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Disentangling the Prospective Relations Between Maladaptive Cognitions and Depressive Symptoms

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Abstract

In a four-wave, cohort-longitudinal design with a community sample of 515 children and adolescents (grades 2 through 9), this study examined the longitudinal structure of and prospective interrelations between maladaptive cognitions and depressive symptoms. Multigroup structural equation modeling generated four major findings. First, the longitudinal structures of maladaptive cognitions and depressive symptoms consist of a single time-invariant factor and a series of time-varying factors. Second, evidence supported a model in which depressive symptoms predicted

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negative cognitions but not the reverse. Third, the time-invariant components of cognition and depression were highly correlated. Fourth, the strength of the depression-to-cognition relation increased with age. Implications regarding the mechanisms underlying clinical interventions with depressed children are discussed.

Keywords

depression; child; adolescent; cognitions; trait-state

Aaron Beck's characterization of depressive cognitions (Beck, 1963, 1976) laid the foundation for much of the current thought regarding the relations between negative cognitions and negative affect. In the current paper, we describe a parallelism between aspects of Beck's schema theory and components of a specific type of latent variable structural equation model, called the trait-state occasion model (TSO; Cole, Martin, & Steiger, 2005). Using cohort-sequential data on children and adolescents, we use the TSO model to test hypotheses about the development of depressive cognitions, their emergent stability, and their prospective relation to depressive symptoms.

Beck (1987; Clark, Beck, & Alford, 1999) suggested that automatic, negative, surface-level thoughts (especially those pertaining to one's self, world, and future) are indicators of deeper depressive cognitive schemas. After their development (possibly linked to loss experiences in childhood; Beck, 1976), schemas are regarded as relatively stable and latent, and may be activated by negative mood or by negative life events that are thematically linked to the schema and/or its generative events. Thus, surface-level thoughts constitute fallible manifest indicators of deeper cognitive schemas that cannot be directly observed. The existence of such schemas is inferred by these (and other) indicators. We find this description analogous to the logic that drives latent variable statistical methods such as structural equation modeling (SEM). In latent variable SEM, the existence of a latent variable (potentially corresponding to an underlying psychological construct) is inferred from the covariances among a set of surface-level, fallible, observable variables (such as those that derive from psychological measures designed to tap the underlying psychological construct). In both schema theory and SEM, a change in the surface-level indicator is conceptualized as a function of deeper, underlying processes plus error, noise, or other various extraneous factors.

The recent development of one specific kind of longitudinal structural equation model may be particularly useful in testing key hypotheses about depressive cognitions and their underlying schemas. Building upon previous trait-state methodological advances (Eid & Langeheine, 1999; Kenny & Zautra, 1995; Steyer & Schmitt, 1994), Cole et al. (2005) developed a TSO structural equation model designed to partition a latent variable into two parts, a completely stable or time-invariant (TI) component and a time-varying (TV) component. This model was subsequently elaborated and extended by Ciesla, Cole, and Steiger (2007) and LaGrange and Cole (2008). Recently, a method for examining structured means was also incorporated into the TSO model (Cole, in press). The TSO model can be used in at least two ways that are relevant to the study of depressive cognitions.

First, the TSO model estimates the degree to which the latent variable reflects something time-invariant versus time-varying. That is, the model provides information about the proportion of variance in the latent variable that is due to something 100% stable or “trait-like” versus something less stable. When applied to different age groups, the TSO model can reveal developmental differences in the degree to which a latent variable is time-invariant. For example in research on children’s attributional style, Cole et al. (2008) demonstrated that a reliable time-invariant (or trait-like) component of children’s attributional style was not evident in a cohort of 7–10 year olds, but was evident in two older cohorts (ages 9–12 and 11–14). They interpreted these results as indicating that attributional style does not become “style-like” until late childhood or early adolescence. This interpretation is consistent with the idea that children’s understanding of and attributions about causal relations change with level of cognitive development (e.g., Folmer et al., 2008; Nicholls, 1978; Nicholls & Miller, 1983; Turner & Cole, 1994).

Understanding the degree to which other kinds of depressive cognitions reflect stable or trait-like processes in children is important for both theoretical and clinical reasons. Generally positive or negative views of self, world, and future are evident at very early ages (Harter, 1986) and may not require the same degree of cognitive sophistication as do causal attributions about complex or ambiguous events (Nicholls, 1978; Folmer et al., 2008). Judging the long-term stability of such cognitions is difficult, however, because measures of negative automatic thoughts (e.g., Automatic Thoughts Questionnaire, ATQ; Hollon & Kendall, 1980; Kazdin, 1990) or Beck’s cognitive triad (e.g., Cognitive Triad Inventory for Children, CTI-C; Kaslow, Stark, Printz, Livingston, & Tsai, 1992) have rarely if ever been administered in long-term longitudinal studies. Reliable estimation of the time-invariant component of a latent variable in TSO models requires that the study and the time lags between waves be long enough to allow test–retest correlations sufficient time to reach their asymptote.

Studies of related cognitions provide mixed results. On the one hand, cross-wave correlations for measures of self-esteem or self-worth are large, between .69 to .71 over intervals of 1 year or longer (Gotlib, Lewinsohn, Seeley, Rohde, & Redner, 1993; Kamakura, Ando, & Ono, 2007). Similarly, some studies of negative cognitions report 6- to 12-month correlations of 0.54 to 0.69 for measures of dysfunctional attitudes, hopelessness, and cognitive errors (Gotlib et al., 1993; Stewart et al., 2004). On the other hand, other studies of dysfunctional attitudes show relatively small test–retest correlations, 0.35 or less over only 5-week intervals (Hankin, 2008; Lee & Hankin, 2009). Given this mix of results, we used 1-year intervals in the current 4-wave study. The length of these intervals and the overall duration of this study enable reliable TSO model estimation of the time-invariant factor (if such a factor truly exists) even if the 1-wave correlations are very large.

Second, the TSO model can be expanded to examine two latent variables simultaneously, thereby enabling the testing of reciprocal predictive, cross-lagged relations. The traditional statistical paradigm for testing longitudinal predictive relations is to regress the dependent variable at Time 2 onto the putative predictor measured at Time 1 while controlling for a measure of the dependent variable at Time 1. This approach can be frustrating when individual differences in the constructs of interest are highly stable over time, as the control

variable leaves little for the predictor to predict. Depressive symptoms and maladaptive cognitions both exhibit high stability. Cross-wave correlations for measures of depressive symptoms are large, especially over intervals of one year or less (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Tram & Cole, 2006). Test-retest correlations for measures of maladaptive cognition are generally smaller but still provide evidence of some stability (e.g., Gotlib et al., 1993; Hankin, 2008; Stewart et al., 2004). Utilizing longitudinal designs with longer interwave time intervals can help in such cases (Cole & Maxwell, 2009), as auto-correlations often diminish as lag time increase. The use of TSO models can help even more.

The advantage of TSO models is that the TI component of the constructs can be extracted and statistically controlled during attempts to predict the TV component. In other words, the investigator can focus on predicting change in that part of the latent variable that actually changes. Cole, Nolen-Hoeksema, Girgus, and Paul (2006) successfully used such methods to examine the prospective relation between negative life events and the TV component of depressive symptoms, controlling for the TI component of depressive symptoms. Compared to more traditional cross-lag statistical techniques, this method may generate greater statistical power for testing prospective relations (Cole & Maxwell, 2009).

Longitudinal studies of prospective relations between negative cognitions and depressive symptoms in children and adolescents provide mixed results. Our review of this literature suggests that these inconsistencies may be due to differences between the selected cognitive and depression measures with regard to either their cross-sectional reliability or their longitudinal stability (or both). First, a few studies have simultaneously examined bidirectional prospective relations between negative cognitions and depressive symptoms (Greening, Stoppelbein, Dhossche, & Martin, 2005; McGrath & Repetti, 2002; Stewart et al., 2004; Timbremont & Braet, 2006). Additionally, we reanalyzed Hankin, Abramson, and Siler's (2001) two-wave study of depression and hopelessness in 270 adolescents, finding no support for $\text{COG} \rightarrow \text{DEP}$ ($\beta = 0.00$) and moderate support for $\text{DEP} \rightarrow \text{COG}$ ($\beta = 0.15, p > .005$). Taken together, these studies provide stronger evidence that depressive symptoms predict negative cognitions ($\text{DEP} \rightarrow \text{COG}$) than the reverse ($\text{COG} \rightarrow \text{DEP}$), especially over longer periods of time.

