Published in final edited form as:

J Abnorm Child Psychol. 2015 February ; 43(2): 355–368. doi:10.1007/s10802-014-9906-5.

Longitudinal Relations between Stress and Depressive Symptoms in Youth: Coping as a Mediator

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Abstract

The present prospective study examined the relations among stressful life events, coping, and depressive symptoms in children at varied risk for depression. Participants were 227 children between 7 and 17 years old (mean age = 12.13 years, $SD = 2.31$, 54.6% female) who were part of a longitudinal study of depressed and nondepressed parents and their children. Youth completed measures assessing stressful life events and coping strategies at four time points over 22 months. Children's depressive symptoms were assessed at each time point by clinical interviews of parents and children, and children's self-report. Structural equation modeling indicated that stressful life events significantly predicted subsequent depressive symptoms. Bootstrap analyses of the indirect effects in three different models revealed that primary control engagement coping and disengagement coping strategies partially mediated the relation between stressful life events and children's depressive symptoms across time. Regarding the direction of effects, more consistent relations were found for coping as a mediator of the link from stress to depressive symptoms than from symptoms to stress. Thus, one potential mechanism by which stressful life events may contribute to depressive symptoms in children is through less use of primary control coping and greater use of disengagement coping strategies. This is consistent with the view that the adverse effects of stress may contribute to impairments in the ability to cope effectively.

Keywords

stress; depression; coping; children; adolescents

Stressful life events have been shown to be significantly linked with psychopathology in children and adults (Grant et al., 2006; Hammen, 2005). In particular, a positive association between stress and depressive symptoms has been found in both community and clinic

samples of children and adolescents (Barrera et al., 2002; Barrett & Heubeck, 2000; Grant, Compas, Thurm, McMahon, & Gipson, 2004; Rudolph et al., 2000; Wadsworth & Compas, 2002). Compas and colleagues (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001; Compas, Malcarne, & Fondarcaro, 1988) proposed that one mechanism underlying the relation between stress and psychopathology may be how individuals respond and adapt to stress. The purpose of the present longitudinal study was to investigate whether the link between stressful life events and depressive symptoms in children was mediated by specific types of coping.

According to Compas and colleagues (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000), responses to stress can be categorized as either involuntary (i.e., automatic reactions) or voluntary attempts to cope with the stressor or its consequences. Involuntary stress reactions are the body's automatic cognitive, behavioral, and emotional responses that may or may not be under conscious control (e.g. intrusive thoughts or physiological reactivity). *Coping* has been defined as the "conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances" (Compas et al., 2001, p. 89). Voluntary coping responses can be further categorized as either engagement with or disengagement from a stressful event or one's emotional reaction to the event. Confirmatory factor analyses have revealed three voluntary coping factors (Connor-Smith et al., 2000). *Primary control engagement coping* involves attempts to directly change the situation or one's emotional reaction to the situation and includes problem-solving, emotional expression, and emotion regulation. *Secondary control engagement coping* involves efforts to adapt to the situation by regulating attention and cognition, such as enacting acceptance, cognitive restructuring, positive thinking, or distraction. *Disengagement coping* involves withdrawing from the source of stress and one's emotions and includes avoidance, denial, and wishful thinking.

Using this conceptual framework of responses to stress (Compas et al., 2001; Compas, 2006), the present longitudinal study investigated the relations among stressful life events, the three voluntary coping approaches, and depressive symptoms in children. Although evidence exists of significant links between stress and coping, and between coping and psychopathology, most of these findings have come from cross-sectional studies (e.g., Fear et al., 2009; Jaser et al., 2005, 2007; Langrock et al., 2002; Wadsworth & Berger, 2006; Wadsworth, Raviv, Compas, & Connor-Smith, 2005). Few investigations have tested a mediation model in which the relation between stress and depression in children is accounted for by coping, using a prospective design across multiple time points.

Cross-sectional studies have demonstrated that under conditions of high stress, individuals tend to use less effective coping strategies (Jaser et al., 2008; Jaser et al., 2005). Specifically, the use of both primary and secondary control coping was found to decrease as stress increased, whereas disengagement coping strategies (e.g., avoidance; denial) increased (Wadsworth & Compas, 2002). High levels of stress also have been associated with higher levels of arousal, intrusive thoughts and rumination, and less secondary control coping (Langrock, Compas, Keller, & Merchant, 2002). Evidence of a strong inverse relation between involuntary stress reactivity and adaptive coping has been interpreted as indicating

that the ability to use advanced cognitive strategies (e.g., cognitive restructuring), may be compromised when stress levels are elevated (Compas, 2006; Valiente et al., 2004).

A significant relation also has been shown between different types of coping and psychopathology. Whereas primary and secondary control engagement coping are associated with better outcomes, strategies involving disengagement from the stressor are associated with more symptoms and lower competence (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Compas et al., 2001). Specifically, disengagement strategies have been shown to be correlated with anxiety and depression (Wadsworth & Compas, 2002), and active coping and distraction have been found to predict lower levels of internalizing symptoms (Sandler, Tein, & West, 1994).

Cross-sectional studies also have shown that coping mediates the link between various types of stressors and symptoms in children. In a sample of offspring of depressed parents, the relations between parent-child interaction stress and children's symptoms of depression and anxiety, and the links between peer and family stressors and children's internalizing symptoms were found to be partially mediated by less use of secondary control coping (Jaser et al., 2005). In youth exposed to poverty-related stressors, secondary control coping also was shown to mediate the relation between stress and symptoms (Wadsworth et al., 2005). In a study of children experiencing parental divorce, avoidance coping strategies were found to mediate the association between stress and depression (Sandler et al., 1994).

