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ORIGINAL PAPER

Mother and Child Depressive Symptoms in Youth with Spina Bifida: Additive, Moderator, and Mediator Models

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Abstract The purpose of the study was to examine the extent to which parenting behaviors influence the relation between maternal and child depressive symptoms in youth with spina bifida and a comparison sample. Previous research has found that maternal depression not only negatively impacts the mother–child relationship, but also places the child at risk for developing depressive symptoms. However, certain parenting behaviors might buffer the association between maternal and youth depression. The influence of maternal depressive symptoms and parenting behavior (i.e., acceptance, behavioral control, psychological control) on youth depressive symptoms were examined in the context of three models: (1) an additive/cumulative risk model, (2) a moderator model, and (3) a mediator model. Data were examined longitudinally at five time points when youth were 8–9 through 16–17 years of age. Results supported an additive/cumulative risk model, but did not support the moderator or mediator models. Low maternal acceptance, high behavioral control, and high psychological control were risk factors for child depressive symptoms at several time points, with maternal depressive symptoms exerting an additional risk at later time points. A group difference between the spina bifida and comparison youth was not supported. Findings indicate that in general, maternal parenting behavior is salient throughout childhood and early adolescence, but maternal depressive symptoms do not exert an influence until mid-adolescence. Family interventions should aim to promote maternal mental health and maternal parenting behaviors to reduce the risk of the development of depressive symptoms in adolescence.

Keywords Maternal depression · Child depression · Adolescence · Parenting · Spina bifida

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Introduction

Maternal depression has long been an area of interest to psychologists because of the high prevalence rates of depression in women of child-bearing age and the negative impact maternal depression can have on the mother–child relationship and child developmental outcomes (Lovejoy et al. 2000). In addition, children of depressed mothers have an increased risk of developing depression themselves (Brennan et al. 2003; Eckshtain et al. 2010). Thus, gaining a greater understanding of the mechanisms through which maternal depression contributes to child depression is an important area of research.

Adolescence is a time when the risk for depression increases, especially in adolescents with chronic illnesses (Appleton et al. 1997). Although much research has examined risk factors associated with the development of depression in adolescents with diabetes (Eckshtain et al. 2010; Jaser et al. 2008), less attention has been paid to other conditions, including spina bifida (SB), which is the focus of the current study. SB is a congenital birth defect that affects 18 of every 1,000 live births in the United States annually (Centers for Disease Control 2008) and is associated with numerous health issues (i.e., hydrocephalus, muscle weakness, orthopedic problems, lack of bowel and bladder control) as well as cognitive, psychological, and social impairments (Bellin et al. 2009; Holmbeck and

Devine 2010). Additionally, adolescents with SB experience an increased risk for depressive symptoms compared to their able-bodied same-age peers (Appleton et al. 1997; Bellin et al. 2009; Holmbeck et al. 2003). However, mechanisms that contribute to the development of depression in adolescents with SB have received scant attention.

Family functioning exerts a powerful influence on the psychosocial development of youth with SB. Due to the fact that youth with SB often have fewer close friendships, they tend to spend more time with their family and thus are more heavily influenced by family relations than typically developing adolescents (Holmbeck and Devine 2010). In fact, one study of youth with SB found that mothers, as opposed to peers, were the most important source of support across several domains (Antle et al. 2009). Thus, it is clear that parents play an important role in the life of their child with SB. However, while many parents cope adaptively with the challenges associated with raising a child with a chronic illness, parents of children with SB have an elevated risk for experiencing high levels of stress, emotional distress, and less adaptive parenting behaviors (e.g., intrusiveness, psychological control; Holmbeck and Devine 2010). Therefore, youth with SB are more likely to be exposed to less adaptive parenting behaviors and to have a parent who is at risk for psychological distress.

Research on relations between parenting behaviors and child adjustment has identified three parenting behaviors that are particularly salient predictors of child adjustment: parental acceptance, behavioral control, and psychological control. Highly accepting parents are loving, approving, warm, involved, and emotionally supportive (Holmbeck et al. 2002); such behaviors facilitate positive psychosocial adjustment in children (Brennan et al. 2003; Jaser et al. 2008). Behavioral control is also typically associated with positive psychosocial adjustment; parents high on this dimension tend to monitor and supervise their child, set limits, regulate their child's activities, and enforce rules (Holmbeck et al. 2002; McKee et al. 2008). Finally, psychological control, which refers to "covert, psychological methods of controlling the child's activities and behaviors that would not permit the child to develop as an individual apart from the parent" (Schaefer 1965, p. 555), is associated with maladaptive outcomes including low self-worth and internalizing problems (Holmbeck et al. 2002).

