and Circumstances (LEC), a checklist consisting of 30 negative life events, adopted from the checklist created by Work, Cowen, Parker, and Wyman (1990) and validated in large, ethnically diverse samples of children and adolescents. Representative items are “Your family had to move a lot” and “A close family member was arrested or in jail.” Items on this checklist range from medium to major life events. Minor events (or daily hassles) were not included. Respondents indicate whether or not the youth has been exposed to each of the events in the past 6 months using a *yes/no* format. Each event was regarded as present if either the youth or the parent so indicated. We chose this life events measure because it was developed for inner city, low SES youth, and contained a high concentration of relevant items given the demographics of our sample. In the current study, means were 9.43 (*SD* = 4.67) on the self-report and 5.67 (*SD* = 4.37) on the parent report, very comparable to the means reported for other urban youth populations (Cowen et al., 1992; Work et al., 1990; Wyman, Cowen, Work, & Parker, 1991). One-week test-retest reliability was .78.

On the youth version, we added an additional component to the checklist. If the respondent endorsed an item, they were asked how upsetting the event was using a 3-point scale (1 = *not much* to 3 = *very much*). A nomothetic impact weight was derived for each event, based on the average of upset ratings for all of the 515 screening participants who had experienced that event. Presence (1) or absence (0) of each event was then multiplied by its nomothetic impact weight. These 30 products were summed to form a composite score. This method avoids the bias that derives from using idiographic weights, which are known to correlate with concurrent levels of depression (Monroe & Simons, 1991). LEC scores were very stable from wave to wave; the slope over time was near zero ($\beta||I|| = .002$, ns), and cross-wave stabilities were quite large (after correcting for the test-retest reliability of the measure, disattenuated *rs* ranged from .81 to .98). In keeping with Cole and Maxwell's (2009) suggestion that risk-outcome research be sensitive to the temporal dynamics of risk factors,

we focused on this stability and used average LEC scores as a Level 2 predictor in our analyses.

Procedures

Initial screening—The initial cognitive risk measures (CASI, CATQ, CTI, and SPPC) were administered to all students at school. In Grades 2 and 3, research assistants (RAs) read all questions to participants in one-on-one sessions, allowing the students to respond verbally or use graphical visual aids. In Grade 4, RAs met with participants in small groups (3-4 students) and read all questions aloud to the participants who made their own responses (under close supervision) on their own questionnaires. At all grade levels, we used laminated response charts as visual aids. To control for order effects, we counterbalanced the questionnaire presentation. RAs (advanced undergraduate and graduate students) were extensively trained before data collection. At the end of each session, students received candy and decorative pencils for their participation. The LEC was also administered during the screening; however, this administration was only used to construct the nomothetic impact weights.

We used three variables to select the HR and LR subsamples. Representing AS was the composite internal-stable-global score from the CASI. Representing NC was the average of standardized scalings of the CATQ and CTI. Representing SC was a linear combination of the five domain-specific subscales of the SPPC. Participants were eligible for the HR group if they scored one standard deviation from the mean in the “unhealthy” direction on any of the three composite risk variables and did not score one standard deviation from the mean in the healthy direction on any of the risk variables. Conversely, a participant who scored one standard deviation from the mean in the healthy direction on any risk variable and did not score one standard deviation from the mean in the unhealthy direction on any risk variable qualified for the LR group. From the screening sample, 56 were eligible for the HR group, 52 of whom consented to the follow-up study. From the screening sample, 148 met our LR criteria. From these, we selected 56 individuals whose group demographics (age, sex, and race) were similar to the HR group demographics. All 112 youths as well as one parent or guardian were invited to the university-based lab for the study. Of these, 100 attended the first session. All individuals who attended the first session completed the study. Participants were offered cab fare or free parking at the university. For their participation, parents received US\$75 for Wave 1, US\$80 for Wave 2, and US\$100 for Wave 3. Youths received snacks and a small toy. In total, 52 HR and 48 LR individuals kept their appointments and completed the study.

A breakdown of the HR participants revealed “high-risk” scores for 46% on the AS composite, for 44% on the NC composite, and 52% on the selfcompetence composite. Of these individuals, 19% met two of the three HR criteria and 12% met all three of the HR criteria. Conversely, a breakdown of the LR participants revealed “low-risk” scores for 56% on the AS composite, for 46% on the NC composite, and 52% on the self-competence composite. Of these individuals, 25% met two of the three LR criteria and 15% met all three of the LR criteria.