This difference could be due to the fact that the longer-interval studies focused on shorter subscales of cognitive measures and full-scale measures of depressive symptoms. All other things being equal, shorter subscales are less reliable. Thus, the control variable (Time 1 cognition) was less reliable in tests of the $\text{DEP} \rightarrow \text{COG}$ hypothesis compared to the control variable (Time 1 depression) in tests of the $\text{COG} \rightarrow \text{DEP}$ hypothesis. It is well known that controlling for a less reliable measure can spuriously inflate estimates of the targeted relation (Kahneman, 1965; see also Cole & Maxwell, 2003). Thus in long-term studies of bidirectionality, it is possible that stronger evidence for $\text{DEP} \rightarrow \text{COG}$ than $\text{COG} \rightarrow \text{DEP}$ could be due to differential reliability of the control variables. In the current study, we avoid this problem by focusing on the relations among latent variables (controlling for differential reliability problems).

Second, most unidirectional studies have focused on the $\text{COG} \rightarrow \text{DEP}$ relation and tended to use somewhat shorter time intervals (e.g., Abela & Sullivan, 2003; Lee, Hankin, &

Mermelstein, 2010; Kennard, Stewart, Hughes, Patel, & Emslie, 2006). In general, empirical support for this prospective relation has been greater when the time intervals have been longer. Over a 6-week interval, Abela and Sullivan (2003) found no evidence that dysfunctional attitudes predicted change in depressive symptoms. Over a 10-week interval, Lee et al. (2010) found that self-perceived social competence predicted change in depressive symptoms with a standardized beta of -0.09 ($p < .035$). Over a 6-month period, Kennard et al. (2006) found still stronger support for the hypothesis that cognitions predict change in depressive symptoms, with standardized betas of -0.13 , -0.22 , and 0.51 for self-efficacy, cognitive errors, and hopelessness, respectively. We speculate that stronger support for the COG $\rightarrow$ DEP hypothesis emerges when time lags are longer, in part because the shorter-term stability of depression measures is quite high. Predicting change in depression is difficult when little change has had time to occur. In the current study, our use of TSO structural equation modeling will control for the completely stable components of both depression and cognition, therefore focusing on those aspects of these variables that actually change over time.

In the mature individual, both the DEP $\rightarrow$ COG and COG $\rightarrow$ DEP processes may be fully operational parts of a truly vicious cycle (Teasdale, 1983a, 1983b), making it difficult to determine the primacy of one process over the other. In youth, when depressive cognitive schemas are under construction and depressive symptoms are relatively new experiences (Beck, Rush, Shaw, & Emery, 1979; Cole, Jacquez, & Maschman, 2001), one process may emerge before the other. Childhood, therefore, presents a potential window during which to observe these processes before they become intertwined.

Hypotheses

First, given that individual differences on measures of depressive cognition and depressive symptoms show moderately high stability, we hypothesize that these constructs reflect the influence of both TI and TV factors, and that the application of the TSO model to longitudinal data on these constructs will provide evidence of both. Second, given preliminary (albeit somewhat inconsistent) support for both the COG $\rightarrow$ DEP model and the DEP $\rightarrow$ COG model, we hypothesize that support for both models will emerge even more clearly when using the TSO model to control for the TI components of the dependent variable. Third, irrespective of the prospective effects, we expect the TI components of both negative cognitions and depressive symptoms to be strongly correlated. Finally, as children grow older and systems underlying the relation of affect and cognition mature, we expect that the strength of both cognition $\rightarrow$ depression effects and depression $\rightarrow$ cognition effects to increase with age.¹ To address these questions, we followed three cohorts of children (in grades 2, 4, and 6 at the beginning of the study) over the course of 4 years. Our use of a relatively large sample of children spanning grades 2 through 9 (ages 7 to 16) allowed us to use the TSO model to test our hypotheses over a range of developmental stages.

¹We do not test hypotheses about the interaction between cognition and negative life events in this study, in part because our theory does not anticipate such an interaction in childhood (when cognitive diatheses are still under construction) and in part because this hypothesis is tested elsewhere (Cole, 1991; Cole et al., 2001; Cole & Turner, 1993; Turner & Cole, 1994).

Method

Participants

At the beginning of this four-wave longitudinal study, we recruited participants from five public elementary and three public middle schools in a midsized southern city.² Of these, 556 (73%) students returned parent consent forms, granting permission to participate. Absenteeism on the data collection day reduced the wave 1 sample to 515 participants. In every cohort, study participants were not significantly different from nonparticipants at the schools from which they were recruited with regard to their sex, age, ethnicity, or participation in the free/reduced lunch program (a crude index of socioeconomic status), with one exception. In cohort 3, study participants were slightly more likely to be female compared to nonparticipants, a finding reflective of the fact that absenteeism in these schools was greater for ninth-grade males than females.

Each year, some students were missing from the sample (primarily due to absenteeism or relocation) and were replaced with other students who were recruited into the study from the same schools. The reasons why some wave 1 participants dropped out of the study were very similar to the reasons why wave 1 nonparticipants were added. Approximately 82% of the former transferred to schools that were not participating in the study. The rest were simply absent on the day of testing. Conversely, approximately 79% of the latter transferred from nonparticipating into participating schools over the course of the study. Again, the rest were listed as absent on the testing day. To be included in data analysis, students had to have participated in at least two waves of data collection. By the end of the study, a total of 892 students had participated, with 129 (14.5%) having exactly 2 waves of data, 301 (33.7%) having exactly three waves of data, and 462 (51.6%) having all four waves of data. Comparing these three groups of participants revealed no significant differences on any of the study variables.

At wave 1, the sample consisted of 161 second graders; 176 fourth graders, and 178 sixth graders. At wave 2, the sample consisted of 173 third graders, 146 fifth graders, and 136 seventh graders. At wave 3, the sample consisted of 164 fourth graders, 166 sixth graders, and 145 eighth graders. At wave 4, the sample consisted of 149 fifth graders, 160 seventh graders, and 98 ninth graders. Approximately, 55.5% of the overall sample was female. The sample was racially heterogeneous, including 63.5% African American, 28.3% White, 3.6% Latino, 1% Asian American, .4% Native American, and 3.2% “other” or “mixed” race. Annual family incomes ranged from less than \$10,000 to more than \$120,000 (*Mdn* = \$25,000).

²To examine school differences, we conducted a series of MANOVAs, comparing the seven schools on the CESD, CDI, CSQ, and CTI-C, one analysis per cohort. All three MANOVAs were significant, although effect sizes were generally small to moderate (i.e., the largest univariate, pairwise effect size was a Cohen's *d* of .31). Students at these schools also differed in age and parent-reported family income. Consequently, we followed up these analyses with MANCOVAs, controlling for participant age and family income. All three of these analyses (one per cohort) were nonsignificant (*ps* > .35). In other words, the only significant school differences were explained by child age and parent income.

Measures

Cognitive Triad Inventory for Children—(CTI-C; Kaslow et al., 1992). The CTI-C consists of 36 questions that assess a child's view of self, future, and world. Twelve items tap into each of these three domains; within these domains, half of the items are positively phrased, and half are negatively phrased. Participants respond based on how they feel "right now." Items are assigned scores of 0 (*no*), 1 (*maybe*), or 2 (*yes*), with negatively phrased items reverse-scored, such that higher scores represent the presence of more positive views/less negative views. (Note: this is opposite the valence of the other measures used in this study; thus, the relations between the CTI-C and other measures were expected to be negative.) Sample items include "I am a good person" and "Nothing is likely to work out for me." LaGrange et al. (2008) found support for good convergent and construct validity for the CTI-C in relation to measures of other cognitive constructs; however, no support emerged for the existence of domain-specific subscales. Kaslow et al. (1992) found high full-scale internal consistency reliability of the CTI-C ($\alpha = .92$). Examining internal reliability of the CTI-C by grade level in the current study revealed no consistent pattern of age-related differences. Cronbach's alphas ranged from .86–.94.