A significant relation among stress, coping, and psychopathology also has been found in some longitudinal studies of children (e.g., Compas et al., 2010; Sandler et al., 1994; Sawyer, Pfieffer & Spence, 2009; Shelton & Harold, 2007). For example, in a sample of inner-city African American youth, maladaptive coping strategies such as avoidance and aggression mediated the relation between exposure to community violence and depressive and anxious symptoms (Dempsey, 2002). Exposure to stressors such as marital conflict (Shelton & Harold, 2007) and the juvenile justice system (Goodkind, Ruffolo, Bybee, & Sarri, 2009) also has been found to predict subsequent depressive and anxious symptoms, and these relations were mediated by the use of maladaptive responses such as withdrawal, emotional venting, and acting out. Finally, Compas and colleagues (2010) reported that increases in children's secondary control coping mediated the effect of a family group cognitive-behavioral preventive intervention on children's depressive symptoms, accounting for approximately half of the significant intervention effect.

Methodological problems of these prior investigations limit the conclusions that can be drawn regarding the extent to which coping mediates the relation between stress and depressive symptoms. First, cross-sectional studies of coping as a mediator can result in misleading results, particularly regarding the direction of effects (Cole & Maxwell, 2003). Second, small sample sizes have limited the ability to use modeling methods that simultaneously control for earlier levels of variables and concurrent associations. Third, because the definitions and measurement of coping have varied quite a bit, comparisons of results across studies have been difficult. Grant and colleagues (2006) called for theoretically-driven, incremental research using comparable constructs and measures to examine specific coping strategies in relation to stress and symptoms. Finally, previous

studies typically have examined one type of coping in response to a specific stressor (e.g. marital conflict; parental depression), but have not explored the three voluntary coping domains within the same sample of youth.

The current prospective study adds to this literature on the relations among stress, coping, and depression in children by addressing several of the limitations of these earlier studies. First, we included a moderate size sample of 227 youth and conducted multiple assessments across 22 months, which allowed us to test the longitudinal direct and indirect effects using autoregressive cross-lag structural equation models, controlling for prior levels of the primary study variables, concurrent correlations, and demographic characteristics (Cole & Maxwell, 2003).

Second, the current study used a theoretically-derived and well-validated conceptualization of coping (Compas, 2006; Connor-Smith et al., 2000). Third, whereas some previous tests of the mediation hypothesis (e.g., Compas et al., 2010) have focused on one type of coping (i.e., secondary control coping), the present study tested mediation models of the three different types of voluntary coping (Compas, 2006): primary control, secondary control, and disengagement.

Finally, children in the current study varied in their risk for psychopathology as a function of parental depression. Offspring of depressed parents are at heightened risk for developing psychopathology as compared to children of non-depressed parents (Beardslee, Gladstone, & O'Connor, 2011; Hammen & Brennan, 2003). Other investigations of offspring of depressed parents have found that secondary control coping partially mediated the relation between the specific stressor of parental depression and children's depressive symptoms (Fear et al., 2009; Jaser et al., 2005; Langrock et al., 2002). Thus, offspring of depressed parents are not only exposed to high levels of adverse life events, but they also tend to lack skills for coping with these stressors. In addition to investigating secondary control coping, the current study of children of depressed and nondepressed parents examined whether primary control and disengagement coping also mediated the relation between stressful life events and depression in youth.

In summary, this study tested the following hypotheses: (a) There will be a positive, prospective relation between stressful life events and depressive symptoms; we also explored whether the reverse direction was true; that is, whether depressive symptoms predicted stress. (b) Higher levels of stressful life events will predict significantly less use of primary and secondary control coping, and greater use of disengagement coping. (c) High levels of primary and secondary control coping and low levels of disengagement coping will predict lower levels of depressive symptoms. (d) The relation between stressful events and depressive symptoms will be partially mediated by coping (i.e., primary control, secondary control, and disengagement coping).

Method

Participants

Participants were 227 parents and children (one parent and one child per family). In 129 families (high risk), a parent had a current Major Depressive Disorder (MDD) as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; American Psychiatric Association, 1994). Exclusion criteria for these parents were a lifetime history of psychosis, organic brain syndrome, or bipolar disorder, or a current or primary diagnosis of substance abuse or dependence, obsessive-compulsive disorder, eating disorder, and certain personality disorders (antisocial, borderline, schizotypal). The comparison group included 98 families (low risk) with parents who were lifetime-free of mood disorders, psychosis, organic brain syndromes, or personality disorders, and during the child's life were free of anxiety disorders or substance abuse/dependence, had not had psychotherapy for longer than two months or eight sessions, and had not been prescribed or used psychotropic medication.

Child participants were 7 to 17 years old (M age = 12.13 years, SD = 2.31). Exclusion criteria were a developmental disability or a significant chronic medical condition. For low-risk families, the enrolled child was selected to be similar in age and sex to a high-risk child. The overall child sample was 54.6% female, 69.6% Caucasian, 21.6% African-American, 1% Asian, and 6.9% multi-racial. High- and low-risk children did not differ significantly in children's age, sex, ethnicity, race, or parents' age or sex; the groups differed significantly on parent education, which therefore was controlled in all analyses (see Garber, Ciesla, McCauley, Diamond, & Schloedt, 2011 for more details about the sample).

Procedure

Depressed parents were recruited from psychiatric clinics where they were receiving treatment for depression. Recruitment of comparison families involved print and radio advertisements, and coordination with local schools, health maintenance organizations, and community agencies. Comparison parents were initially screened over the phone, and if eligible, then were scheduled for a full evaluation to further assess inclusion and exclusion criteria.