Once in the lab, youths and their parents completed paper-and-pencil measures and were interviewed separately about the child's depressive symptoms. One RA administered the CDI-C to the youth (as well as other measures not part of the current study), and another RA interviewed the parent about the youth (i.e., CDRS and K-SADS-PL). When finished, the parent and child/adolescent switched rooms; the parent completed the questionnaires while the youth was interviewed. All participants and RAs were naïve as to the youths' risk status. Of course, it is possible that RAs could guess participant risk status based on information obtained during the study. To test this possibility, we explicitly asked RAs to guess the risk status of each participant immediately after each data-collection session. At every wave, chi-square tests revealed that the accuracy of these guesses was no better than chance ($ps > .30$).

Results

Descriptive Statistics

Means and standard deviations for the screening sample and the HR/LR subsamples are reported in Table 2. On the measures of cognitive risk, the means for HR and LR subsamples were significantly different from one another. Not surprisingly, the HR/LR subsample means bracketed those for the screening sample. We also estimated the overtime stabilities of all the study variables. The median Lag 1 (12 month) and Lag 2 (24 month) correlations were .69 and .47 for the depression composite, .22 and .20 for the AS composite, .49 and .42 for the NC composite, and .62 and .57 for the self-competence composite.

Relatively few participants in the HR/LR subsample experienced a major depressive episode over the course of the study. Nevertheless, the pattern of these results is informative. *DSM-IV* diagnoses based on both parent and youth KSADS information revealed that five developed a major depressive episode. Four of these were in the HR group. Examination of the screening data revealed that they were so categorized because of their NC scores, not their AS or SC scores. Two of these cases emerged between screening and Wave 1, two more between Waves 1 and 2, and the last between Waves 2 and 3. Overall, the incidence of major depression was too infrequent to warrant statistical analysis, so we include this information for descriptive purposes only.

Empirical Overlap Among Cognitive Risk Measures

Correlations among the cognitive risk indices (for both the screening sample and the HR/LR subsample) appear in Table 3. Correlations involving the SPPC composite were negative (not surprising given that it is scaled such that high scores reflect positive self-cognition, opposite to the direction in which the other measures are scaled). Absolute values of the correlations ranged from .24 to .58 in the screening sample and .23 to .69 in the subsample. In the screening sample, the correlation between the NC and self-competence composites was significantly larger than the other two correlations ($ps < .05$).

For the subsample, correlations between the initial measures of cognitive risk and subsequent depressive symptoms over time are reported in Table 4. Somewhat different patterns emerged for the three types of cognitive risk. For AS, the correlations were small

and nonsignificant at every wave. For NC, the correlation was strong at Wave 1 but diminished over time, becoming relatively small and nonsignificant by Wave 3. For our index of self-competence, correlations with depressive symptoms were of moderate size and significant at all three waves.

Latent Growth Curve Analysis

We used latent growth curve analysis (LGC) to examine the relationship of various risk factors and their interactions with negative life events to the trajectory of depression scores over time. (We conducted these analyses using AMOS version 16 and double-checked the results using LISREL version 8.53.) A generic version of this model is depicted in Figure 1. Level 2 predictors consisted of Risk, Events (i.e., the within-subject average level of negative life events), and the Risk $\times$ Events interaction. These three variables predicted a latent intercept and slope, extracted from depression scores obtained at each of the three waves. Waves 1, 2, and 3 were encoded as 0, 1, and 2, respectively, so that the latent intercept represented depressive symptoms at Wave 1. We conducted four versions of this analysis, in which one of four predictors (Risk Group, AS, NC, or Self-Competence) served as the Risk variable. Our composite measure of depressive symptoms served as the dependent variable in each analysis.

All of these models fit the data well. All chi-squares were nonsignificant, and the NFI, IFI, and CFI were relatively large, ranging from .94 to .99. In none of these models, however, was the Risk $\times$ Events interaction statistically significant. Indeed, the partial R^2 for these interactions ranged from .003 to .009, smaller than what Cohen's (1977) criteria suggest would constitute even small effect sizes. Consequently, we reestimated these models eliminating the interactions. These models also fit the data well, as shown in Table 5. The numeric LGC results for depressive symptoms, Models 1 through 4, appear in Table 6, where the B s and β || I ||s represent the unstandardized and standardized coefficients, respectively, for the paths connecting predictors to the latent growth parameters. Figure 2 contains graphical displays of the Table 6 results for each dependent variable. In each graph, the expected values for a given risk group and level of life events (defined as high = +1 SD , low = -1 SD) for each wave were derived from the equations

$$DV_{\text{wave}} = \text{Intercept} + \text{Slope} * \text{Wave},$$

where Intercept = $B_0, \text{Int} + B_1, \text{Int RG} + B_2, \text{Int LE}$ and Slope = $B_0, \text{Slp} + B_1, \text{Slp RG} + B_2, \text{Slp LE}$, where the B s are the parameter estimates in Table 6 and waves were encoded 0, 1, and 2.