Automatic Thoughts Questionnaire—(ATQ; Hollon & Kendall, 1980). The ATQ is a 30-item questionnaire that assesses negative automatic thoughts. On 5-point scales, individuals rate (1 = *not at all*; 5 = *all the time*) how often in the past week they have had specific negative thoughts. Scores range from 30 to 150 with higher scores representing higher levels of negative automatic thoughts. Sample items include "Something has to change" and "I don't think I can go on." The ATQ was designed to assess spontaneous negative self-statements and intrusive cognitions experienced by depressed adults. Kazdin (1990) assessed the validity of the ATQ in a clinical sample of 250 children (ages 5–13) and found a .96 internal consistency, moderate item-total score correlations ($r_s = .39-.81$), and good convergent and discriminant validity. To ensure comprehension of the questions, we used slight wording changes as recommended by Kazdin (1990). Cronbach's alphas for the ATQ in the current study ranged from .89 to .96. Reliability estimates did not systematically covary with age or cohort.

Children's Depression Inventory—(CDI; Kovacs, 1981). The CDI is a 27-item self-report questionnaire that assesses the number and severity of depressive symptoms in children. Each of the items consists of three statements scored from 0 to 2, in order of increasing severity. Children select one sentence from each triad that best describes themselves for the past two weeks (e.g., "I am sad once in a while," "I am sad many times," or "I am sad all the time"). Psychometric studies of the CDI have shown high degrees of internal consistency, test-retest reliability, and construct validity, especially in nonclinic populations (Carey, Faulstich, Gresham, & Ruggiero, 1987; Kazdin, French, & Unis, 1983; Kovacs, 1985; Lobovits & Handal, 1985; Saylor, Finch, Spirito, & Bennett, 1984; Smucker, Craighead, Craighead, & Green, 1986). Due to concerns expressed by school administrators, we dropped the suicide item from the questionnaire. This 26-item version of the CDI has shown a high degree of internal consistency in prior research ($\alpha = .90$; Cole, Hoffman, Tram, & Maxwell, 2000). Timbremont, Braet, and Driessens (2004) found good predictive and discriminant validity of children's CDI scores in predicting depressive disorders in a

clinic-referred sample. Cronbach's alphas in the current study ranged from .73 to .88. Reliability estimates did not systematically covary with age or cohort.

Center for Epidemiological Studies–Depression Scale for Children—(CES-DC; Weissman, Orvaschel, & Padian, 1980). The CES-DC is a 20-item self-report inventory based on the Center for Epidemiological Studies–Depression Scale (CES-D; Radloff, 1977), with slight modifications to some of the items to facilitate children's understanding. To aid comprehension, we created a chart to serve as a visual aid to assist the younger children (second through fourth graders) in answering the questions. On 3-point scales (0 = *rarely or none of the time*; 2 = *most or all of the time*), participants rate how often they experienced each symptom during the past week (e.g., "I felt like crying this week"). Weissman et al. (1980) provided evidence of convergent validity with the CDI in children. Faulstich, Carey, Ruggiero, Enyart, and Gresham (1986) reported good internal consistency, 2-week test–retest reliability, and construct validity for psychiatric inpatient children and adolescents. Further psychometric support for the use of the measure with children and adolescents derives from studies by Fendrich, Weissman, and Warner (1990) and Hilsman and Garber (1995). In the current study, Cronbach's alphas ranged from .81 to .87. Reliability estimates did not systematically covary with age or cohort.

Procedure

Doctoral psychology students and advanced undergraduates administered the questionnaires during regular school hours. The measures included in this study were a subset of instruments from a larger study (Cole et al., 2008). The questionnaires were printed on optical scan sheets to allow for computerized scoring. In order to control for order effects, we counterbalanced the order of questionnaires. A research assistant worked one-on-one with each second grader, writing the responses as the child replied verbally or pointed at the answer on various response charts. We met with the third and fourth graders in small groups (3–4 students). With fifth through ninth graders, we administered the measures in large groups (20–30 students). In each case, a research assistant read the items aloud, requiring all students to proceed at the same pace. In the large groups, additional research assistants circulated around the classroom answering questions that arose. Research assistants made follow-up visits to the schools to collect data from students that were absent from the group administrations. This procedure was repeated once per year for four consecutive years.

Results

We tested our hypotheses with a series of TSO models. We began with four univariate models—one for each of the four measures included in the study. We then tested a series of bivariate models, each with one cognitive and one depression measure. Each model was run as a multigroup analysis with three groups corresponding to the three cohorts of participants. We used full information maximum likelihood estimation to handle the various patterns of missing data.

Preliminary Analyses

Table 1 displays the correlations among the four measures for each of the three cohorts. The correlations were computed using multiple imputation methods (SAS PROC MI and PROC MIANALYZE) to handle missing data; however, the TSO models were tested using FIML methods. The two methods yield asymptotically identical results when the missing at random assumptions hold. Box's test of homogeneity of covariance matrices revealed significant differences between all three cohorts ($ps < .01$). Consequently, we used multigroup SEM data analytic methods to examine cohort differences more completely. Also in Table 1 are the means and *SDs* for all measures. Mixed model analyses (with Cohort as a between-subjects effect and Grade as a within-subjects effect) revealed significant linear trends over grade for the CES-DC, ATQ, and CTI-C ($ps < .05$), suggesting that depressive symptoms and negative cognitions increase with grade level. (Note: the CTI-C is scaled in the opposite direction from the ATQ. These trends are evident in Figure 1. Cohort effects were nonsignificant, $ps > .20$. Test of nonlinear effects were also nonsignificant, $ps > .20$.³ All four measures showed a degree of skewness and kurtosis that were generally within the ranges for which maximum likelihood procedures typically provide robust estimates (e.g., Amemiya & Anderson, 1990; Browne, 1987; Browne & Shapiro, 1988; Mooijart & Bentler, 1991; Olsson, Foss, Troye, & Howell, 2000; Shapiro, 1986). Specifically, skewness estimates ranged from -1.09 for the CTI-C to 0.36 for the ATQ, and kurtosis estimates ranged from -0.44 for the CES-DC to 1.18 for the CDI.

Univariate TSO Models

The creation of univariate models allowed us to examine parameter estimates of each measure on its own. Figure 2 depicts a univariate TSO model applied to four waves of CES-DC data. We tested similar models for the CTI-C, CDI, and ATQ. To extract latent variables, we randomly divided each measure into three parcels. We formed different random parcels for each wave. In Figure 2, the three parcels of CES-DC items are shown loading onto the latent depression variable at each of the four time points corresponding to our four waves of data collection (Dep 1 through Dep 4). The variance for each of these four latent variables is then partitioned into that which is explained by a single TI factor and one of four TV factors (TV 1 through TV 4). The autoregressive paths ($a1$ through $a3$) connecting the TV factors represent the predictive relations from each occasion to the next for a given cohort (or group, labeled g). The variance of TV at time t that is not predicted by TV at $t-1$ is explained by the residuals. For each cohort, the expected value and variance of TI are depicted as $E(TI_g)$ and $V(TI_g)$, respectively. For TV1, the mean and variance in each group is $E(TV1_g)$ and $V(TV1_g)$, respectively. In each cohort, the latent intercepts for TV at

³Although we had no hypotheses about sex or race, our sample was diverse with regard to both of these demographics. Consequently, we tested for mean and covariance differences between males and females and between Caucasian and African-American participants. First, regarding sex, a series of MANOVAs (one for each cohort) on the CESD, CDI, CSQ, and CTI-C revealed no significant multivariate differences between males and females participants in any cohort, $ps > .10$. Univariate tests revealed that boys in the youngest cohort at wave 4 had larger CTI-C scores than did girls ($p < .046$). Any correction for even the family-wise alpha would have eliminated this finding. Box's tests of homogeneity of covariance matrices across gender, one for each cohort, were all nonsignificant ($ps > .10$). Second, MANOVAs on race revealed significant differences between Caucasian and African-American participants in cohorts 1 and 2, $ps < .001$. African-American students scored higher than Caucasian students on the CESD and the CTI. We also noted significant differences on parent-reported family annual income, with Caucasian families reporting substantially higher incomes ($\sim \$47,613$) than did African-American families ($\sim \$21,443$), $p < .001$. When we repeated these MANOVAs controlling for family income, all race differences were nonsignificant, $ps > .20$.

