Dynamic Longitudinal Associations between Parents' Depressive Symptoms and Children's Explanatory Style

By

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TABLE OF CONTENTS

LIST OF TABLES

Table Page

LIST OF FIGURES

Figure Page

CHAPTER I

INTRODUCTION

One in five children in the United States lives with a parent who has had depression (National Research Council and Institute of Medicine, 2009). Parental depression is a potent risk factor for psychiatric symptoms, both internalizing and externalizing, in children (Beardslee, Gladstone, & O'Conner, 2011; Goodman, Rouse, Connell, Broth, Hall, & Heyward., 2011). Offspring of depressed parents represent an important population for studying the processes underlying risk for depression. Goodman and Gotlib (1999) proposed several possible mechanisms through which this intergenerational transmission of psychopathology occurs, including genetic heritability, innate neuroregulatory dysfunction, the stressful context of living with a depressed parent, and children's exposure to parent's negative cognitions, behaviors, and affect.

Parental depression has been associated with negative parenting behaviors, such as displays of negative affect and antagonism toward their children, as well as disengagement and withdrawal (Lovejoy, Grczyk, O'Hare, & Neuman, 2000). Although these dysfunctional parenting behaviors are most apparent during a depressive episode, they often are maintained even after remission (Lovejoy et al., 2000; Weissman, Paykel, & Klerman, 1972). Such negative parenting behaviors have been linked with the development of children's negative cognitive styles (e.g., Garber & Flynn, 2001; Mezulis, Hyde, & Abramson, 2006), which are known to be a vulnerability for depression.

Cognitive models of depression propose that negative beliefs about the self, world, future, and the causes of life events significantly increase risk for depression, particularly in the

context of stress (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978; Beck, 1967, 1976). Studies have shown that negative cognitions significantly predict increases in depressive symptoms after exposure to stress in both adults (e.g. Abramson et al., 2002) and children (e.g., Abela & Hankin, 2008; Carter & Garber, 2011). According to the hopelessness theory of depression (Abramson et al., 1989), "individuals who make stable, global attributions, infer negative characteristics about the self, and anticipate negative consequences when negative life events occur are more likely to become depressed than individuals who do not exhibit this negative cognitive style." The Children's Attributional Style Questionnaire (Seligman, Kaslow, Alloy, Peterson, Tanenbaum, & Abramson, 1984), which measures these global, stable, and internal attributions in children correlates highly with children's depressive symptoms (Gladstone & Kaslow, 1995; Joiner & Wagner, 1995) and has been found to predict future levels of depressive symptoms (Seligman et al., 1984), particularly when children are experiencing stress (Dixon & Ahrens, 1992).

Children of depressed parents have been found to have more negative cognitive processing styles than offspring of non-depressed parents (e.g., Goodman, Adamson, Riniti, & Cole, 1994; Joorman, Talbot, & Gotlib, 2007; Murray, Woolgar, Cooper, & Hipwell, 2001; Taylor & Ingram, 1999). Moreover, maternal depression history has been found to significantly predict changes in children's self-worth and depressive symptoms (Garber & Cole, 2010).

In addition, the relation between mothers' and children's cognitions and symptoms may be bidirectional. For example, parents of children with psychopathology have been found to be at higher risk for depressive symptoms themselves (Civic & Holt, 2000), and mothers may be more likely to experience a depressive episode following a child's episode (Hammen, Burge, & Adrian, 1991). Indeed, dynamic changes over time between mothers' and children's depressive

symptoms have been reported (Kouros & Garber, 2010). Transactional patterns between children's symptoms and maternal depression have been found, with a variety of maladaptive child behaviors being associated with dynamic relations to parents' depressive symptoms (Gross, Shaw & Moilanen, 2008; Gross, Shaw, Burwell, & Nagin, 2009; Elgar, Curtis, McGrath, Waschbusch, & Stewart, 2003).