Risk group—In Model 1, the main effects for Risk Group and Events significantly predicted the latent intercept of the depressive symptoms trajectory (representing Wave 1 depressive symptoms approximately 6 months after the risk assessment). HR youth and youth with high levels of negative life events had higher levels of depressive symptoms. These two effects had an additive (but not multiplicative) impact on depressive symptoms, such that participants with high cognitive risk plus many negative life events had the highest depression scores, whereas participants with low cognitive risk and few negative life events

had the lowest levels of depressive symptoms. As shown in Figure 2, this pattern persisted across all three waves of the study; however, the strength of these effects diminished over time. To test these effects at subsequent waves, we recentered the model slope coefficients at and reestimated the model.³ At Wave 2, both the Risk main effect ($p < .005$) and the Events main effect ($p < .001$) remained. By Wave 3, only the main effect for Events remained significant ($p < .006$). In other words, cognitive risk was associated with increased levels of depressive symptoms over and above the effects of negative life events at 6 and 18 months but not at 30 months. To ascertain which of the three types of cognitive risk were responsible for these effects, we repeated these analyses using AS, NC, or self-competence as the risk factor.

AS—When we used AS as the risk factor (Model 2), we found a strong main effect for Events on the latent intercept at Wave 1 ($p < .001$, see Table 6), Wave 2 ($p < .001$), and Wave 3 ($p < .001$). Neither the AS main effect nor its interaction with Events was significant at any wave. At every wave, youth with more negative life events had higher levels of depressive symptoms, although this effect diminished over time (see Figure 2). No support emerged for either a diathesis–stress model or a cognitive main effect model at any wave when using AS.

NC—When we used NC as the risk factor (Model 3), we found evidence of main effects for cognitive risk and negative events at Waves 1 and 2 ($ps < .002$; see Table 6). At Wave 3, only the main effect of Events remained significant ($p < .006$). The main effect of NC on the slope was also significant; however, the Risk $\times$ Events interaction was not significant at any wave. Over- and-above negative life events, NC predicted depressive symptoms at 6 and 18 months, but this effect diminished over time and was not significant at 30 months (see Figure 2). No support emerged for the cognitive diathesis–stress model at any wave for the NC composite risk index.

Self-competence—When we used self-competence as the risk factor (Model 4), we found evidence of Risk and Events main effects at Wave 1 ($ps < .01$), Wave 2 ($ps < .01$), and Wave 3 ($ps < .02$). At every wave, lower levels of SC (i.e., higher cognitive risk) were associated with higher levels of depressive symptoms, over and above the significant but diminishing effects of negative life events (see Figure 2). Again, the Risk $\times$ Events interaction was nonsignificant.

Ethnic differences—We also tested for ethnic differences in the LGC model parameters, using a two-group LGC approach. We limited the analysis to White and African American participants because of the small numbers of other ethnic groups. All tests of group differences in intercepts, slopes, and structural parameters were nonsignificant. We did, however, detect significant mean differences on our measures of NC ($t_{94} = 3.21, p < .002$) and negative life events (LEC; $t_{94} = 3.68, p < .001$). The African American means were higher than White means on both NC (Cohen's $d = .77$) and LEC ($d = .90$). Differences on AS, SPPC, and the depressive symptom composite were all nonsignificant ($ps > .10$).⁴

Unique and combined effects—To ascertain whether one type of cognitive risk contributed to the prediction of depressive symptoms over and above the others, we repeated the previous LGC analyses using AS, NC, and self-competence (as well as negative life events) as concomitant predictors of depressive symptoms. As shown in Table 5, the model fit the data well. Table 7 shows that the pattern of significant effects changed with the parameterization of the intercept. When the intercept was encoded to represent Wave 1, NC and negative events both predicted the intercept, over and above AS and SC. There was also a negative effect of NC on the slope.⁵ When we reparameterized the model so that the intercept represented Wave 2 depressive symptoms, SC emerged as a significant predictor of the intercept, along with NC and negative events. The pattern changed again when we set the intercept to be Wave 3 depressive symptoms; SC and negative events remained significant, but NC became nonsignificant.

Unexplained variance—To put the role of these cognitive diatheses into a broader perspective, it is useful to examine the proportion of variance in depressive symptoms that they actually explain. As shown in Table 8, negative events explained between 13.5% and 19.0% of the variance in depressive symptoms. Over and above negative events, the three cognitive measures taken together explained 25.3% of the remaining variance at Wave 1, 17.2% at Wave 2, and 5.4% at Wave 3. Despite their statistically significant contributions, these three cognitive diatheses leave a relatively large proportion of the variance in depressive symptoms unexplained.