Although the link between maternal depression and child depression has been well established in the developmental literature (Lovejoy et al. 2000; McKee et al. 2008), and to a lesser extent in the pediatric literature (Eckshtain et al. 2010; Jaser et al. 2008), specific parenting behaviors that might influence these associations have received less attention. Additionally, previous research examining parenting behaviors and youth outcomes in children with depressed mothers has yielded inconsistent results. In general, specific parenting behaviors (i.e., greater acceptance, lower psychological control) have been found to be related to resilient child outcomes (i.e., absence of psychological distress; Brennan et al. 2003). However, when specific outcomes are examined, some have found that parenting behaviors are related to the development of child depressive symptoms (Eckshtain et al. 2010) whereas others suggest parenting behaviors are only related to child externalizing problems (McKee et al. 2008). Although not always consistent, these findings suggest that parenting behaviors should be examined when considering relations between maternal and child depressive symptoms.

Previous research with youth with SB also suggests that parenting variables might impact the risk for child depression. Perceived parental support has been shown to be negatively related to depressed mood, especially in girls (Appleton et al. 1997). In addition, greater satisfaction with family functioning has been linked to lower levels of depressive symptoms (Bellin et al. 2009). Given these findings as well as findings from the broader developmental and pediatric literature (i.e., Brennan et al. 2003; Eckshtain et al. 2010; Jaser et al. 2008), it is reasonable to expect that parenting variables may influence or moderate the relation between maternal and youth depressive symptoms in adolescents with SB.

Our study examined the influence of three parenting variables (acceptance, behavioral control, psychological control) on the relation between maternal and child depressive symptoms in a sample of youth with SB and a matched comparison group of typically developing youth. Three different models were examined (see Fig. 1). First, an additive/cumulative risk model was examined, in which maternal depressive symptoms and parenting behaviors were expected to have additive effects on child symptoms (statistically represented by two main effects). Second, parenting variables were examined as moderator variables, whereby associations between maternal depressive symptoms and child symptoms would vary as a function of quality of parenting (represented as statistical interaction effects). Third, a meditational model was tested by examining whether earlier reports of parental depressive symptoms influenced later parenting behaviors, which then influenced subsequent levels of child depressive symptoms.

It was hypothesized that the pattern for maternal acceptance would be consistent with previous literature such that higher levels of maternal acceptance would be associated with lower levels of child depressive symptoms (Appleton et al. 1997; Bellin et al. 2009; Eckshtain et al. 2010). Conversely, high levels of psychological control were expected to be associated with higher levels of child depressive symptoms, which is also consistent with previous research (Butler et al. 2007). On the other hand, findings from previous research examining behavioral control