Studies have not yet examined, however, how dynamic changes in parents' depressive symptoms and children's cognitions are related. The primary purpose of the current study was to investigate this association between parents' depressive symptoms and children's cognitions over time. More specifically, we explored whether parents' depressive symptoms predicted children's subsequent attributional style, controlling for the autocorrelations among study variables.

A second aim of this study was to investigate the direction of the relation between parents' depressive symptoms and children's cognitions. That is, does children's attributional style predict parents' depressive symptoms, controlling for their prior symptoms. The third aim was to examine if the relation between parents' depressive symptoms and children's attributional style was bidirectional, and to explore whether a single dynamic systems model could account for changes over time in both parents' depressive symptoms and children's attributional style simultaneously.

CHAPTER II

METHOD

Participants

Participants were 227 parent-child dyads, with one parent and one child per family. Parents were either diagnosed with a current Major Depressive Disorder (n = 129) as defined in the *Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition* (DSM–IV; American Psychiatric Association, 1994) or were lifetime-free of mood disorders (n = 98), psychotic disorders, organic brain syndromes, or personality disorders, and during the child's life were free of adjustment disorders, anxiety disorders, or substance abuse/dependence, had not had more than eight sessions of psychotherapy, or had used psychotropic medication for the treatment of a psychiatric disorder.

Parents were 76% female and were between the ages of 24-62 years old (*Mean* = 42.19, *SD* = 6.92. Parents had between 7 and 22 years of education (*Mean* = 15.00, *SD* = 2.59) and reported as white, non-Hispanic (71.4%), African American (22%), Asian (1.8%), more than one race (1.8%), or chose not to report race/ethnicity (3%).

Exclusion criteria for parents were a lifetime diagnosis of any psychotic or paranoid disorder, organic brain syndrome, mental retardation, bipolar I or II, or a current or primary diagnosis of substance abuse or dependence, obsessive-compulsive disorder, eating disorder, certain personality disorders (antisocial, borderline, schizotypal), or unwillingness to participate in treatment for depression for parents experiencing an MDE.

Child participants were between 7- and 17-years-old (*Mean* = 12.53, *SD* = 2.33), 53% female, and reported as white (70.5%), African American (21.6%), Asian (.4%), more than one race (7%), or chose not to report race/ethnicity (n=.4%).

Exclusion criteria for the children were a developmental disability or a chronic medical condition. Children lived with the target parent at least half the time. For non-depressed families, the enrolled child was selected to be similar in age, sex, and race to a child in the sample of offspring of depressed parents.

Neither parents nor children differed significantly by risk group on any of the demographic variables, except parental education level [$t(217)=2.23$, p=.027]. Demographics for each risk group are reported in Table 1.

Measures

Parents' Psychopathology

The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 1997) was used to evaluate psychopathology in parents. A randomly selected subset of taped interviews was used to assess inter-rater reliability, yielding kappa coefficients $\geq$ .80.

Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) is an interview-based measure of the severity of depression. The 17-item version used here yields scores ranging from 0 to 52; higher scores indicate greater severity. The HRSD has high inter-rater reliability (i.e., $\geq$ .84). Intra-class correlation in this study was .96.

Beck Depression Inventory, Second Edition (BDI-II; Beck et al., 1996; Beck, Steer, & Garbin, 1988) is a 21-item self-report measure rated on a scale ranging from 0 (absence of

symptoms) to 3 (most severe level of the symptom). Scores can range from 0 to 63; higher scores indicate more depression. Coefficient alpha in this sample across all time points was ≥ .91.

Children's Measures

The *Children's Depression Inventory* (CDI; Kovacs, 1992) is a 27-item self-report measure of children's symptoms of depression rated on a 3-point scale. Total scores can range from 0 to 54, with higher scores indicating more depression. Coefficient alpha for the CDI at T1 in this sample was .84.