Discussion

This study represents the first longitudinal investigation of the combined and unique effects of AS, NC, and SC on depressive symptoms in children and young adolescents. Three main findings emerged. First, some empirical overlap exists among these three types of cognitive diatheses, especially between NC and SC. Second, the combination of AS, NC, and SC was related to depressive symptoms at 6, 18, and 30 months; however, the magnitude of this effect diminished over time. These effects were primarily due to NC and SC, not AS. The relative (or unique) utility of NC and SC as predictors varied with the time lag. Third, although main effects for negative life events and cognitive risk were significant, their interaction was not, suggesting an additive (not multiplicative) form for the diathesis–stress model of depression during this age range. That said, we hasten to add that substantial variance in depressive symptoms remained unexplained by all of our cognitive diathesis measures plus negative life events. We expand on each of these findings below.

Our first major finding pertains to the overlap between AS, NC, and SC. At a definitional level, these three constructs have clear points of convergence. Blaming oneself in a stable and global fashion (AS), harboring negative thoughts about oneself (NC), and believing that one is fundamentally incompetent in one or more domains (SC) would seem to share some theoretical common ground. Empirical evidence of the overlap, however, was mixed. On one hand, correlations between AS and the other two constructs, although significant, were quite modest. Furthermore, AS was unrelated to all measures of depressive symptoms. We speculate that the weak correlations involving AS may be the result of the cognitive developmental level of our (relatively) young participants. We have argued elsewhere that

AS may not be well developed or serve as a style-like cognitive diathesis for depression until early adolescence (Cole et al., 2008; Cole & Turner, 1993; Turner & Cole, 1994). Prior to this age, AS may still be under construction. Alternatively, these low correlations could be the result of measurement problems. Despite the fact that we used a sophisticated, interview-based measure of AS, it (like most measures of AS) asks the participants to imagine and report about their cognitive responses to hypothetical situations. This task requires a degree of abstract reasoning, metacognitive skill, and working memory that may not come easily to younger participants during the early waves of this study. On the other hand, correlations between NC and SC were relatively strong. We believe that two things are responsible for the magnitude of this correlation. One is a degree of conceptual overlap between these constructs. SC represents youths' beliefs about their competence (or incompetence) in several specific, developmentally relevant domains. Harter (1988) suggested that children and early adolescents construct beliefs about their global self-worth out of these domain-specific self-perceptions. This more generalized construction is very similar to the self-cognitions that constitute one third of Beck's (1963) cognitive triad and, in a sense, may constitute its developmental building blocks. As such, we would expect a correlation between these building blocks and aspects of Beck's negative self-schema. The other reason for the strong correlation may be a low degree of differentiation between these cognitive constructs, as a function of the participant's level of cognitive development. Although Beck conceived the cognitive diathesis for depression as having a tripartite structure (self, world, and future), developmental analysis of measures like the CTI-C suggest that the structure may be much simpler, especially at younger ages (LaGrange et al., 2008). Given the egocentricity so characteristic of youth, our younger participants' conception of world and future may be part and parcel of a relatively undifferentiated self-construct, further contributing to the strong correlation with SC.

Our second major finding pertained to the combined and unique effects of AS, NC, and SC on depressive symptoms over time. Although the combination of all three cognitive risk factors was significantly related to subsequent levels of depressive symptoms, the contributions of AS, NC, and SC were quite different from one another. As mentioned above, AS was unrelated to all measures of depressive symptoms. The relationship of NC to depressive symptoms was strongest at 6 months but diminished to nonsignificant levels as the time lag increased. The contribution of SC to depressive symptoms (although significant in isolation) was nonsignificant at 6 months when NC was added to the analysis. At 18 and 30 months, however, the contribution of SC became stronger, as the contribution of NC became weaker. In effect, NC served as a better short-term predictor and SC served as a better long-term predictor of depressive symptoms.

When interpreting these results, it is important to remember that the predictive utility of cognitive risk is partially due to the overtime stability of AS, NC, and SC. With a 12-month stability of only .22, AS shares only 4% variance with itself 1 year later, seriously compromising its long-term potential for prediction. For NC, the 12-month stability was approximately .49. This moderate level may have facilitated its short-term predictive power but diminished its longer term utility. The SC measure had the greatest stability ($r = .62$), a feature that may have enabled it to outlast NC as a longer term predictor. Of course, one

potential reason for instability is developmental change. Developmental theory and research implicate the construction of self-competence as a key developmental task of middle childhood and early adolescence, the resolution of which has a variety of long-term psychological implications (Cole et al., 1996, 1997; Garber, 1984; Harter, 1988; Harter & Whitesell, 1996). Conversely, AS and negative cognitive schemas are largely based on theory and research focused on adults. Although AS and NC may be highly stable constructs in the mature adult, they may still be changing during late childhood and early adolescence.