waves 2, 3, and 4 are $\text{Int}(\text{TV}_{2g})$, $\text{Int}(\text{TV}_{3g})$, and $\text{Int}(\text{TV}_{4g})$, respectively. We set one manifest variable factor loading for each latent variable to unity, to identify the model. Similarly, the unstandardized loadings of the Dep latent variables onto the TI and TV factors were also set to 1.0. We constrained the TI factor means and variances to be equal across groups. We also fixed the variance of the cohort 1 TV factor to be zero. Collectively, these constraints identified the model and allowed us (a) to define TI factor variance as the portion of the total variance that is completely stable across waves and across cohorts, and (b) to define the intercepts of TV to represent deviations around an invariant grand mean of TI.

For each measure (i.e., ATQ, CTI-C, CDI, CES-DC), we tested three hierarchically nested versions of this model. Model 1 served as the base model in which we placed no constraints on model parameters other than those described above. For all variables, this model fit the data well by all criteria except the chi-square. With large sample sizes, however, the chi-square can reveal small discrepancies between the model and the data to be significant. We designed Model 2 as a comparison to Model 1 in order to test for cohort effects on autoregressive paths, latent means, and latent intercepts. Specifically, Model 2 contained the following constraints: (a) $a_{31} = a_{12}$, (b) $a_{32} = a_{13}$, (c) $\text{Int}(\text{TV}_{31}) = E(\text{TV}_{12})$, (d) $\text{Int}(\text{TV}_{41}) = \text{Int}(\text{TV}_{22})$, (e) $\text{Int}(\text{TV}_{32}) = E(\text{TV}_{13})$, and (f) $\text{Int}(\text{TV}_{42}) = \text{Int}(\text{TV}_{23})$. In Model 3 we added within cohort equivalence constraints on the auto-regressive paths: $a_{11} = a_{21} = a_{31} = a_{12} = a_{22} = a_{32} = a_{13} = a_{23} = a_{33}$. Goodness-of-fit indices for all of these models are presented in Table 2. These include the chi-square, the root-mean-square-error of approximation (RMSEA; Steiger & Lind, 1980), the Tucker-Lewis index (TLI; Tucker & Lewis, 1973), and the comparative fit index (CFI; Bentler, 1990). RMSEA values less than .06 indicate a good fit (Hu & Bentler, 1999). TLI and CFI values greater than .95 indicate a good fit (Hu & Bentler, 1999). All models fit the data well. Furthermore, changes in chi-square between pairs of nested models were nonsignificant. Consequently, we based all parameter estimates on the most parsimonious Model 3.

Table 3 displays key parameter estimates, including the TI factor mean and variance, the TV factor means (or intercepts) and variances, and the standardized autoregressive path coefficients for each model. It also displays the proportions of variance due to TI versus TV. We found significant TI variance with all measures except for the CDI. Significant TV variance was found at all grade levels on all four measures. In other words, a TI and a TV factor emerged for the ATQ, CTI-C, and CES-DC. For the CDI, only the TV factor was significant. The proportion of TI variance to total variance ranged from .17 to .22 on the ATQ, from .34 to .54 on the CTI-C, from .31 to .50 on the CES-DC, and from .02 to .05 on the CDI. Autoregressive paths were significant for the ATQ (ranging from 0.29 to 0.44), CDI (ranging from 0.52 to 0.79), and the CES-DC (ranging from 0.17 to 0.25), but not for the CTI-C.

Bivariate TSO Models

After testing the four univariate TSO models, we paired each of the cognitive measures with each of the measures of depressive symptoms to form four bivariate models. Figure 3 depicts one such bivariate model, involving the ATQ and the CES-DC. The cross-lag paths (c_{1g} through c_{3g} and d_{1g} through d_{3g}) represent the predictive relations between the TV

component of the ATQ and the TV component of the CES-DC. In each model, we allowed the residual of each construct to correlate with the same-wave residual of the other construct (paths e_g , f_g , and h_g). Correlations were also allowed between the two TV factors at wave 1 and between the two TI factors. In addition to the parameterizations and constraints described for the univariate models, the covariance of the latent TI cognition variable with the latent TI depression variable was also constrained across groups to aid convergence.

For each pair of variables, we tested four models. Model 1 served as a base model, containing all of the previously described constraints, but containing no equality constraints on either the autoregressive paths or the cross-lag paths. According to all indices (except the chi-square), Model 1 fit the data well for all four pairs of variables (see Table 4). In Model 2, we added cross-cohort equality constraints on selected autoregressive paths and cross-lag paths so that the comparison of Model 2 to Model 1 would test cohort effects on the regression weights (e.g., $a_{31} = a_{12}$, $b_{31} = b_{12}$, $c_{31} = c_{12}$, $d_{31} = d_{12}$, $a_{32} = a_{13}$, $b_{32} = b_{13}$, $c_{32} = c_{13}$, $d_{32} = d_{13}$). These constraints did not perturb the fit of any of the models (see Table 4) and resulted in an increase in the reliability of the parameter estimates. Thus, we used Model 2 to examine the parameter estimates of interest and as a basis to which Models 3 and 4 could be tested. As we were also interested in developmental changes in the selected path coefficients, we constructed two additional models. In Model 3, we constrained all cognition $\rightarrow$ depression paths to be equal (i.e., $c_{11} = c_{21} = c_{31} = c_{12} = c_{22} = c_{32} = c_{13} = c_{23} = c_{33}$). As some of these constraints constituted the cohort-effect constraints in Model 2, Model 3 had only 6 more degrees of freedom than Model 2. As shown in Table 4, these constraints did not adversely affect goodness-of-fit. Conversely, Model 4 constrained all depression $\rightarrow$ cognition paths to be equal (i.e., $d_{11} = d_{21} = d_{31} = d_{12} = d_{22} = d_{32} = d_{13} = d_{23} = d_{33}$). Model 4 also had 6 more degrees of freedom than Model 2. As shown in Table 4, Model 4 provided a significantly worse fit to the data compared to Model 2, in all four of our analyses. Table 5 displays estimates of the cross-lag path coefficients, autoregressive path coefficients, and correlations involving key variables in each model.

The focal question of the current study was whether or not maladaptive cognitions and depressive symptoms prospectively predict one another after controlling for the completely stable aspects of each construct. The left-side columns of Table 5 address this question. These values are the estimates of the cross-lag path coefficients from Model 2 for each pair of variables. From columns 1 and 2, we see no support for the hypothesis that prior maladaptive cognition predicts later depressive symptoms. Of the 28 path coefficients (from 4 models $\times$ 7 lags) only 3 reached a .05 level of statistical significance, and they are actually in a direction opposite to that which would be expected according to theory (given that the CTI-C is scored such that high scores represent healthy, adaptive cognitions).

The second 2 columns of Table 5 display estimates of the relation between prior depressive symptoms and later maladaptive cognitions. Out of 28 coefficients, 14 were significant with all in the direction predicted by theory (i.e., increased depressive symptoms predicted increased negative cognitions). Significant coefficients occurred more often at the higher grade levels and had standardized values that ranged from 0.30 to 0.87 (when the ATQ was paired with either the CDI or the CES-DC) and from -0.31 to -0.80 (when the CTI-C was paired with either measure of depression).

Two other sets of parameters were the auto-regressive path coefficients and the correlations between the latent TI variables. The auto-regressive path coefficients are displayed in the latter four columns of Table 5. For measures of depression, 17 out of 28 were significant. For measures of cognition, only six were significant (and one of those was negative). The latent TI correlations were all quite large. Specifically, correlations between the invariant components of depression and cognition were 0.88 for the ATQ and CDI, 0.91 for the ATQ and CES-DC, -0.79 for the CTI-C and CDI, and -0.88 for the CTI-C and CES-DC.