Children's explanatory style was assessed with the 48-item *Children's Attributional Style Questionnaire* (CASQ; Seligman et al., 1984), which measures children's beliefs about the causes of 24 positive and 24 negative events. Children select one of two possible causes for each event, either internal or external, stable or unstable, or global or specific. Lower total scores indicate a more depressive explanatory style, such that negative events would be explained using an internal, global, and stable cause, and positive events using an external, specific, and unstable cause. For example, "a good friend tells you that he hates you because:" either (a) "My friend was in a bad mood that day" (external), or (b) "I wasn't nice to my friend that day" (internal). Composite scores ranged from -.50 to 1.00, with higher scores indicating a more positive attributional style. Coefficient alphas for the total score in this sample ranged from .25-.52.

Assessments of parents and children were conducted at baseline (T1), at four months (end of depressed parents' treatment; T2), and then every six months: month 10 (T3), month 16 (T4), month 22 (T5).

CHAPTER III

DATA ANALYTIC APPROACH

Latent difference score models. Latent difference score (LDS) modeling (McArdle & Hamagami, 2001) was used to capture change in parents' depressive symptoms and in children's explanatory style over time. At each measurement occasion (t) the variance of the measured dependent variable (DV) (y_t^M) is decomposed into a true score (y_t^T) and a unique (error) score (e_t). Change is modeled in the true scores rather than the measured variables, which is advantageous because the error variance is removed from the measurement of the DV.

In the LDS approach, we estimated latent difference scores (〖Δy〗_l^T) representing the rate of change in the latent true score between adjacent measurement occasions, where l represents a specific time lag between adjacent time points. The latent difference score can be a function of multiple processes. In a dual change score (DCS) model, the latent difference score is a function of a constant latent slope (α×s) and one's true score at the prior measurement occasion (β×y_(t-1)^T), such that 〖Δy〗_l^T= α×s+ β×y_(t-1)^T. Thus, change is constantly related to some unobserved change process and proportionally related to one's prior state. The unconditional DCS model estimates a mean starting value (μ_0), a mean constant rate of change (μ_s), the proportional change coefficient (β), and error variance 〖(e〗_t), which is presumed to be constant over time. Additionally, the models allow for individual differences in starting levels (y_1^T) and in the rate of constant change (s), by estimating parameters σ_0^2 and σ_s^2, respectively, and their covariance (ϱ_(0,s)). Thus, the model estimates seven parameters total.

The DCS model can be simplified in several ways. In the proportional change score (PCS) model, the constant slope mean, variance, and covariance with initial levels are set to 0

(leaving 4 estimated parameters) and the rate of change at each time lag is simply proportional to one's prior state (β). In the constant change score (CCS) model, all three slope parameters are estimated but the proportional change coefficient (β) is fixed at 0 (6 estimated parameters), so that the rate of change is simply a function of the constant slope (s). These simpler models are hierarchically nested within the DCS model allowing for chi-square likelihood ratio tests (χ_LR^2) to directly compare their likelihood functions.

LDS analyses also can accommodate multiple change processes simultaneously, allowing for flexible dynamic associations between two time-varying constructs. In bivariate LDS models, levels or change in one time-varying construct that is modeled using any of the LDS specifications (BCS, PCS, CCS) affects levels of change in another time-varying construct modeled with an LDS specification. These can be unidirectional effects, where one time varying process affects another, or bidirectional effects, where there are reciprocal effects between the two time-varying constructs. For example, in a bivariate DCS model (BDCS), separate DCS models are specified for two time-varying processes. These models are then combined and allowed to influence one another. The rate of change at a specific time-lag for the first construct (〖Δy〗_(l=t-(t-1))^T) could be regressed on levels of the other time-varying process at the prior time point 〖(x〗_(t-1)^T). Simultaneously, the latent rate of change in the second process (〖Δx〗_(l=t-(t-1))^T) could be regressed on levels of the first time-varying construct at the prior time point 〖(y〗_(t-1)^T). Cross-process regressions are called "coupling" parameters and their estimates are expressed using γ coefficients.