Our third finding pertained to the combined effects of cognitive risk and negative life events. The classic diathesis–stress model, in which the cognitive diathesis enhances the depressive effect of negative life events, was not supported. None of the cognitive measures interacted with overall level of negative life events in the prediction of depressive symptoms. Instead, we found support for an additive model in which negative events and cognitive risk (NC and SC) both contributed to the expression of depressive symptoms. Participants with high overall levels of negative events *and* strong cognitive risk showed more signs of depression than did participants with only one such risk factor. Furthermore, youths with either high levels of negative events *or* strong cognitive risk showed more signs of depression than did those with neither risk factor. Elsewhere, we have suggested that the classic diathesis–stress interaction may not characterize the role of AS until adolescence, when abstract reasoning (if not formal operations) enable the individual to endow negative events with meaning not inherent in the events themselves and then (over-)generalize the implications (e.g., Cole & Turner, 1993; Turner & Cole, 1994). Although we urge caution in the interpretation of null results, this study joins a growing literature that is commensurate with this developmental position (see Cole et al., 2008). Negative thinking (NC and SC) and negative events have an additive, but not multiplicative, effect on depressive symptoms in later childhood and early adolescence.

These findings should be viewed in the context of a broader set of factors predicting depression in youth. All three cognitive diatheses plus negative life events collectively explained only 34% of the variance in depressive symptoms 6 months later. This percentage dropped to 31% at 18 months and 18% at 30 months. Although cognitive diatheses are responsible for a substantial portion of the explained variance, an even larger portion of the variance of depressive symptoms remains unexplained. To explain the variance in the expression of depressive symptoms more completely, future investigations should consider the combined (and possible interactive effects) of biological and genetic factors in conjunction with cognitive and environmental variables (e.g., Caspi & Moffitt, 2006).

The current research has potentially important implications for the prevention and treatment of depression in youth. Reviews of the early adolescent depression treatment and prevention literatures (Gladstone & Beardslee, 2009; Horowitz & Garber, 2006; Weisz, Rudolph, Granger, & Sweeney, 1992) suggest that many of these empirically supported programs are based on cognitive and cognitive behavioral models of depression and focus on factors such as flexible thinking, AS, and problem solving (e.g., Clarke et al., 2001; Gillham, Reivich, Jaycox, & Seligman, 1995). Despite the efficacy of these programs, some investigators have questioned the role of AS. For example, in the Penn Resiliency Project, Gillham and Reivich (1999) noted that AS did not mediate the effect of their prevention program on depressive

symptoms in youth and suggested that it “may not be a risk factor for depressive symptoms” (p. 461). Horowitz and Garber (2006) emphasized the need for such interventions to take the youth’s level of cognitive development into account. Results of the current project support the speculation that AS may not be the mechanism around which to target intervention efforts. Instead, cognitive programs focusing on altering NC and increasing SC might have a stronger influence on depressive symptoms.

Several shortcomings and limitations of the current study suggest important avenues for future research. First, is that our results pertain to the number and severity of depressive symptoms, not to major depression itself. Despite our focus on an at-risk subsample from a relatively large screening sample, only 5 participants manifested a major depressive episode over the course of the study. Most of these came from our HR group. Although this incidence is far greater than the base rate of major depression in this age group, our numbers were too small to permit statistical analysis. We hope that these findings encourage similar work with larger and more severely disturbed samples in the future.

Second, the interval between waves in this study was 1 year. Although some longitudinal studies of children’s cognitive vulnerability have used intervals this long or longer (e.g., Giaconia, Pakiz, Silverman, Frost, & Lefkowitz, 1993; Hankin et al., 1998), many have used shorter intervals (e.g., Abela, 2001; Hammen & Goodman-Brown, 1990; Nolen-Hoeksema, Girgus, & Seligman, 1986). At this point, no one knows how much time must elapse for cognitive style, negative life events, or their interaction to generate depression. Indeed, the requisite time interval may not be the same for all three types of predictors. This raises the possibility that a 1-year time lag may be better suited for the detection of main effects than it is for the detection of cognition-events interactions. Clearly, more research is needed to unpack the temporal dynamics that underlie the longitudinal relationships between cognition, stress, and depression in children.

The third potential concern is that although the cognitive risks factors correlated with the intercepts of the depression trajectories (i.e., the level of depressive symptoms at each subsequent wave of the study), they were not significantly related to the slopes. In other words, cognitive risk was associated with subsequent levels of depression but not change in depression. This suggests three interesting possibilities (not mutually exclusive). One is that NC and self-competence become relatively stable risk factors at a relative early age (Cole, Maxwell, et al., 2001) and maintain risk for depressive symptoms year after year. The second is that AS is a relatively unstable risk factor during middle childhood and early adolescence (Cole et al., 2008) that does not convey risk for depression that lasts as long as 12 months. And the third possibility is that of reciprocal effects. On one hand, cognitive diatheses may predispose depression; on the other hand, depression may contribute to the construction of cognitive vulnerabilities. All of these possibilities constitute interesting avenues for future research.