Child assessments were conducted by different individuals than those doing the parent evaluations. Participants were part of a longitudinal study of depressed and nondepressed parents and their children (Garber et al., 2011). The present analyses included the four time points when all of the constructs of interest (i.e., stressful life events, coping, and depressive symptoms) were assessed -- baseline (T1), and 4 months (T2), 16 months (T3), and 22 months (T4) post baseline. Parent informed consent and child assent were obtained from all participants before data collection. All procedures were approved by the Institutional Review Boards for the Protection of Human Subjects in research.

Measures

The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 1997) was used to evaluate parents' psychopathology. A randomly selected subset (N = 23, 10%)

of taped interviews was used to assess inter-rater reliability, yielding kappa coefficients = .80 for diagnoses of MDD.

Children's Measures—The *Life Events Questionnaire* (LEQ; Johnson, & McCutcheon, 1980) measures children's report of stressful life events (SLEs) within a given time frame. The LEQ includes 50 SLEs such as: *A family member or close relative died. You got into serious trouble at school. There were more arguments/fights between your parents.* Scores can range from 0 to 50, with higher scores indicating a greater number of stressful events during the time period. Children reported about events that had occurred since the previous assessment. For example, stressful life events reported at T2 occurred during the interval between T1 (baseline) and T2 (4 months post baseline). The negative life events items on the LEQ have been found to have good validity (Greene, Walker, Hickson, & Thompson, 1985) and reliability (Brand & Johnson, 1982). Test-retest reliability over a two-week period for the LEQ was found to be 0.72 (Brand & Johnson, 1982).

The *COPE Inventory* (Carver, Scheier, & Weintraub, 1989) is a multidimensional measure of coping; individuals rate each item on a four-point Likert scale (1 = *Usually do not do this at all*; 4 = *Usually do this a lot*) indicating the extent to which they use each coping strategy. Internal consistency, test-retest reliability, and convergent and discriminant validity have been well documented for the *COPE* (Carver et al., 1989). The 60 items of the *COPE* can be divided into 15 subscales measuring conceptually and empirically distinct aspects of coping. These subscales have been shown to correlate with the factors of primary control, secondary control, and disengagement coping as defined and measured by Connor-Smith and colleagues (2000).

We created coping composites using subscales from the *COPE* that were equivalent to the three voluntary coping factors: primary control, secondary control, and disengagement coping described by Connor-Smith et al. (2000). The *COPE* subscales comprising the *primary control coping* composite are Planning, Active Coping (e.g., take action to change a problem: "I take additional action to try to get rid of the problem"), Seeking Social Support for Instrumental Reasons, and Seeking Social Support for Emotional Reasons. The *secondary control coping* composite includes Acceptance, Positive Reinterpretation and Growth subscales (e.g., changing negative thoughts about a stressor: "I try to see it in a different light, to make it seem more positive"). The *disengagement* composite includes Behavioral Disengagement, Denial, and Mental Disengagement subscales (e.g., engaging in activities to avoid thinking about a stressor: "I go to the movies or watch TV to think about it less"). As per Connor-Smith et al. (2000), we computed the coping factor scores as proportions of the total score for all responses (e.g., sum of scores on primary control items/sum of all items) to control for overall responding bias. Internal consistency across all time points was as follows: primary control coping, $\alpha = .88$; secondary control coping, $\alpha = .86$; disengagement coping, $\alpha = .72$).

The *Children's Depression Inventory* (CDI; Kovacs, 1992) is a self-report measure of depressive symptoms in children. Each of the 27 items lists three statements of increasing severity. Total scores can range from 0 to 54, with higher scores indicating more depression. Internal consistency, test-retest reliability, and convergent validity have been well

documented for the CDI (Kovacs, 1992). Coefficient alpha for the CDI in this sample was .84 across all time points.

Depressive symptoms also were assessed at each time point with the *Children's Depression Rating Scale-Revised* (CDRS-R; Poznanski, Mokros, Grossman, & Freeman, 1985). Children and parents were interviewed with the CDRS-R regarding the extent of the children's depressive symptoms during the previous 2 weeks. Summary scores based on parent and child reports were used in the analyses. Nine depressive symptoms (e.g., anhedonia, sadness, suicidal ideation) were rated on a 7-point severity scale (1 = *symptom not present*, 7 = *symptom severe*); total scores could range from 9 to 63. Coefficient alpha for the CDRS-R across times points was α .79. The CDI and the CDRS-R were significantly correlated at each time point (r s ranged from .44 to .59). A depression composite score was created by standardizing and then averaging the CDI and CDRS-R scores. Internal consistency for the depression composite was α .88 across all time points.

Data Analysis Plan

Structural equation modeling (SEM) using AMOS 17.0 (Arbuckle, 2008) was used to test for longitudinal cross-lagged effects. This method was chosen because of its ability to test models with multiple dependent variables, model mediating variables, and model error terms. We used full information maximum likelihood (FIML) estimation, which handles missing data.

Separate autoregressive cross-lag SEMs were fit for each type of coping. The models tested whether stressful life events directly predicted depressive symptoms and indirectly predicted depressive symptoms through the hypothesized mediators -- primary control coping, secondary control coping, or disengagement coping. As recommended by Cole and Maxwell (2003) for testing longitudinal mediation, all relevant autoregressive paths were included to reduce potential bias and provide a stringent test of longitudinal direct and indirect effects in each model. Detailed results of the autoregressive and direct paths between stress and depressive symptoms in each SEM model are available upon request. All models controlled for concurrent correlations among variables; additionally, risk (i.e., parental depression) was entered into the model and controlled at each time point. Child age and sex were included as covariates; to be conservative, paths to the study variables with which age or sex were correlated at $p < .10$ were included in the model.