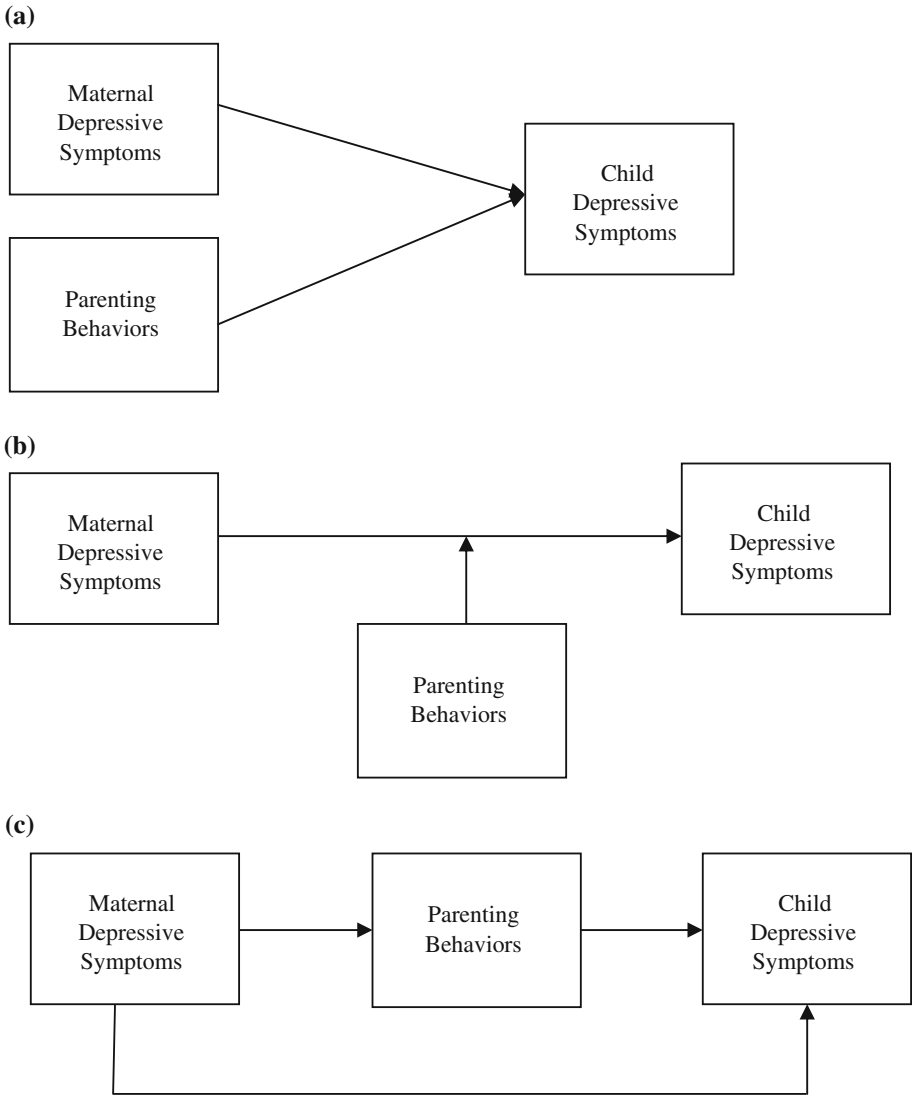

Fig. 1 Models of analyses conducted for each parenting variable (acceptance, behavioral control, psychological control). **a** Additive/cumulative risk model, **b** Moderator model, **c** Mediator model

have not been consistent. Although researchers typically assert that behavioral control is associated with lower levels of child depressive symptoms (Holmbeck et al. 2002; McKee et al. 2008), some have found that behavioral control exerts a negative influence, particularly on the functioning of older adolescents (i.e., Butler et al. 2007), while others found that behavioral control exerts no significant influence on child functioning (i.e., McKee et al. 2008). Due to the mixed findings from previous work, no hypotheses were proposed for behavioral control. Finally, given that youth with chronic illnesses spend more time with their parents and are more likely to depend on their parents, rather than peers, for support (Antle et al. 2009; Holmbeck and Devine 2010), it was expected that the relation between parenting behaviors and youth outcomes would be more robust in the SB sample than in the comparison sample. In other words, it was hypothesized that a group difference would be found and that the effect sizes would be larger in the SB sample than in the comparison sample.

Method

Sample

Participants in this study were part of a larger longitudinal study examining psychosocial adjustment of youth with SB during the transition to adolescence, funded by the March of Dimes (Holmbeck et al. 2003, 2010). At the time of the first data collection (Time 1), 68 families of eight- and nine-year-olds with SB (37 males, 31 females; $M = 8.34$ years) and a matched comparison sample of 68 typically developing eight- and nine-year-olds ($M = 8.49$ years) were interviewed in their homes. Participants in the comparison group were matched with those in the SB

group on the following demographic variables: child age, child gender, child ethnicity, birth order, family structure (intact versus not intact), socioeconomic status, and age of parents (see Holmbeck et al. 2003, for further details on the demographics of each sample).

A significant difference was found between the samples on a measure of receptive language (Peabody Picture Vocabulary Test–Revised; PPVT–R; Dunn and Dunn 1981): $M = 92.49$ ($SD = 18.49$) for the SB sample and $M = 108.97$ ($SD = 15.06$) for the comparison sample. This finding was expected given results from previous research which indicate that children with SB typically score in the low-average range on measures of verbal IQ (Wills et al. 1990). Due to the fact that lower receptive vocabulary scores were viewed as part of the symptom presentation in youth with SB (much like ambulation difficulties, for example) and because children with SB are usually mainstreamed into classrooms with their typically developing peers, no attempt was made to match the samples on this variable. However, PPVT–R scores were included as a covariate in analyses.

Data collection for the larger longitudinal study occurred every 2 years. The present study included data from the first through fifth waves of data collection (Time 1 through Time 5) when youth were 8–9 through 16–17 years of age, respectively. The number of families that participated over time were as follows: Time 2, 67 SB (99% retention rate) and 66 comparison (C; 97%); Time 3, 64 SB (94%) and 66 C (97%); Time 4, 60 SB (88%) and 65 C (96%); and Time 5, 52 SB (76%) and 61 C (90%). When the initial sample at Time 1 was compared to the remaining sample at Time 5, no differences in child age, gender, ethnicity, maternal age, or maternal marital status were found between those families who were included in analyses versus those not included for either the SB or comparison groups. However, families in the comparison sample from high SES backgrounds were more likely to complete the measures over time relative to comparison families from low SES backgrounds, χ^2 (1) = 4.2, $P < 0.05$. This difference between families from high versus low SES backgrounds in the SB sample was not significant, χ^2 (1) = 3.5, $P > 0.05$. Additionally, children with SB who were included in analyses had higher PPVT-R scores compared to those who were not included, $t(65) = 2.87$, $P < 0.01$, whereas there were no such significant differences in the comparison sample, $t(66) = 1.31$, $P > 0.05$.