Fourth, we must resist the temptation to conclude in favor of the null hypothesis, with respect to our nonsignificant diathesis–stress interactions. Even though our results are consistent with a growing literature suggesting that such interactions do not emerge until later adolescence and that a main effects model suffices in middle childhood and early

adolescence (Cole et al., 2008; Lakdawalla et al., 2007; cf. Abela & Hankin, 2008; Abela & Skitch, 2007), this study did not follow its participants long enough to witness the possible later emergence of these interactions. Even though a power analysis revealed that our sample size was sufficient to detect a moderate effect, even small interaction effects can be important. We encourage future researchers to consider carefully the value of longitudinal research focused on the long-term predictive utility of diathesis–stress interactions in developmental psychopathology.

Finally, we caution against the overgeneralization of these results. Our sample consisted of urban youths with low to medium SES, some of whom have experienced substantial negative life events. Almost three fourths of the sample was African American. Replication of these results in other populations is needed before extrapolating from these results to other groups.

Acknowledgments

Funding

The author(s) disclosed that they received the following support for their research and/or authorship of this article: This research was supported in part by Grant numbers R01MH64650 from the National Institute on Mental Health and P30HD15052 from the National Institute of Child Health and Human Development to Vanderbilt University and by a generous endowment from Patricia and Rodes Hart.

Bios

David A. Cole is chair of psychology and human development at Vanderbilt University, Nashville, Tennessee. His research interests center around development psychopathology in general and childhood depression in particular. Working from the premise that the origins and perpetuation of mental illness have large interpersonal components, he studies the relationship between childhood depression and patterns of parent-child interactions, peer relationships, and competency-based feedback from significant others. He further suggests that cognitive models of depression must take into consideration the level of the child's cognitive development. His studies imply that adult models of depression need substantial modification when applied to children. Related interests include the assessment of childhood disorders and the prediction of adolescent suicide. Understanding suicidal ideation and suicidal behavior in the context of adolescent development is an underlying theme of this research. Other areas of research include developing new methodologies for clinical and psychopathology research, validating clinical assessment instruments, and examining change in nonexperimental research designs.

Farrah M. Jacquez, PhD, is an assistant professor and clinical child psychologist at the University of Cincinnati who specializes in improving the health of children and families in under-served communities. In her research, she is interested in utilizing community-based research methods to address health disparities. Current projects focus on obesity intervention for children and preventing type 2 diabetes in adolescents.

Beth LaGrange, PhD, received her degree from Vanderbilt University under the supervision of Dr. David Cole. Her research interests include the use of statistical models to examine and refine measures of depression in children.

Ashley Q. Pineda, PhD, acquired her degree from Vanderbilt University and completed her pre-doctoral internship at the Children's Hospital at Stanford University. She is currently Adjunct Faculty at Vanderbilt, where she provides clinical supervision for doctoral students, and is in private practice, where she provides therapy and psychological evaluation for children, adolescents, and adults.

Alanna E. Truss, PhD, is a clinical psychologist who earned her degree from Vanderbilt University and completed her pre-doctoral internship at Duke University Medical Center. Alanna returned to Vanderbilt University Medical Center for her post-doctoral training and she is currently working in the Vanderbilt Center of Excellence for Children in State Custody. Dr. Truss' major areas of interest include the impact of trauma on children and evidence-based interventions for children and families with trauma.

Amy S. Weitlauf, MS, is conducting dissertation research under the supervision of Dr. David Cole at Vanderbilt University. Her research interests focus broadly on developmental psychopathology, and specifically on the applicability of attributional models of depression to children and adolescents. Amy will begin a predoctoral internship at the University of North Carolina in September 2010.

Carlos Tilghman-Osborne, MS, is conducting dissertation research under the supervision of Dr. David Cole at Vanderbilt University. His current research interests include the refinement of the constructs of guilt and shame in young populations. He will begin his predoctoral internship in child and adolescent psychiatry at Vanderbilt University.

Julia Felton, MS., is conducting dissertation research under the supervision of Dr. David Cole at Vanderbilt University. Her current research interests include the relations between rumination, depression, and body image in adolescents. She will begin her predoctoral internship at the Medical University of South Carolina in August 2010.