Model fit was assessed using the following indices: the relative χ^2 index (χ^2/df), where scores below 2 suggest a good fit between the model and sample data (Bollen, 1989); the comparative fit index (CFI; Bentler, 1990) with scores above .90 indicating acceptable fit and above .95 indicating good model fit; and the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993) in which .06 or less is considered a good model fit.

The significance of the direct effects between stressful life events (SLEs) and depression was determined by comparing the t ratio of the unstandardized path coefficients with a critical $t(0.05)$ of 1.96. The significance of the indirect effect among SLEs, coping, and depressive symptoms was calculated using a bias-corrected bootstrap procedure, in which an original data set is used to create a large number of randomly drawn additional data sets of

the same size. Over many bootstrap re-samples, typically between 500–5000, an empirical approximation of the sampling distribution of the statistic can be generated and used for hypothesis testing. The bootstrap procedure conducted in the present analyses resulted in a 95% bias-corrected confidence interval of the indirect effect of stressful life events on depressive symptoms.

The structural equation models were conducted without imputation of missing data. The bootstrap analyses, however, do not allow for missing values within a data set; therefore imputation for the bootstrap procedure was conducted using the estimation procedures provided by Amelia II: A Program for Missing Data (Honaker & King, 2010). These procedures resulted in five multiply imputed data sets; bootstrap analyses were run on each. An overall confidence interval of the indirect effect was obtained by averaging the results across these bootstrap analyses. The path coefficients and significance tests of the direct effects within each model were nearly identical to those presented below.

Results

Descriptive Statistics

Table 1 presents the means, standard deviations, and correlations among the study variables at each of the four time points. In general, the bivariate correlations of the variables in the hypothesized mediation models were significant. Child age and sex were included as covariates when they were correlated ($p < .10$) with variables in the model. Regarding the paths included, females reported significantly more stressful life events (SLEs) than males occurring between T2 and T3 ($r = -.17, p = .02$) and between T3 and T4 ($r = -.22, p = .002$); the correlation between child sex and stressful life events (SLEs) occurring between T1 and T2 was marginal ($r = -.135, p = .051$). Older children had significantly higher levels of depressive symptoms at T2 ($r = -.14, p = .03$) and T3 ($r = .14, p = .046$). Correlations between age and coping were nonsignificant; a few marginal associations were found in the direction of older children reporting higher levels of secondary control coping at T1 and primary control coping at T4, and less use of disengagement coping at T3 and T4 (see Table 1). These correlations are noted here only to indicate for which analyses age or sex was included as a control variable and are not intended to be interpreted substantively.

Primary Control Coping

We first tested the model that included stressful life events (SLEs), primary control coping, and depressive symptoms measured at all four time points. The model included direct paths between SLEs and depressive symptoms, indirect paths to and from the hypothesized mediator (i.e., primary control coping), and autoregressive and reciprocal paths among all variables. This model provided a good fit to the sample data, $\chi^2 (29, N = 227) = 41.86, \chi^2/df = 1.44, CFI = .99, RMSEA = .04$.

Direct effects—Inspection of the unstandardized path coefficients between stressful life events and depressive symptoms indicated that SLEs occurring between T1 and T2 significantly predicted higher levels of depressive symptoms at T2, $b = .05, SE = .01, p < .$

01. Stressful life events occurring between T2 and T3 significantly predicted higher levels of depressive symptoms at T3, $b = .04$, $SE = .01$, $p = .01$.

Analyses of the reverse direction (i.e., depressive symptoms to subsequent SLEs) revealed that higher levels of depressive symptoms at T1 significantly predicted more SLEs between T2 and T3, $b = 1.24$, $SE = .28$, $p < .01$, and symptoms at T2 significantly predicted SLEs between T3 and T4, $b = .72$, $SE = .25$, $p < .01$. Thus, the direct effect between children's depressive symptoms and stressful events was positive, such that prior SLEs predicted subsequent increases in depressive symptoms, and higher levels of depressive symptoms predicted increases in SLEs.

Indirect Effects—Analyses of the cross-lagged paths between stress and primary control coping indicated that higher levels of SLEs between T2 and T3 significantly predicted less use of primary control coping at T3, $b = -.002$, $SE = .001$, $p = .03$; the relation of SLEs between T3 and T4 to primary control coping at T4 was not significant. Primary control coping at T2 significantly predicted more SLEs between T3 and T4, $b = 10.54$, $SE = 3.75$, $p < .01$.

Examination of the paths between primary control coping and depressive symptoms indicated that the relation between higher levels of depressive symptoms at T1 and less use of primary control coping at T2 was as follows: $b = -.008$, $SE = .004$, $p = .06$. Greater use of primary control coping at T3 significantly predicted lower levels of depressive symptoms at T4, $b = -1.77$, $SE = .83$, $p = .03$, and higher levels of depressive symptoms at T3 significantly predicted less use of primary control coping at T4, $b = -.01$, $SE = .005$, $p = .03$.

Confidence intervals of the indirect effects of stressful life events on depressive symptoms (95% CI: 0.012 – 0.063) based on 500 bootstrap samples did not include zero. An indirect relation between SLEs at T3 and depressive symptoms at T4 was found indicating that the relation between SLEs and depressive symptoms was mediated by primary control coping. That is, stressful events predicted lower levels of primary control coping, which in turn predicted higher levels of depressive symptoms (Figure 1).