Modeling sequence. We first evaluated several competing LDS models (DCS, PCS, and CCS) for parents' depressive symptoms and children's explanatory style, separately. We selected

a final single-process LDS model for each construct using χ_LR^2 tests and the Bayesian Information Criterion (BIC). We also tested whether the assumptions of common error variance 〖(e〗_t) over time and constant proportional change coefficients (β) were tenable, relaxing these assumptions as needed. Then we combined the best LDS models for parental depressive symptoms and children's explanatory style into a BDCS model, allowing reciprocal effects whereby levels of one process at time t-1 influenced the rate of change in the other time-varying process at the subsequent time lag Δ_(t-(t-1)), and vice versa. Constant slopes (s) for both processes were regressed on the intercept (representing baseline levels) of the other process. Covariances between the two intercepts and constant slopes were freely estimated. Finally, covariates representing pre-study characteristics of the parent and child were added as predictors of the latent intercepts and slopes for both processes.

CHAPTER IV

RESULTS

Table 1 reports demographic characteristics of the depressed parents and their children as compared to those of the non-depressed parents and their offspring. At Time 1, depressed parents reported significantly higher levels of depressive symptoms than non-depressed parents. Children of depressed parents also reported significantly higher levels of depressive symptoms on the CDI than children of non-depressed parents; therefore, children's T1 CDI scores were included in the model as a control variable, in addition to children's age, sex, and risk group (i.e., parents' depression history).

Table 2 presents the means, standard deviations, and correlations of the study variables. Correlations of parents' depressive symptoms (BDI) across the five time points ranged from .51 to .77 (all p's < .01). Correlations of children's attributional style (CASQ) across the five time points ranged from .47 to .76, (all p's < .01). Correlations of parents' BDI and children's CASQ across the five time points ranged from -.10 (n.s.) to -.33 (p < .01).

The bivariate latent differences score model provided a close fit to the data: $\chi^2 =$ 1100.115, *df* = 60, p = .00; comparative fit index (CFI) = .987; root mean square error of approximation (RMSEA) = .042, 90% confidence interval (CI) = .000 and .069; standardized RMS residual (SRMR) = .025. Figure 1 presents the overall model. Level of parents' depressive symptoms was negatively associated with changes in children's attributional style across each time lag (p's = .025-.033). With higher parental depressive symptoms, we see greater reductions (worsening) of children's attributional style at the subsequent time lag. The rate of linear change in parent depressive symptoms was associated with linear change in children's attributionals,

p=.33. Higher (better) starting levels of attributional style (intercept) were nearly significantly negatively associated with constant change in parents' depressive symptoms (constant slope). A one-unit increase in the attributional style intercept (starting levels) was associated with a 0.80 decrease in the linear slope (constant change) of parent depressive symptoms, p=.05. That is, a more positive attributional style at baseline was associated with less increase in parents' depressive symptoms over time. This single dynamic model addresses longitudinal and bidirectional change for both parent depression and children's attributions simultaneously.

CHAPTER V

DISCUSSION

Several interesting findings emerged from this study. First, significant associations were found between the level of parents' depressive symptoms and the degree to which children's attributional style worsened over time; higher levels of depression in parents were associated with a worsening of children's attributional style at each time lag. Second, a more positive explanatory style in children at baseline predicted less increase in parents' depressive symptoms over time. Finally, parents who entered the study with lower depressive symptoms were more likely to have children whose attributions either improved or worsened less than parents who entered the study with higher levels of depressive symptoms.

The associations found between level of parents' depressive symptoms and worsening of children's attributional style extends prior evidence from cross-sectional studies that offspring of depressed parents report more negative cognitions as compared to children of non-depressed parents (e.g. Taylor & Ingram, 1999; Garber & Robinson, 1997). The current study showed a prospective relation between parental depression and children's attributions across two years. Thus, children's cognitive vulnerability may persist as long as parents continue to experience depressive symptoms.