Judy Garber, PhD, is a professor of psychology at Vanderbilt University. Her research focuses on the etiology, course, outcome, treatment, and prevention of depression in children and adolescents. Currently, Dr. Garber is coordinating a multisite study funded by NIMH examining the effectiveness of a cognitive-behavioral program for the prevention of depression in at risk adolescents.

Danielle H. Dallaire, PhD, completed her degree in developmental psychology at Temple University in 2003 and after completing a post-doctoral research position at Vanderbilt University in 2006, has been an assistant professor in the psychology department at The College of William & Mary. Her research interests include children's social and emotional development, how children and families cope with separation from parents, including how parental incarceration affects children, parent-child attachment relationships, and developmental psychopathology.

Jeff A. Ciesla, PhD, is an assistant professor of clinical psychology at Kent State University. His research involves risk for depression, with two foci: (1) the combined influence of rumination and maladaptive beliefs, and (2) models of life stress, including the stress sensitization, generation, and autonomy models. Additionally, he is interested in the application of SEM and HLM statistical techniques in psychopathology research.

Melissa A. Maxwell, MS, recently completed a dissertation under the supervision of Dr. David Cole at Vanderbilt University. Her research and clinical interests are tied to the intersection of gender and health across the lifespan. She will begin her predoctoral internship at the Durham VA Medical Center in July 2010.

Lynette Dufton, PhD, obtained her degree in clinical psychology from Vanderbilt University in 2009. Currently, Lynette is a post-doctoral fellow at the University of Washington.

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Figure 1. Path diagram of generic latent growth curve analysis, in which risk, events, and their interaction predict the trajectory of depression scores across three waves

Figure 2. Bar charts of depressive symptoms (composite of parent and youth CDI and CDRS) by high/low cognitive risk group, high/low negative life events (± 1 SD), and wave

Table 1

Demographic Characteristics of the Screening, High-Risk, and Low-Risk Samples

Characteristic	Screening sample	<u>High-/low-risk sample</u>	
		Low risk	High risk
<i>N</i>	515	48	52
Age (<i>SD</i>)	8.54 (1.23)	8.50 (1.22)	8.51 (1.43)
Sex (male : female)	232 : 283	24 : 24	27 : 25
Race			
White (%)	27.3	29.3	21.3
Black (%)	68.5	66.5	76.8
Hispanic (%)	0.9	2.1	1.9
Asian (%)	0.6	0	0
Multiethnic (%)	1.8	2.1	0
Other (%)	0.9	0	0
Number of siblings	1.7	1.6	1.8
Parents divorced (%)	24.5	18.9	25.8
Mean income (US\$)	24,337	23,677	18,780 ^a

^aHigh- and low-risk groups were significantly different ($p < .05$).

Table 2

Means (and Standard Deviations) for Screening Measures

Risk measure	Screening sample	Subsample	
		Low risk	High risk
CASI composite	88.00 (22.53)	74.93 (19.34)	100.66 (21.04)
CTI-C	19.62 (15.29)	10.56 (10.29)	29.09 (14.06)
CATQ	68.62 (22.14)	55.54 (19.67)	83.22 (17.21)
SPPC composite	90.77 (18.20)	105.02 (9.18)	75.27 (11.61)

Note: CASI = Children's Attributional Style Inventory; CTI-C = Cognitive Triad Inventory–Child Version; CATQ = Children's Automatic Thoughts Questionnaire; SPPC = Self-Perception Profile for Children. All differences between low- and high-risk means were significant ($p < .001$).

Table 3

Correlations Among Risk Variables

Measure	Attributional style	Negative cognitions	Self-competence
Attributional style	—	0.26**	−0.23*
Negative cognitions	0.32***	—	−0.69***
Self-competence	−0.24***	−0.58***	—

Note: Screening sample below and subsample above the diagonal.

*
 $p < .05$.

**
 $p < .01$.

 $p < .001$.

Table 4

Correlations of Initial Cognitive Risk Variables With Measures of Depressive Symptoms Over Time in the Subsample

Cognitive risk factor	Depressive symptoms (composite of parent and youth CDI and CDRS)		
	Wave 1 (6 months)	Wave 2 (18 months)	Wave 3 (30 months)
Attributional style	.08	.05	.08
Negative cognitions	.49***	.26*	.18
Self-competence	-.36***	-.37***	-.32**

Note: CDI = Children's Depression Inventory; CDRS = Child Depression Rating Scale.

*
 $p < .05$.

**
 $p < .01$.

 $p < .001$.