Secondary Control Coping

The next model included direct paths between stressful life events and depressive symptoms, indirect paths to and from the hypothesized mediator (i.e., secondary control coping), and autoregressive and reciprocal paths among all variables. The model provided a good fit to the data, $\chi^2 (29, N = 227) = 43.34$, $\chi^2/df = 1.49$, CFI = .99, RMSEA = .05.

Direct effects—Direct effects between stressful life events and depressive symptoms were similar to those in the model for primary control coping; SLEs between T1 and T2 significantly predicted higher levels of depressive symptoms at T2, $b = .05$, $SE = .01$, $p < .01$. SLEs between T2 and T3 significantly predicted T3 depressive symptoms, $b = .04$, $SE = .01$, $p = .01$, and SLEs between T3 and T4 significantly predicted T4 depressive symptoms, $b = .03$, $SE = .02$, $p = .04$. Inspection of the paths from depressive symptoms to stressful life events revealed that higher levels of depressive symptoms at T1 significantly predicted a greater number of SLEs between T2 and T3, $b = 1.28$, $SE = .27$, $p < .01$, and

higher levels of depressive symptoms at T2 significantly predicted more SLEs between T3 and T4, $b = .55$, $SE = .26$, $p = .04$.

Indirect effects—The cross-lagged paths among stressful life events, secondary control coping, and depressive symptoms revealed a significant negative path coefficient between T1 depressive symptoms and T2 secondary control coping, $b = -.008$, $SE = .003$, $p = .01$. Additionally, greater use of secondary control coping at T1 significantly predicted lower levels of depressive symptoms at T2, $b = -2.90$, $SE = 1.27$, $p = .02$, and greater use of secondary control coping at T2 significantly predicted lower levels of depressive symptoms at T3, $b = -2.95$, $SE = 1.16$, $p = .01$. A significant indirect effect between SLEs and depressive symptoms through secondary control coping was not found; therefore bootstrap analyses were not conducted (see Figure 2).

Disengagement Coping

The third model included direct paths between stressful life events and depressive symptoms, indirect paths to and from the mediator (i.e., disengagement coping), and autoregressive and reciprocal paths among all variables. The model provided an acceptable fit to the data, $\chi^2 (28, N = 227) = 46.76$, $\chi^2/df = 1.67$, CFI = .99, RMSEA = .05.

Direct effects—The pattern of direct effects between stressful life events and depressive symptoms was similar to that observed in the primary and secondary control coping models. SLEs occurring between T1 and T2 significantly predicted higher levels of depressive symptoms at T2, $b = .05$, $SE = .01$, $p < .01$, and SLEs between T2 and T3 significantly predicted level of depressive symptoms at T3, $b = .04$, $SE = .01$, $p = .01$. Additionally, the relation of SLEs between T3 and T4 level of depressive symptoms at T4 was as follows: $b = .03$, $SE = .02$, $p = .06$.

Examining the reverse direction of effects indicated that depressive symptoms at T1 predicted a greater number of stressful life events occurring between T2 and T3, $b = 1.22$, $SE = .28$, $p < .01$, and depressive symptoms at T2 significantly predicted more SLEs between T3 and T4, $b = .79$, $SE = .27$, $p < .01$. Thus, there was a positive, reciprocal relation between stressful events and depressive symptoms, such that prior SLEs predicted increases in depressive symptoms, and higher levels of depressive symptoms predicted subsequent increases in SLEs.

Indirect effects—Inspection of the cross-lagged paths between stressful life events and disengagement coping indicated that SLEs occurring between T2 and T3 significantly predicted greater use of disengagement coping at T3, $b = .003$, $SE = .001$, $p < .01$; the relation between stressful events between T3 and T4 and disengagement coping at T4 was as follows: $b = .002$, $SE = .001$, $p = .07$. Greater use of disengagement coping at T2 significantly predicted decreases in SLEs at T4, $b = -7.56$, $SE = 3.74$, $p = .04$.

Regarding the paths between disengagement coping and depressive symptoms, greater use of disengagement coping at T1 significantly predicted higher levels of depressive symptoms at T2, $b = 2.28$, $SE = .80$, $p < .01$; a positive path coefficient also was found from T3 disengagement coping to depressive symptoms at T4, $b = 1.59$, $SE = .81$, $p = .05$. In the

reverse direction, T1 depressive symptoms significantly predicted greater use of disengagement at T2, $b = .02$, $SE = .004$, $p < .01$. Higher levels of depressive symptoms at T2 significantly predicted less use of disengagement at T3, $b = -.01$, $SE = .004$, $p = .01$. Finally, the path from T3 depressive symptoms to T4 disengagement was significant and positive, $b = .01$, $SE = .01$, $p = .02$.

Confidence intervals of the indirect effects of stressful life events on depressive symptoms (95% CI: 0.013 – 0.063) based on 500 bootstrap samples did not include zero. The significant indirect effects indicate that stressful life events occurring between T2 and T3 predicted higher levels of disengagement coping at T3, which in turn predicted higher levels of depressive symptoms at T4. Thus, the relation between stressful life events and depressive symptoms was partially mediated by disengagement coping. Additionally, the confidence interval for the indirect effects of depressive symptoms on stressful life events (95% CI: 0.371 – 1.601) based on 500 bootstrap samples also did not include zero. The significant indirect effects indicated that higher levels of T1 depressive symptoms predicted greater use of disengagement coping at T2, which in turn predicted decreases in stressful life events occurring between T3 and T4 (see Figure 3).