The relation between parents' levels of depressive symptoms at baseline and children's attributional style over time may have been due to several factors. In particular, depressed parents and their children likely have a common genetic vulnerability or experienced shared stressful life events. Moreover, parents with more severe and sustained depression expose their children to its negative consequences for longer periods of time. Parents with higher levels of

depression may express more negative cognitions themselves or engage in more negative parenting behaviors that likely affect children's cognitions, such as their explanations about the causes of stressful events (e.g., Goodman & Gotlib, 1999).

Children's explanatory style at the baseline assessment was related to the trajectory of parents' symptoms over time. In particular, a more positive attributional style in children predicted less increase in parents' depressive symptoms across the two years. This finding is congruent with prior research showing a bidirectional relation between children's and parents' psychopathology (e.g., Hammen, Burge, & Adrian, 1991).

Negative reciprocal patterns between depressed mothers and their children have been reported previously (Frye & Garber, 1996; Hammen, Burge, & Stansbury, 1990). In the current study, we found that more negative child attributions predicted higher subsequent levels of parents' depressive symptoms. Children's negative attributional style has been shown to be associated with hostility and fear (Ciarrochi, Heaven, & Davies, 2007), depression (Seligman et al., 1984, Prinstein & Aikins, 2004), and ADHD (Rucklidge & Tannock, 2001), all of which can be challenging and distressing to parents. Thus, children's cognitions, behaviors, and psychopathology might create problems for or evoke distress in parents, which is consistent with a pattern of bidirectional relations.

Limitations, Future Directions, and Clinical Implications

Limitations of the present study highlight directions for future research. First, we cannot rule out the possibility that the observed relations between parents' depression and children's attributions were due to some third (unmeasured) variable such as genes or stress to explain the link between parents' depression and children's cognitions. Efforts to elucidate biological

mechanisms implicated in the transmission of depression, such as genetic risk (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Dick, 2011; Kendler, Aggen, & Neale, 2013) and brain and central nervous system functioning (Disner, Beevers, Haigh, & Beck, 2010), are beginning to be investigated; associations between these potential mechanisms and children's attributional style need to be explored.

Second, the current study focused on children's attributions, which are a central feature of the helplessness and hopelessness theories of depression (Abramson et al., 1978; 1989). Other types of negative cognitions (e.g., self-worth; dysfunctional attitudes), however, have been linked to depression in children (Hankin & Abela, 2008) and should be studied prospectively in relation to changes in parents' depressive symptoms. Third, although the current sample included fathers, the sample size did not allow us to examine possible sex differences in the relations between parental depression and children's attributions.

Finally, it is possible that a different length of time between evaluations might have yielded a different patterns of results. For example, assessments of parents' depression and children's cognitions closer in time might have resulted in even higher correlations between them. Parametric studies are needed to determine the duration between assessment intervals that is most likely to show the strongest associations.

The results of this study have important clinical implications. Research has shown that children's attributional style can be improved with intervention (Gillham, Hamilton, Freres, Patton, & Gallup, 2006). Moreover, evidence also is accumulating that the risk experienced by children of depressed parents can be reduced through preventive interventions (Compas, Forehand, Keller, Champion, Rakow, Reeslund, … & Merchant, 2009; Garber, Clarke,

Weersing, Beardslee, Brent, Gladstone, … & Shamseddeen, 2009). Prevention programs should aim to both reduce parents' depression *and* teach children strategies for dealing with stress, which will reduce the likelihood of their developing or maintaining cognitive vulnerabilities for depression.

In summary, the current study provided further evidence of a significant and prospective relation between parental depression and children's negative cognitions (i.e., attributional style). The findings also were consistent with the view that the relation between children's cognitions and parents' depressive symptoms may be bidirectional. As both parents' depressive symptoms and children's negative attributions can be modified, interventions should aim to both reduce parents' depression and prevent the development of negative cognitions in at-risk children.