We tested our developmental hypotheses in the context of the bivariate TSO model. Specifically, comparisons of Model 3 to Model 2 test whether the effect of cognition on depressive symptoms changes from grade level to grade level. As shown in Table 4, none of these tests were significant ($ps > .07$). Comparisons of Model 4 to Model 2 test whether the effect of depressive symptoms on cognition changes with grade level. These tests were all significant ($ps < .05$). The developmental pattern was clear in every analysis; the effect of depression on cognition was larger (and more likely to be statistically significant) at the older grade levels.

Measurement Overlap

One possible criticism of the previous results is that the measures included in these analyses contained some content overlap. Some items in the two depression measures assessed highly cognitive content. Consequently, we deleted from the CDI and CES-DC those items that either (a) contained primarily cognitive content and/or (b) yielded evidence of consistent cross-loadings onto a factor on which all items from one of the cognitive measures loaded in an item-level confirmatory factor analyses (as in Lengua, West, & Sandler, 1998). Conversely, items from the two cognitive measures were deleted if they either (a) contained primarily depressive content and/or (b) yielded consistent evidence of cross-loadings onto a factor on which all items from one of the depression measures loaded.⁴ The complete list of deleted items appears in the Appendix. After removing these items, we reanalyzed the data. As before, all models fit the data well, and the overall pattern of results was unchanged. Standardized path coefficients for the cross-lag paths were close to those in the original models (on average, $\pm .02$). None of the cross-lag paths that were significant in the original analyses became nonsignificant, or vice versa. In all cases, the direction of the effects remained the same. The only noteworthy change was that correlations between the latent TI variables were somewhat smaller in the reanalysis: 0.82 for the ATQ and CDI, 0.82 for the ATQ and CES-DC, -0.71 for the CTI-C and CDI, and -0.78 for the CTI-C and CES-DC.

Discussion

Four major findings derived from this study. First, we discovered that both time-invariant and time-varying factors characterize three of our four measures of maladaptive cognition and depressive symptoms. Second, we found support for the depression $\rightarrow$ cognition model, but not the cognition $\rightarrow$ depression model. Third, the strength of the depression $\rightarrow$ cognitions

⁴To avoid capitalization on chance, we restricted statistical exclusion to items that exhibited cross-loadings at more than one wave. Items that were excluded because of the cross-loading criteria represented a subset of those excluded because of the content analytic criteria.

relation increased with age. Fourth, the time-invariant components of cognition and depression were highly correlated. Implications of these findings, as well as limitations and suggestions for future research, are described below.

Our first finding pertained to the longitudinal structure of our measures. Researchers have long been aware that measures of depressive symptoms and depressive cognitions are highly stable over time. Our application of a TSO structural equation model (Cole et al., 2005) revealed that three out of four of such measures used in the current study can be broken into two types of longitudinal factors: a time-invariant (or trait-like) factor and a series of time-varying (more state-like) factors. More specifically, a time-varying component emerged for all four measures, account for approximately half of the variance in the CTI-C and the CES-DC, 81% of the reliable variance in the ATQ, and virtually all of the variance in the CDI. Time-invariant factors emerged for three of the four measures, accounting for significant variance in the ATQ (~19%), the CTI-C (~53%) and the CES-DC (~42%). In the case of the CDI, a time-invariant factor did not emerge but the time-varying component that emerged was nevertheless highly stable over time (with standardized auto-regressive coefficients ranging from 0.52 to 0.81). We should note, however, that the time-invariant factor for the CDI was not perfectly stable. Thus, for all measures of depressive symptoms and cognitions, we discovered a highly stable component, either a time-varying component that accounted for almost all of the variance or a time-invariant factor that accounted for 19% to 48% of the variance.

The existence of a strong time-invariant component for the cognitive measures helps to explain the high stability of individual differences on such measures that have often been reported (e.g., Gotlib et al., 1993; Stewart et al., 2004). This stable trait-like component also lends support to theoretical claims about the stability of depressive schemas (Beck, 1987; Clark et al., 1999). This support relies on the idea that the time-invariant latent variable underlying a time-series of surface-level cognitive measures represents the effects of an underlying stable depressive schema. These results do not explain other reports (e.g., Hankin, 2008; Lee & Hankin, 2009) of relatively low stability over relatively short time intervals for measures like the Children's Dysfunctional Attitudes Scale (CDAS; Lewinsohn, Joiner, & Rohde, 2001). We do note, however, that the CDAS is a short measure (only nine items) with modest reliability ($\alpha = .70$), factors that could attenuate its test-retest correlations.

Our second finding was that depressive symptoms predicted later maladaptive cognitions but not vice versa. All four of our bivariate models provided moderate to strong support for depression $\rightarrow$ cognition models. One such model is Lewinsohn, Steinmertz, Larson, and Franklin's (1981) scar hypothesis. The scar model suggests that negative cognitions persist after a major depressive episode, potentially increasing risk for relapse. Although the current study did not focus on episodes of clinical depression per se, it does lend credence to such theories at a subclinical level, insofar as depressive symptoms predicted subsequent negative cognitions. During middle childhood and early adolescence, one's cognitive style and self-concept are still under construction (Beck et al., 1979; Cole, 1996; Cole et al., 2001; Erikson, 1968). During these phases of development, individuals may be particularly vulnerable to forming beliefs and schemas that will prove difficult to alter once in place.

Results of the current study suggest that experiencing even subclinical levels of depressive symptoms during childhood and early adolescence can contribute to the development of a negative cognitive style.

Our lack of support for the cognition → depression model should be interpreted with caution, as such interpretations derive from support for the null hypothesis. With this in mind, we have noted elsewhere that cognitive diatheses are unlike genetic diatheses, in that people are not born with fully developed schemas and maladaptive cognitions. These must be learned (Cole & Turner, 1993; Turner & Cole, 1994; Cole, Martin, & Powers, 1997). The question therefore arises, can such cognitions predispose depression while they are still under construction? We suggest not. Until such negative self-schemas are fully developed, children engage in the strategic allocation of importance: divesting from negative self-domains and investing in the positive (Harter, 1987, 1990). We suggest that this very process mitigates the impact of negative self-cognitions (Cole, 1991; Cole et al., 2008).

Our third finding was that the time-invariant components of cognition and depression were very highly correlated with each other, with absolute values for r in the 0.70 to 0.80 range (after controlling for item-overlap). Such correlations may reflect several causal processes that are not part of the models tested in the current study. First, these correlations could be the result of depression → cognition or cognition → depression effects that take less than one year to unfold. If such short-term cause-effect relations do exist, they should be evident in longitudinal studies that utilize briefer interwave intervals, unless the effects are so fast or so intertwined as to be virtually inseparable (Teasdale, 1983a, 1983b). Second, these correlations reflect the effects of one or more “third variables” that drive both depressive symptoms and negative cognition. A stable component of one’s personality or a genetic predisposition, for example, could render an individual susceptible to both depressive symptoms and maladaptive cognitions. Third, the time-invariant factor that underlies maladaptive cognition and the time-invariant factor that may underlie depressive symptoms may be one and the same. This model is actually consistent with Beck’s suggestion that depressive cognitive schemas are a part of depression rather than a risk factor for the disorder: “I suggest that cognitive phenomena such as unmitigated negative self-references and expectancies are an *integral part of depression*; consequently, they are hardly capable of causing themselves” (Beck, 1984, p. 1113). We note, however, that the results of the current study suggest that this can be true only for the 19% to 53% of maladaptive cognitions (and/or depressive symptoms) that is time invariant.

Fourth, among older participants, stronger support emerged for the depression → cognition relation than it did among the younger children. In all four analyses, the depression → cognition relation was stronger and more likely to be statistically significant at the older grade levels. Furthermore, these age differences were statistically significant when depressive symptoms were measured by the CDI. The exact age at which depression → cognition effects emerged varied with the choice of measures: around grades 4–5 for the CTI-C and CDI, but not until grades 7–8 for the ATQ and CES → DC. Developmentalists have documented changes in children’s self-cognitions around the ages of 7 to 8 (Crain, 1996; Harter, 1986). Younger children typically have an inflated sense of their personal competence, regarding themselves as one of the best in most performance domains.