Table 5

Goodness-of-Fit Indices for Latent Growth Curve Analyses

Model: Risk factor	χ^2/df	<i>df</i>	NFI	IFI	CFI	RMSEA	<i>p</i> close
Model 1: Risk group	5.39	3	0.97	0.98	0.98	0.030	.76
Model 2: Attributional style	3.85	3	0.97	0.99	0.99	0.018	.86
Model 3: Negative cognitions	6.98	3	0.96	0.98	0.97	0.039	.63
Model 4: Self-competence	3.30	3	0.98	1.00	1.00	0.011	.90
Model 5: All predictors	6.92	5	0.97	0.99	0.99	0.021	.92

Note: NFI = normed fit index; IFI = incremental fit index; CFI = comparative fit index; RMSEA = root mean squared error of approximation; *p* close = *p* value for test of the null hypothesis that the RMSEA = .05.

Table 6

Latent Growth Curve Analyses: Effects of Cognitive Risk and Negative Events on Latent Intercepts and Slopes for Composite of Self- and Parent-Reports of Depressive Symptoms (CDI and CDRS)

Effect	Unstandardized <i>B</i>	SE (<i>B</i>)	Standardized β I	<i>t</i>	<i>P</i>
Model 1: RG, <i>low</i> (0) vs. <i>high</i> (1)					
Intercept ^a	−0.203	.255		−0.798	.425
RG → intercept	1.224	.365	0.333	3.356	.001
NE → intercept	0.547	.189	0.296	2.895	.004
Slope ^a	−0.060	.135		−0.443	.658
RG → slope	−0.376	.194	−0.238	−1.939	.052
NE → slope	−0.029	.100	−0.037	−0.293	.770
Model 2: AS					
Intercept ^a	0.44	.18		2.40	.017
AS → intercept	0.05	.18	0.03	0.29	.773
NE → intercept	0.74	.19	0.41	3.87	.001
Slope ^a	−0.26	.09		−2.75	.006
AS → slope	−0.01	.09	−0.01	−0.08	.937
NE → slope	−0.09	.10	−0.12	−0.89	.376
Model 3: NC					
Intercept ^a	0.39	.17		2.34	.019
NC → intercept	0.73	.16	0.43	4.56	.001
NE → intercept	0.47	.18	0.25	2.59	.010
Slope ^a	−0.24	.09		−2.70	.007
NC → slope	−0.27	.09	−0.36	−3.17	.002
NE → slope	0.01	.10	0.01	0.10	.918
Model 4: SC					
Intercept ^a	0.39	.17		2.24	.025
SC → intercept	−0.47	.17	−0.29	−2.78	.006
NE → intercept	0.60	.19	0.33	3.16	.002
Slope ^a	−0.25	.09		−2.71	.007
SC → slope	0.05	.09	0.08	0.57	.568
NE → slope	−0.08	.10	−0.10	−0.76	.449

Note: RG = risk group; AS = attributional style; NC = negative cognitions; SC = self-competence; NE = negative events.

^a.Regression intercepts.

Table 7

Latent Growth Curve Parameter Estimates From Models Reparameterized With Intercept Set at Different Waves

Effect	With Wave 1 set to 0			With Wave 2 set to 0			With Wave 3 set to 0		
	Unstandardized <i>B</i>	<i>SE(B)</i>	Standardized $\beta I $	Unstandardized <i>B</i>	<i>SE(B)</i>	Standardized $\beta I $	Unstandardized <i>B</i>	<i>SE(B)</i>	Standardized $\beta I $
Intercept	0.37*	0.16		0.13	0.14		-0.11	0.16	
NE → intercept	0.43*	0.18	0.23	0.43**	0.15	0.28	0.43*	0.18	0.28
AS → intercept	-0.31	0.17	-0.18	-0.19	0.14	-0.14	-0.07	0.17	-0.05
NC → intercept	0.73***	0.19	0.43	0.40*	0.15	0.29	0.06	0.18	0.05
SC → intercept	-0.26	0.18	-0.15	-0.31*	0.15	-0.22	-0.35*	0.17	-0.24
Slope	-0.24**	0.09							
NE → slope	0.00	0.10	0.00						
AS → slope	0.12	0.09	0.16		Same			Same	
NC → slope	-0.33***	0.10	-0.45						
SC → slope	-0.04	0.10	-0.06						

Note: NE = negative events; AS = attributional style; NC = negative cognitions; SC = self-competence.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 8

Proportion of Variance in Depressive Symptoms Explained by Cognitive Diatheses and Life Events

Variance components	Wave 1	Wave 2	Wave 3
Proportion of variance explained by NE alone (R^2)	0.172	0.190	0.135
Proportion of variance explained by NE, AS, NC, SC (R^2)	0.339	0.309	0.179
Raw change in R^2 due to AS, NC, and SC, controlling for NE	0.167	0.119	0.044
Proportion of unexplained variance explained by AS, NC, and SC	0.253	0.172	0.054

Note: NE = negative events; AS = attributional style; NC = negative cognitions; SC = self-competence.