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Table 1: Demographic characteristics and parents' baseline depressive symptom scores

CHILDREN	**High-Risk N = 129**	**Low-Risk N = 98**
Age [Mean (*SD*)]	12.38 (2.39)	12.72 (2.22)
Girls [N (%)]	68 (52.7%)	53 (54.1%)
Ethnicity/Race [N (%)]		
White, non-Hispanic	90 (69.8%)	70 (71.4%)
African-American	27 (20.9%)	22 (22.4%)
Asian	1 (.8%)	0 (0%)
Multi-racial	10 (7.8%)	6 (6.1%)
Children's Depression Inventory*	8.10 (6.66)	4.51 (4.32)
Attributional Style Total Score*	.44	.59
PARENTS	**Depressed N = 129**	**Non-depressed N = 98**
Female [N (%)]	94 (72.9%)	78 (79.6%)
Age [Mean (*SD*)]	41.43 (7.21)	43.12 (6.22)
Parent Education [Mean (*SD*)]*	14.66 (2.53)	15.43 (2.61)
BDI-II [Mean (*SD*)]*	25.40 (12.03)	1.76 (2.61)
HRSD [Mean (*SD*)]*	20.09 (6.05)	1.65 (1.92)

* $p < .05$; *SD* = Standard Deviation; BDI-II = Beck Depression Inventory, second edition; HRSD = Hamilton Rating Scale for Depression

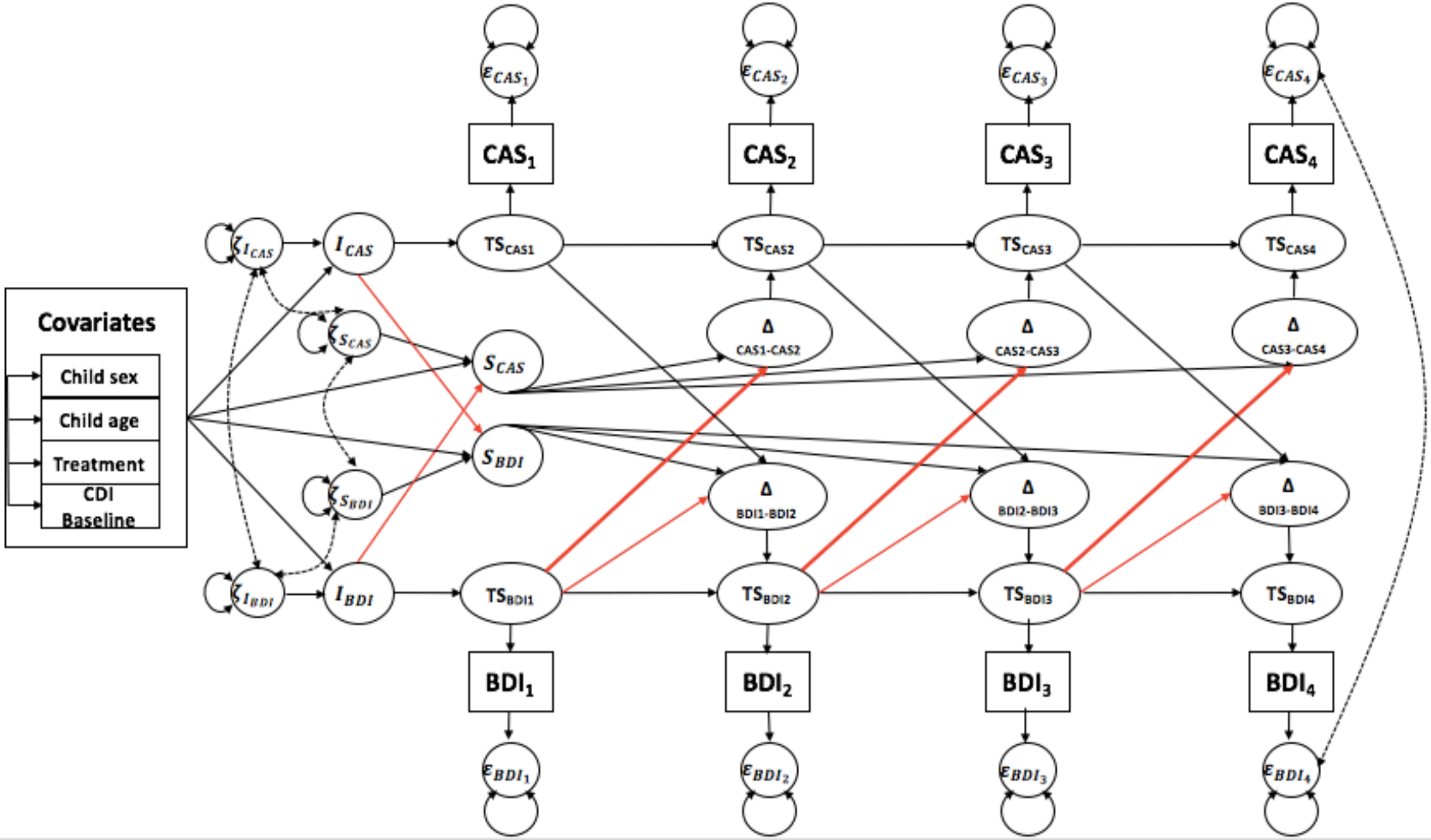
Table 2. Means, Standard Deviations, and Correlations among Study Variables