Theorists have attributed this to their tendency to rely upon intrapersonal comparisons when making such judgments, reasoning “I’m better at this than I was last year, therefore I’m good.” During the 7-to-8-year-old transition, children become increasingly capable of making interpersonal comparisons and grow increasingly motivated to do so. This “fall from grace” may represent the loss of a developmentally normative self-protection mechanism, leaving children more vulnerable to the negative cognitive effects of many things, including depression.

Certain limitations of the current study suggest avenues for future research. First, we have not tested the viability of our model for all potential subgroups. On the one hand, our examination of race and sex revealed no evidence of moderation—providing some support for the generalizability of our results across two major demographics. In depression research, such generalizability has often gone untested, and when it has been examined, results have been mixed (compare Herman, Ostrander, & Tucker, 2007; Jacobs et al., 2008; Kennard et al., 2006; Kistner, David, & White, 2003; Lemos-Miller & Kearney, 2006; Stein et al., 2010; Woolgar & Tranah, 2010). On the other hand, our examination of age differences did reveal evidence of significant moderation, potentially reflecting important developmental differences. Other potential moderators exist that we did not examine, including stressful life events (e.g., Abela, & Sullivan, 2003; Hilsman & Garber, 1995; Lewinsohn et al., 2001), hostile or coercive parenting (Bruce et al., 2006), parental mental health (Goodman & Gotlib, 2002), and socioeconomic status (Lovejoy, Graczyk, O’Hare, & Neuman, 2000). An important future direction would be to examine the moderational effects of such variables when applying the TSO model to depression-related cognitions.

Second, we used a 1-year time lag in this study. It is possible that the optimal time lag for detecting cognition → depression effects is shorter. If so, such effects could have come and gone between assessments, leading us to underestimate the complete effect of cognition on depressive symptoms. A study with shorter lag times could reveal longitudinal effects of depressive cognitions that were missed by the current study.

Third, our sample was limited to relatively nondepressed individuals. Although these findings shed light on the early stages of negative cognitions and depressive symptoms, we cannot assume that these relations generalize to more severely depressed children. Epidemiological studies have discovered relatively low rates of major depression during this period of development. One-month prevalence estimates range from 2% in late childhood (Costello et al., 2002; Rutter, 1994) to 6% during late adolescence (Blazer, Kessler, McGonagle, & Swartz, 1994). Replication of the current study with samples that include more severely depressed children would enhance the generalizability of the results.

Fourth, care should be taken not to overinterpret our nonsignificant findings about the effect of maladaptive cognition on depressive symptoms. Besides the typical reasons for not concluding in favor of the null hypothesis, this study focuses on only the CTI-C and the ATQ administered once per year to children and young adolescents. Measures of other kinds of maladaptive cognitions, administered more (or less) frequently to different age groups may generate very different results.

With these caveats in mind, our findings may have two implications for depression-related interventions in child and adolescent populations. First, the existence of a time-invariant component of children's maladaptive cognitions lends credence to the idea that cognitive behavior therapy must continuously challenge automatic thoughts and dysfunctional assumptions in order to affect the underlying depressive schema. Indeed, this is part of the reason researchers give the efficacy of CBT in preventing recurrence: it has worked to change the underlying schema (Clarke et al., 2001; Collins & Dozois, 2008; Compas et al., 2009; Horowitz & Garber, 2006). Second, our finding that child and adolescent depression contributes to the development of maladaptive cognitions suggests that successful depression interventions may have double impact. Not only might such interventions diminish depression directly but they may also forestall the development of maladaptive cognitive scars in the depression postdrome.

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Appendix. Items Deleted Because of Content Overlap

The following items were deleted for reanalysis, based on content overlap. Cognitive items were dropped from the CDI and CES-D. Items were dropped from the CTI-C and ATQ if they overlapped in content with one or more of the residual items on the CDI or CES-D.

CDI

Nothing will ever work out for me.

I do everything wrong.

I am bad all the time.

I am sure that terrible things will happen to me.

I hate myself.

All bad things are my fault.

I can never be as good as other kids.

Nobody really loves me.

CESD-C

I felt like I was just as good as other kids.

I felt like something good was going to happen.

I felt like things I did before didn't work out right.

I felt like kids I knew were not friendly or that they didn't want to be with me.

I felt people didn't like me this week.

I felt lonely, like I didn't have any friends.

CTI-C

Schoolwork is no fun.

I do my schoolwork okay.

The things I do every day are fun.

Bad things happen to me a lot.

ATQ

Nothing feels good anymore.

I can't get anything started.

Figure 1.
Means plotted by cohort and grade level. (Note: the ATQ and CTI are scaled in opposite directions.)

Figure 2.

Univariate TSO model of depressive symptoms. CES = Center for Epidemiological Studies Depression Scale for Children; TV = time-varying; TI = time-invariant.

Figure 3. Bivariate TSO model depicting relations between depressive symptoms and cognitive style. ATQ = Automatic Thoughts Questionnaire; CES = Center for Epidemiological Studies Depression Scale for Children; TV = time-varying; TI = time-invariant.

Table 1

Correlations, Means, and Standard Deviations for Cohorts 1, 2, and 3

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Cohort 1 (grades 2–5)																
1. CDI wave 1	1															
2. CES-DC wave 1	.33***	1														
3. ATQ wave 1	.36***	.31***	1													
4. CTTI-C wave 1	-.20*	-.34***	-.09	1												
5. CDI wave 2	.26**	.12	.15	.07	1											
6. CES-DC wave 2	.31***	.36***	.19~	-.12	.45***	1										
7. ATQ wave 2	.34***	.16~	.20*	-.03	.61***	.48***	1									
8. CTTI-C wave 2	-.38***	-.26**	-.25*	.08	-.50***	-.69***	-.43***	1								
9. CDI wave 3	.22*	.25**	.14~	-.02	.51***	.30***	.26***	-.33***	1							
10. CES-DC wave 3	.22*	.29***	.11	-.16	.26***	.35***	.14~	-.32***	.52***	1						
11. ATQ wave 3	.15	.15	.21*	.04	.40***	.33***	.41***	-.38***	.65***	.45***	1					
12. CTTI-C wave 3	-.33***	-.21*	-.22**	.24*	-.19*	-.26***	-.11	.31***	-.40***	-.57***	-.33***	1				
13. CDI wave 4	.19	-.05	-.17	.01	.29*	.16	.25*	-.23~	.41***	.39***	.33*	-.22*	1			
14. CES-DC wave 4	.28*	-.09	.09	.09	.41***	.17	.44***	-.27~	.36***	.32***	.37**	-.17	.60***	1		
15. ATQ wave 4	.18	.01	-.22	-.01	.28*	.24~	.32**	-.27*	.37***	.37***	.33*	-.21~	.73***	.70***	1	
16. CTTI-C wave 4	-.29*	-.03	.12	-.05	-.31*	-.14	-.34***	.30*	-.42***	-.34***	-.36**	.30**	-.59***	-.56***	-.62***	1
Mean	10.96	21.94	76.04	51.12	9.03	18.21	64.53	54.58	8.17	15.83	57.58	55.75	8.86	22.81	55.00	53.82
SD	6.44	10.14	22.05	15.83	6.36	11.28	22.87	10.90	6.94	11.35	21.05	11.32	7.86	10.76	22.94	11.97
Cohort 2 (grades 4–7)																
1. CDI wave 1	1															
2. CES-DC wave 1	.49***	1														
3. ATQ wave 1	.60***	.56***	1													