Risk as a Moderator

In a series of post-hoc multi-group analyses, we tested whether the significant indirect paths in the mediation models among stressful life events, coping, and children's depressive symptoms found for primary control and disengagement coping differed as a function of risk (i.e., parental history of depression). First, a model that allowed all regression paths to be freely estimated between the two risk groups (unconstrained model) was compared to a model in which these paths were constrained to be equal between the two groups. If the χ^2 difference test between these two models was significant, we tested the two indirect paths in the model to examine which relations were significantly moderated by risk. For both primary control and disengagement coping, the unconstrained model provided a significantly better fit than the constrained model [Primary Control: $\chi^2(38) = 70.80$, $p = .001$; Disengagement: $\chi^2(39) = 72.30$, $p = .001$]. SLEs occurring between T2 to T3 significantly predicted T3 primary control coping for children at low, but not high, risk, $\chi^2(1) = 5.34$, $p = .02$. Similarly, T2 to T3 SLEs predicted T3 disengagement for the low risk group, $\chi^2(1) = 7.01$, $p = .008$. Relations between either primary control or disengagement coping at T3 and depressive symptoms at T4, however, did not differ by risk. Thus, the relations between stressful life events and primary control or disengagement coping differed by risk, whereas the link between coping and depression did not differ by risk.

Discussion

Several important findings emerged from this longitudinal study of the relations among stressful life events, coping, and depressive symptoms in children who varied in risk for depression. First, stressful events significantly predicted children's depressive symptoms over time. Second, primary control engagement coping and disengagement coping each partially mediated the relation between stressful life events and subsequent levels of children's depressive symptoms. These findings are consistent with Compas' (2006) model

suggesting that the adverse effects of stress likely contribute to impairments in coping, and such maladaptive coping strategies (e.g., less use of primary control coping, and greater use of disengagement) then increase internalizing problems (Compas et al., 2001).

We first investigated the simple bivariate correlations among the components of the hypothesized mediation model separately. Stressful life events correlated positively with depressive symptoms, positively with disengagement coping, and negatively with primary control and secondary control coping, both concurrently and prospectively. These associations are similar to earlier reports showing that children who experience higher levels of stress report lower levels of complex coping strategies such as problem-solving and cognitive restructuring (e.g., Valiente et al., 2004).

Next, primary and secondary control coping correlated negatively with depressive symptoms, which parallels previous evidence that greater use of primary and secondary control coping is associated with lower levels of internalizing symptoms (e.g., Compas et al., 2001). Finally, similar to results reported by Compas and colleagues (e.g., Compas et al., 2001; Wadsworth & Compas, 2002; although see Jaser et al., 2007), we found positive concurrent and prospective associations between disengagement coping and depressive symptoms. Thus, the bivariate correlations among the variables in the hypothesized mediation model were significant.

We then tested the relations among these variables in the mediation models. As expected (e.g., Grant et al., 2004; Grant et al., 2006), analyses of the *direct* effects indicated that stressful life events predicted subsequent depressive symptoms. Analyses of the *indirect* effects revealed that primary control and disengagement coping strategies partially mediated the relation between stressful events and depressive symptoms. Thus, coping may be one salient pathway through which stressful life events affect children's responses to stress (Compas, 2006).

Finally, we conducted a series of post-hoc multi-group analyses to examine whether the significant indirect effects from stressful life events to children's depressive symptoms through coping found in the primary control and disengagement models differed as a function of risk (i.e., parental depression history). Results indicated that in both models, stressful life events predicted decreases in primary control coping and increases in disengagement coping in low, but not high, risk youth, but the paths between coping and depressive symptoms did not differ by risk. Thus, although some components of the models varied as a function of parental depression (i.e., risk), the overall models testing coping as a mediator of the relation between stress and depression were not significantly different by risk.

The present study builds on prior research in several ways. First, most previous investigations of the Compas (2006) model have tested mediation with cross-sectional data, which potentially can result in inaccurate conclusions (see Cole & Maxwell, 2003). The present prospective study utilized structural equation modeling that included autoregressive paths among stress, coping, and depressive symptoms to assess these relations across four time points over 22 months. Our findings were consistent with one previous study showing

that maladaptive coping (e.g., avoidance) mediated the concurrent relation between stressors associated with parental divorce and depressive symptoms in children (Sandler et al., 1994). The current study was the first longitudinal test of whether the three different types of voluntary coping strategies mediated the stress-depression link in a sample of children at varied risk for depression.

Second, prior studies of offspring of depressed parents have assessed children's coping with the specific stressor of having a depressed parent and found that secondary control coping mediated the relation between that particular stressor and children's depressive symptoms (e.g., Fear et al., 2009; Jaser et al., 2005, 2007). The present sample also included offspring of depressed parents as well as children of nondepressed parents. Interestingly, we found a significant mediation effect for primary control and disengagement coping, but not for secondary control coping. One difference between our study and those of Compas and colleagues is that we assessed a broader range of stressful life events than just parental depression. Thus, whereas secondary control coping may be adaptive when dealing with a specific uncontrollable stressor such as parental depression, greater use of primary control coping (e.g., problem-solving) and less use of disengagement coping (e.g., avoidance) may be more adaptive when dealing with a broader array of life stressors, at least some of which are controllable.