	M (SD)	Child Age	Child Sex	Parent Sex	T1 BDI	T2 BDI	T3 BDI	T4 BDI	T5 BDI	T1 CASQ	T2 CASQ	T3 CASQ	T4 CASQ	T5 CASQ
Child Age	12.53 (2.33)	--	.04	.21**	-.01	-.12	-.04	.00	-.06	-.08	-.02	-.07	-.01	-.07
Child Sex	--	.04	--	.13	.01	.04	.05	-.04	-.02	-.12	-.04	-.04	-.03	-.01
Parent Sex	--	.21**	.13	--	.04	.03	.01	.07	.15*	-.04	.06	.12	.09	.07
T1 Parent BDI	14.98 (14.90)	-.01	.01	.04	--	.62***	.57***	.52***	.51***	-.15*	-.10	-.10	-.10	-.10
T2 Parent BDI	8.36 (10.15)	-.12	.04	.03	.62***	--	.77***	.65***	.65***	-.14	-.17*	-.24**	-.19**	-.31***
T3 Parent BDI	6.80 (8.49)	-.04	.05	.01	.57***	.77***	--	.73***	.63***	-.17*	-.25***	-.24**	-.21**	-.27***
T4 Parent BDI	6.34 (9.40)	.00	-.04	.07	.52***	.65***	.73***	--	.64***	-.16*	-.16*	-.12	-.16*	-.22**
T5 Parent BDI	7.55 (10.86)	-.06	-.02	.15*	.51***	.65***	.63***	.64***	--	-.13	-.19*	-.11	-.08	-.22**
T1 CASQ	.508 (.28)	-.08	-.12	-.04	-.15*	-.14	-.17*	-.16*	-.13	--	.64***	.60***	.48***	.47***
T2 CASQ	.583 (.28)	-.02	-.04	.06	-.10	-.17*	-.25***	-.16*	-.19*	.64***	--	.66***	.54***	.58***
T3 CASQ	.612 (.26)	-.07	-.04	.12	-.10	-.24**	-.24*	-.12	-.11	.60***	.66***	--	.66***	.60***
T4 CASQ	.621 (.28)	-.01	-.03	.09	-.10	-.19**	-.21**	-.16*	-.08	.48***	.54***	.66***	--	.76***
T5 CASQ	.619 (.29)	-.07	-.01	.07	-.10	-.31***	-.27***	-.22**	-.22**	.47***	.58***	.60***	.76***	--

*p < .05; **p < .01; ***p < .001

T = Time; BDI = Beck Depression Inventory; CASQ = Children's Attributional Style Questionnaire

Figure 1. Latent Difference Score Model



Fit indices: χ^2 (60)= 1100.115, p = .00; RMSEA= .042; 90% CI [.00, .07]; CFI=.99.
Red lines indicate significant pathways at a p=.05 level.