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
4. CTL-C wave 1	-.44***	-.52***	-.35***	1												
5. CDI wave 2	.37***	.49***	.39***	-.29***	1											
6. CES-DC wave 2	.35**	.46***	.35***	-.29***	.52***	1										
7. ATQ wave 2	.24*	.42***	.38***	-.27***	.77***	.50***	1									
8. CTL-C wave 2	-.32**	-.56***	-.33***	.43***	-.54***	-.71***	-.53***	1								
9. CDI wave 3	.32**	.41***	.34***	-.23*	.65***	.55***	.65***	-.48***	1							
10. CES-DC wave 3	.33***	.39***	.35***	-.19*	.44***	.48***	.39***	-.37***	.62***	1						
11. ATQ wave 3	.30*	.38***	.31**	-.31**	.51***	.51***	.55***	-.36***	.75***	.59***	1					
12. CTL-C wave 3	-.25*	-.36***	-.29***	.23*	-.40***	-.43***	-.39***	.40***	-.62***	-.71***	-.56***	1				
13. CDI wave 4	.37**	.36***	.36***	-.18	.51***	.48***	.56***	-.42***	.65***	.53***	.50***	-.42***	1			
14. CES-DC wave 4	.18	.36***	.27**	-.26**	.30**	.61***	.27*	-.46***	.53***	.55***	.51***	-.49***	.61***	1		
15. ATQ wave 4	.23	.18	.22	-.14	.27*	.43***	.43***	-.35***	.51***	.45***	.51***	-.36***	.82***	.60***	1	
16. CTL-C wave 4	-.11	-.31**	-.19	.28*	-.34***	-.50***	-.36***	.59***	-.58***	-.53***	-.47***	.57***	-.67***	-.72***	-.64***	1
Mean	8.55	17.80	62.99	52.92	9.08	14.07	61.63	56.95	8.17	13.44	50.69	56.76	7.58	19.63	49.86	58.29
SD	6.19	11.27	21.26	15.52	7.87	11.15	24.39	11.40	7.67	10.92	19.32	11.93	7.11	10.14	21.22	11.48

Cohort 3 (grades 6-9)

1. CDI wave 1	1															
2. CES-DC wave 1	.58***	1														
3. ATQ wave 1	.67***	.55***	1													
4. CTL-C wave 1	-.41***	-.43***	-.36***	1												
5. CDI wave 2	.49***	.29***	.31***	-.19~	1											
6. CES-DC wave 2	.48***	.53***	.39***	-.23*	.56***	1										
7. ATQ wave 2	.36***	.26***	.30***	-.28**	.77***	.53***	1									
8. CTL-C wave 2	-.44***	-.41***	-.25***	.23*	-.58***	-.71***	-.55***	1								
9. CDI wave 3	.40***	.42***	.35***	-.28*	.60***	.47***	.45***	-.37***	1							

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
10. CES-DC wave 3	.47***	.49***	.36***	-.40***	.49***	.56***	.43***	-.47***	.62***	1						
11. ATQ wave 3	.40***	.51***	.49***	-.40***	.50***	.47***	.48***	-.34***	.74***	.63***	1					
12. CTI-C wave 3	-.45***	-.43***	-.33***	.41***	-.65***	-.53***	-.51***	.52***	-.76***	-.70***	-.59***	1				
13. CDI wave 4	.41***	.30*	.20	.01	.61***	.51***	.42***	-.62***	.71***	.55***	.50***	-.61***	1			
14. CES-DC wave 4	.40***	.44**	.28*	-.07	.35***	.37***	.23~	-.32**	.36***	.61***	.35***	-.39***	.64***	1		
15. ATQ wave 4	.32~	.45*	.32~	-.14	.34***	.39*	.42***	-.33***	.50***	.44***	.46***	-.42***	.61***	.65***	1	
16. CTI-C wave 4	-.32*	-.27	-.06	.17	-.40***	-.42***	-.25*	.55***	-.42***	-.41***	-.34***	.58***	-.67***	-.49***	-.39***	1
Mean	9.77	15.38	59.17	56.18	9.25	15.83	55.27	56.38	9.01	15.00	56.47	57.53	9.20	23.91	55.24	55.72
SD	6.76	11.03	24.35	13.14	7.03	11.49	24.10	11.76	7.69	11.29	24.26	11.29	7.49	12.44	24.21	12.04

Note. ATQ = Automatic Thoughts Questionnaire; CTI-C = Cognitive Triad Inventory for Children; CDI = Children's Depression Inventory; CES-DC = Center for Epidemiological Studies Depression Scale for Children. The CTI-C and ATQ are scaled in opposite directions.

* $p < .05$.

** $p < .01$.

Table 2

Goodness-of-Fit for Three Nested Univariate Models for Each Measure

Model	df	χ^2	χ^2	RMSEA	TLI	CFI
ATQ						
Model 1: base model	164	194.20	—	0.014	0.99	0.99
Model 2: no cohort effects	170	205.60*	11.40	0.015	0.99	0.99
Model 3: no within- or between-cohort effects	176	212.20*	6.60	0.015	0.99	0.99
CTI-C						
Model 1: base model	164	212.95*	—	0.018	0.98	0.99
Model 2: no cohort effects	170	223.33*	10.38	0.019	0.98	0.99
Model 3: no within- or between-cohort effects	176	233.42*	10.09	0.019	0.98	0.99
CDI						
Model 1: base model	164	198.00*	—	0.015	0.98	0.99
Model 2: no cohort effects	170	203.40*	5.40	0.015	0.98	0.99
Model 3: no within- or between-cohort effects	176	214.10*	10.70	0.016	0.98	0.99
CES-DC						
Model 1: base model	164	193.00	—	0.014	0.98	0.99
Model 2: no cohort effects	170	201.80*	8.80	0.014	0.98	0.99
Model 3: no within- or between-cohort effects	176	214.60*	12.80*	0.016	0.98	0.98

Note. ATQ = Automatic Thoughts Questionnaire; CTI-C = Cognitive Triad Inventory for Children; CDI = Children's Depression Inventory; CES-DC = Center for Epidemiological Studies Depression Scale for Children; TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Squared Error of Approximation.

* $p < .05$.
 ** $p < .01$.
 *** $p < .001$.

Table 3

Estimates for Univariate Models

Grade	TI factor		TV factor		Proportion of variance due to			Standardized auto-regression coefficient		
	Mean	Variance	Mean	Variance	TI	TV	Coh. 1	Coh. 2	Coh. 3	
ATQ										
2	39.06***	21.42**	0.00	84.40***	0.20	0.80				
3	39.06***	21.42**	-6.50***	104.58***	0.17	0.83	0.31***			
4	39.06***	21.42**	-7.77***	76.78***	0.22	0.78	0.39***			
5	39.06***	21.42**	-6.60***	103.28***	0.17	0.83	0.30***	0.29***		
6	39.06***	21.42**	-10.63***	86.19***	0.20	0.80		0.44***		
7	39.06***	21.42**	-9.32***	97.75***	0.18	0.82		0.31***	0.33***	
8	39.06***	21.42**	-8.33***	104.01***	0.17	0.83			0.37***	
9	39.06***	21.42**	-6.70***	88.05***	0.20	0.80			0.36***	
CTI-C										
2	25.41***	14.25***	0.00	20.06***	0.42	0.58				
3	25.41***	14.25***	2.47***	12.18***	0.54	0.46	-0.11			
4	25.41***	14.25***	1.90**	27.46***	0.34	0.66	-0.03			
5	25.41***	14.25***	2.75***	13.26***	0.52	0.48	-0.05	-0.10		
6	25.41***	14.25***	2.57***	20.52***	0.41	0.59		-0.04		
7	25.41***	14.25***	3.40***	12.03***	0.54	0.46		-0.06	-0.07	
8	25.41***	14.25***	3.39***	11.61***	0.55	0.45			-0.05	
9	25.41***	14.25***	2.71**	12.34***	0.54	0.46			-0.04	
CDI										
2	5.19***	0.26	0.00	4.49*	0.05	0.95				
3	5.19***	0.26	-0.66**	7.72**	0.03	0.97	0.55***			
4	5.19***	0.26	-0.70***	8.27***	0.03	0.97	0.64***			
5	5.19***	0.26	-0.08	13.30***	0.02	0.98	0.62***	0.52***		