Finally, regarding the direction of the relations among stress, coping, and depression, we found significant paths from depressive symptoms to coping in all three models, although the strength and pattern of these effects were not consistent. For example, a significant prospective, lagged effect was found from depressive symptoms to disengagement coping to stressful life events. The path between disengagement coping and subsequent stressful life events was negative, however, suggesting that disengagement may have served to reduce exposure to later stressors, possibly through withdrawing from the world and others. Overall, more evidence was found for coping as a mediator of the prospective relation from stressful life events to depressive symptoms, rather than the reverse. Nevertheless, a connection between depressive symptoms and subsequent stress is consistent with the stress generation hypothesis (Hammen, 1991), and highlights the importance of examining possible bidirectional relations among these constructs over time.

Strengths of this study included the use of autoregressive cross-lag structural equation modeling to test theoretically-derived mediation models across four time points over 22 months, a moderate sample size, and careful diagnoses of parents' depression. Limitations of this study also should be noted as they provide directions for future research. First, the measures of stressful life events and coping were based on children's reports, and therefore their correlations might have been inflated due to shared method variance. The measure of depressive symptoms, however, was comprised of clinician's ratings based on an interview with the parent and child, in combination with children's self-report, and therefore the relation of stress and coping to depressive symptoms was less likely due to common informant effects only. Future studies should include multiple sources (e.g., parents, teachers, peers) and multiple methods of assessing all three constructs of interest, with the caveat that different informants and methods may provide distinct, although equally valid perspective that do not necessarily agree (De Los Reyes, 2013).

Second, using a dispositional measure of coping (Carver et al., 1989) had both advantages and disadvantages. On the one hand, asking children how they cope in general allowed for the examination of their strategies in response to more than a single type of adverse event. On the other hand, the efficacy of some coping responses may be context-specific; therefore, asking children how they cope in general may not capture the specific interplay among particular stressors, coping, and depressive symptoms (Compas, Connor, Harding, Saltzman, & Wadsworth, 1999; Jaser et al., 2007; McMahon, Grant, Compas, Thurm, & Ey, 2003). Perhaps one useful approach to studying coping would be to assess children's responses to multiple, specific stressors. Another limitation was that we were not able to examine involuntary stress responses because the COPE has a relatively small number of items capturing this type of response to stress.

Third, in the current study the time intervals between assessments differed, ranging from 4 to 12 months. More recent stressors tend to exert a greater impact on mental health outcomes than more distal ones (Compas, 2004; Compas, Howell, Phares, Williams, & Ledoux, 1989). As an independent variable becomes more distal, the influence of random factors becomes increasingly possible (Shrout & Bolger, 2002). Indeed, the observed relations might have been even stronger if shorter and more evenly spaced time intervals had been used. The frequency of assessments, however, must be balanced with the burden placed on participants. Although more frequent and more closely spaced assessments might be desirable, they are not always practical and might increase attrition. Future prospective studies should include more frequent data collection waves and identify the temporal spacing that best captures the timing among the constructs of interest. Nevertheless, the observed associations among stress, coping, and depressive symptoms over both the shorter and longer intervals used in the current study suggest that these findings might be robust even when there is variability in the assessment time intervals.

Fourth, the study included children ages 7 to 17 years old, which is a period when considerable change occurs in children's executive functioning and capacity to implement various coping strategies. That is, children's coping tends to shift developmentally, transitioning from more behavioral strategies to those requiring cognition and meta-cognition (e.g. primary and secondary control coping) during adolescence and young adulthood (Zimmer-Gembeck & Skinner, 2011). In the present study, the correlations between age and coping were mostly nonsignificant or marginal in the direction of older children reporting greater use of primary and secondary coping strategies and less use of disengagement. Given that the majority of the children in the sample were about 12 years old, it is possible that the correlations between age and coping were not large or significant due to a restriction of range (i.e., most children were between 10 and 14 years old). In addition, the use of a dispositional measure of coping did not allow us to explore possible age-related changes in children's use of different coping strategies in response to specific stressors (Zimmer-Gembeck & Skinner, 2011). Thus, the absence of a relation between age and coping in the present study should be interpreted with caution.

Finally, using a sample that varied in risk for depression also had both benefits and limitations. Including both high and low risk youth results in a larger range of scores on the measures of interest (i.e., stressful life events, coping, depressive symptoms). Second, the

use of an at-risk sample allowed us to directly compare our results to other studies of coping that have been conducted in samples of offspring of depressed parents. A possible limitation of using a high-risk sample, however, is that the findings might not generalize to a purely normative sample. Additionally, parents comprising the low risk control group were lifetime-free of most forms of psychopathology and had not received extensive psychotherapy or taken medication for a psychiatric disorder. As such, the low risk parents may have been a “supernormal” sample and less representative of the larger population of all adults in the U.S., almost half of whom meet criteria for a mental disorder at some point in their lifetime (Kessler et al., 2005).

In conclusion, the present prospective study found evidence consistent with a mediation model in which the experience of stressful life events predicted the coping strategies used, which then predicted increases in depressive symptoms. Prolonged stress has a significant adverse effect on the brain, specifically those regions responsible for higher order executive functions that regulate successful adaptation to stress (McEwen, 1998; 2005). The current study showed that stressful life events predicted children’s depressive symptoms, in part, through their use of certain coping strategies, over and above the contribution of existing symptom levels. These findings can inform the development of targeted interventions aimed at decreasing maladaptive coping such as disengagement, and increasing strategies (e.g., primary control coping) that predict greater well-being in children experiencing high levels of stress.