Grade	TI factor		TV factor		Proportion of variance due to			Standardized auto-regression coefficient		
	Mean	Variance	Mean	Variance	TI	TV	Coh. 1	Coh. 2	Coh. 3	
6	5.19***	0.26	-0.50*	10.51***	0.02	0.98		0.75***		
7	5.19***	0.26	-0.46*	10.79***	0.02	0.98		0.76***	0.65***	
8	5.19***	0.26	-0.27	11.40***	0.02	0.98			0.71***	
9	5.19***	0.26	0.14	9.13***	0.03	0.97			0.79***	
CES-DC										
2	11.04***	10.32	0.00	12.52***	0.45	0.55				
3	11.04***	10.32	-1.73***	15.87***	0.39	0.61	0.19**			
4	11.04***	10.32	-2.49***	16.12***	0.39	0.61	0.21**			
5	11.04***	10.32	-2.25***	22.59***	0.31	0.69	0.17**	0.21**		
6	11.04***	10.32	-3.53***	14.86***	0.41	0.59		0.22**		
7	11.04***	10.32	-1.12*	14.06***	0.42	0.58		0.25**	0.19**	
8	11.04***	10.32	-3.10***	12.20***	0.46	0.54			0.25**	
9	11.04***	10.32	1.90**	10.53***	0.50	0.50			0.23**	

Note. Estimates with differing subscripts within a panel are significantly different from one another ($p < .05$). ATQ = Automatic Thoughts Questionnaire; CTLC = Cognitive Triad Inventory for Children; CDI = Children's Depression Inventory; CES-DC = Center for Epidemiological Studies Depression Scale for Children.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 4

Goodness-of-Fit for Four Bivariate TSO Models for Each Pair of Measures

Model	df	χ^2	Compare models	df	χ^2	RMSEA	TLI	CFI
CDI and ATQ								
1. Base model	739	1193.01***	—	—	—	.026	.97	.98
2. No cohort effects	747	1200.03***	1 vs.2	8	7.02	.026	.98	.98
3. Equivalent dep → cog effects	754	1212.99***	3 vs.2	6	12.96*	.026	.98	.98
4. Equivalent cog → dep effects	754	1205.74***	4 vs.2	6	5.71	.026	.98	.98
CDI and CTI-C								
1. Base model	739	1492.91***	—	—	—	0.034	0.96	0.97
2. No cohort effects	747	1502.11***	1 vs.2	8	9.20	0.034	0.96	0.97
3. Equivalent dep → cog effects	754	1521.92***	3 vs.2	6	19.81**	0.034	0.96	0.97
4. Equivalent cog → dep effects	754	1513.74***	4 vs.2	6	11.63	0.034	0.96	0.97
CES-DC and ATQ								
1. Base model	739	1302.22***	—	—	—	0.029	0.97	0.98
2. No cohort effects	747	1312.11***	1 vs.2	8	9.89	0.029	0.97	0.98
3. Equivalent dep → cog effects	754	1326.91***	3 vs.2	6	14.80*	0.029	0.97	0.98
4. Equivalent cog → dep effects	754	1321.75***	4 vs.2	6	9.64	0.029	0.97	0.98
CES-DC and CTI-C								
1. Base model	739	1143.56***	—	—	—	0.025	0.98	0.98
2. No cohort effects	747	1152.34***	1 vs.2	8	8.78	0.025	0.98	0.98
3. Equivalent dep → cog effects	754	1165.93***	3 vs.2	6	13.59*	0.025	0.98	0.98
4. Equivalent cog → dep effects	754	1159.29***	4 vs.2	6	6.95	0.025	0.98	0.98

Note. ATQ = Automatic Thoughts Questionnaire; CTI-C = Cognitive Triad Inventory for Children; CDI = Children's Depression Inventory; CES-DC = Center for Epidemiological Studies Depression Scale for Children; TLI = Tucker Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Squared Error of Approximation.

* $p < .05$.

** $p < .01$.

 $p < .001$

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Table 5

Standardized (Std.) and Unstandardized (Unst.) Estimates of Cross-Lag Regression Coefficients for Four Bivariate Models

Lag ^a	Cog. → Dep.		Dep. → Cog.		Dep. → Dep.		Cog. → Cog.	
	Unst.	Std.	Unst.	Std.	Unst.	Std.	Unst.	Std.
Model 2 estimates for the ATQ and CDI								
2→3	-0.03	-0.08	0.47	0.26	0.43	0.38	-0.11	-0.12
3→4	-0.09	-0.27	1.06	0.31	0.96***	0.84	0.07	0.08
4→5	0.04	0.07, 0.08	1.01	0.30, 0.26	0.68***	0.55, 0.51	0.23	0.20, 0.20
5→6	0.07	0.22	1.81**	0.87	0.57*	0.59	-0.14	-0.21
6→7	-0.08	-0.21, -0.22	1.92**	0.71, 0.59	0.81***	0.89, 0.78	-0.26	-0.22, -0.29
7→8	-0.23*	-0.68	2.15**	0.85	1.05***	0.93	-0.32	-0.33
8→9	-0.10	-0.30	2.13**	0.77	1.03***	0.91	-0.26	-0.28
Model 2 estimates for the ATQ and CES-DC								
2→3	-0.08	-0.21	-0.58 ⁺	-0.28	0.18	0.20	0.08	0.07
3→4	-0.15*	-0.33	-0.30	-0.12	0.17	0.17	0.31**	0.34
4→5	0.05	0.09	0.18	0.08, 0.09	0.11	0.09, 0.12	0.27*	0.24, 0.24
5→6	0.09	0.24	0.24	0.12	0.07	0.07	0.26**	0.36
6→7	0.08	0.15, 0.18	0.24	0.12, 0.09	0.29 ⁺	0.30, 0.28	0.16	0.13, 0.15
7→8	0.05	0.11	0.67*	0.29	0.37*	0.40	0.17	0.18
8→9	-0.10	-0.25	0.51*	0.22	0.80***	0.76	0.22	0.25
Model 2 estimates for the CTI-C and CDI								
2→3	0.13*	0.43	0.23	0.14	0.19	0.08	-0.17*	-0.35
3→4	0.14	0.18	0.33	0.19	0.67***	0.56	-0.15	-0.14
4→5	0.01	0.00, 0.01	-0.54**	-0.34, -0.35	0.59***	0.49, 0.46	-0.06	0.07, 0.10
5→6	-0.18	-0.21	-0.70**	-0.52	0.52***	0.54	-0.24	-0.20
6→7	0.08	0.13, 0.14	-0.77***	-0.64, -0.54	0.63***	0.75, 0.60	-0.10	-0.10, -0.12
7→8	0.05	0.06	-0.82***	-0.80	0.79***	0.70	-0.35	-0.37

Lag ^a	Cog. → Dep.		Dep. → Cog.		Dep. → Dep.		Cog. → Cog.	
	Unst.	Std.	Unst.	Std.	Unst.	Std.	Unst.	Std.
8→9	0.20	-0.29	-0.56**	-0.41	0.45**	0.49	0.47	0.43
Model 2 estimates for the CTI-C and CES-DC								
2→3	0.14	0.23	0.10	0.14	0.14	0.14	-0.15	-0.35
3→4	0.36	0.29	-0.17	-0.14	0.25	0.25	-0.32	-0.28
4→5	0.04	0.02, 0.03	-0.21	-0.28, -0.24	0.11	0.10, 0.11	-0.01	-0.01, -0.02
5→6	0.25	0.20	-0.27	-0.21	0.09	0.08	-0.41	-0.32
6→7	0.11	0.15, 0.13	-0.36*	-0.29, -0.27	0.29*	0.33, 0.29	-0.25	-0.27, -0.28
7→8	0.23	0.24	-0.84**	-0.75	0.54**	0.59	0.48*	0.46
8→9	0.24	0.21	-0.38*	-0.37	0.57**	0.63	0.54*	0.51

Note. ATQ = Automatic Thoughts Questionnaire; CTI-C = Cognitive Triad Inventory for Children; CDI = Children's Depression Inventory; CES-DC = Center for Epidemiological Studies Depression Scale for Children. The CTI-C and ATQ are scaled in opposite directions.

^aLag means grade k to grade k + 1.

+ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.