Future studies also should explore the developmental trajectories of coping, especially in the context of specific stressors. Such research is needed to better understand the normative development of coping skills as well as how the emergence of various coping profiles affects the long-term consequences of different stressors. Identifying when developmental shifts in coping occur and how those differ for children experiencing different types of stress (e.g., acute vs. chronic; controllable vs. uncontrollable) could help researchers and clinicians target youth who are at greatest risk for negative outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported in part by grants from the National Institutes of Health (R01MH57822, R01MH57834, R01MH057977; T32 MH018921; and UL1 RR024975/TR000445). These funding agencies had no further role in the study design, data collection, analysis or interpretation of data, or writing of this report. The authors are grateful to all of the children and parents who participated in this study. We also would like to acknowledge Steven Hollon, Robert DeRubeis, Richard Shelton, Jay Amsterdam, Sona Dimidjian, and Bruce Compas for their assistance with this work.

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Figure 1. Primary control coping mediation model

Direct paths between Depressive Symptoms and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but are not depicted. Covariates included age, sex, and risk, which were included as direct paths to all variables in the model. Unstandardized parameter estimates (with standard errors in parentheses) are presented. Significant paths (* $p < .05$; ** $p < .01$) are depicted with a bold line; marginal effects ([†] $p < .10$) are depicted with a dotted line, and nonsignificant paths are depicted with a gray line.

Figure 2. Secondary control coping mediation model

Direct paths between Depressive Symptoms and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but are not depicted. Covariates included age, sex, and risk status, which were included as direct paths to all variables in the model. Unstandardized parameter estimates (with standard errors in parentheses) are presented. Significant paths ($*p < .05$; $**p < .01$) are depicted with a bold line; nonsignificant paths are depicted with a gray line.

Figure 3. Disengagement control coping mediation model

Direct paths between Depressive Symptoms and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but are not depicted. Covariates included age, sex, and risk status, which were included as direct paths to all variables in the model. Unstandardized parameter estimates (with standard errors in parentheses) are presented. Significant paths ($*p < .05$; $**p < .01$) are depicted with a bold line; marginal effects ($^{\dagger} p < .10$) are depicted with a dotted line, and nonsignificant paths are depicted with a gray line.

Table 1

Means, Standard Deviations and Bivariate Correlations among Study Variables

	M	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.
1. Risk	.57	.50	--																				
2. T1 CDI	6.56	6.03	.30**	--																			
3. T1 LEQ	8.32	6.28	.28**	.44**	--																		
4. T1 Primary Control Coping	.51	.05	-.12	-.38**	-.10	--																	
5. T1 Secondary Control Coping	.26	.04	-.13	-.21**	-.19**	-.10	--																
6. T1 Disengagement Coping	.23	.06	.18*	.45**	.20**	-.80**	-.53**	--															
7. T2 CDI	4.59	6.1	.18**	.65**	.37**	-.32**	-.23**	.42**	--														
8. T2 LEQ	4.22	4.8	.23**	.36**	.46**	-.09	-.14	.17*	.55**	--													
9. T2 Primary Control Coping	.51	.05	-.06	-.27**	-.06	.46**	-.03	-.37**	-.23**	-.05	--												
10. T2 Secondary Control Coping	.26	.04	-.12	-.33**	-.18*	.04	.55**	-.37**	-.28**	-.18*	-.25**	--											
11. T2 Disengagement Coping	.23	.06	.13	.46**	.17*	-.45**	-.34**	.60**	.40**	.16*	-.76**	-.43**	--										
12. T3 CDI	4.34	5.57	.24**	.56**	.45**	-.26**	-.12	.29**	.51**	.49**	-.15*	-.28**	.34**	--									
13. T3 LEQ	3.8	3.93	.32**	.47**	.56**	-.18*	-.15*	.25**	.50**	.59**	-.17*	-.16*	.27**	.50**	--								
14. T3 Primary Control Coping	.52	.05	-.14*	-.21**	-.15*	.35**	.14	-.38**	-.16*	-.11	.42**	.08	-.44**	-.30**	-.23**	--							
15. T3 Secondary Control Coping	.26	.04	-.08	-.10	-.16*	-.02	.37**	-.21**	-.12	-.17*	-.01	.52**	-.35**	-.23**	-.13	-.24**	--						
16. T3 Disengagement Coping	.21	.06	.12**	.26**	.24**	-.32**	-.38**	.50**	.23**	.22**	-.38**	-.42**	.64**	.43**	.30**	-.77**	-.44**	--					
17. T4 CDI	4.21	5.42	.29**	.56**	.29**	-.25**	-.13	.29**	.50**	.42**	-.23**	-.30**	.40**	.61**	.38**	-.32**	-.17*	.41**	--				
18. T4 LEQ	3.87	3.8	.32**	.37**	.54**	-.06	-.08	.10	.44**	.50**	-.05	-.17*	.15*	.44**	.70**	-.20**	-.13	.27**	.39**	--			
19. T4 Primary Control Coping	.51	.06	-.12	-.25**	-.10	.37**	.22**	-.45**	-.28**	-.05	.34**	.20*	-.43**	-.36**	-.29**	.64**	-.04	-.56**	-.40**	-.24**	--		
20. T4 Secondary Control Coping	.27	.04	-.06	-.21**	-.15	-.05	.29**	-.14	-.17*	-.16*	.08	.43**	-.36**	-.23**	-.10	-.04	.57**	-.34**	-.24**	-.14	-.21**	--	
21. T4 Disengagement Coping	.22	.06	.15	.36**	.18*	-.31**	-.38**	.50**	.37**	.16*	-.36**	-.46**	.63**	.47**	.33**	-.57**	-.32**	.74**	.52**	.31**	-.80**	-.42**	--

Note. SD = Standard Deviation; T = Time point; LEQ = Life Events Questionnaire; CDI = Children's Depression Inventory

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