Participant Recruitment

Children with SB were originally identified and recruited from four sources: a children's hospital, a children's hospital that cares exclusively for children with physical disabilities, a university-based medical center, and a statewide SB association. The majority of youth with SB had myelomeningocele (82%); 12% had lipomeningocele, and 6% had another type of SB. The location of spinal lesion also varied: 32% sacral, 54% lumbar, and 13% thoracic. Most children with SB had a shunt (71%), with an average of 2.50 ($SD = 2.91$) shunt surgeries prior to Time 1. Sixty-three percent of participants with SB ambulated with braces, 18% used a wheelchair, and 19% walked unassisted. The Time 1 sample of 68 children with SB did not differ significantly from children of families who declined to participate in terms of lesion level, χ^2 (2, $N = 116$) = 0.62, $P > 0.05$, or type of SB, χ^2 (1, $N = 119$) = 1.63, $P > 0.05$. Families in the comparison group were recruited by contacting schools at which participating children with SB were enrolled (see Holmbeck et al. 2003, for a description of the recruitment and matching procedures).

Within the final sample of participating families, all children and biological mothers took part in the study at Time 1. Fifty-five (81%) biological fathers/step-fathers of children with SB and 52 (76%) biological/step-fathers of comparison children also participated. The majority of families were White ($n = 113$; 86.76%), but the sample was diverse with respect to socioeconomic status ($M = 44.79$; $SD = 21.46$), as measured by the Hollingshead Four Factor Index (Hollingshead 1975) of socioeconomic status.

Procedure

Prior to data collection, human subjects' approval was granted by the Institutional Review Board (IRB) at the researchers' home institution and by IRBs at all cooperating hospitals from which participants were recruited. Data for each wave of the study were collected during three-hour visits to each family's home, which were conducted by trained graduate and undergraduate research assistants. Each session began with a brief overview of study goals and a review of confidentiality issues. Parents provided consent for themselves and their children to participate and youth provided assent for their own participation at each time point. Parents also signed a release of information form for medical chart reviews and for children's teachers to complete a set of questionnaires. Families then completed several questionnaire packets, a series of videotaped family interaction tasks, and audio-recorded self-administered interviews. To aid comprehension of measures, research assistants were available to read questionnaires aloud to participants when needed. In addition, laminated cards illustrating Likert-scale item response options were provided to youth to facilitate accurate responses. Families received monetary compensation for their participation at each time point.

Measures

Data used for the current study were obtained through mother- and youth-report on questionnaire measures. Mothers and children each reported on their own emotional functioning. Mother- and youth-report of parental behaviors were each obtained to gain multiple perspectives on these behaviors. This data collection strategy also served to decrease the impact of common method variance by employing mother-report for the independent variable (i.e., maternal depressive symptoms), child-report for the dependent variable (i.e., child depressive symptoms), and both mother- and child-report for the proposed intervening parenting behaviors (i.e., acceptance, behavioral control, and psychological control).

Demographics

The Parent Demographic Questionnaire (PDQ), developed for this study, was used to assess factors such as child age, parent education level, and parent occupation.

Socioeconomic Status (SES)

The Hollingshead Four Factor Index of socioeconomic status was used to assess SES (Hollingshead 1975). SES was derived by assigning a score to mothers' and fathers' occupations and education level. Education and occupation scores were combined and averaged across caregivers to calculate the family SES. In the case of single-parent families, or two-parent families in which only one parent was employed, that individual's score was used to represent the family. Higher scores reflected higher SES.

Maternal Depressive Symptoms

The Depression subscale of the Symptom Checklist—Revised (SCL-90-R; Derogatis et al. 1976) was used to assess maternal depressive symptoms. The Depression subscale, which has demonstrated acceptable internal consistency in previous research ($\alpha = 0.90$; Derogatis et al. 1976), consists of 13 items from the larger 90 item measure. Items are rated on a five-point rating scale, ranging from 0 (not at all distressed) to 4 (extremely distressed), for symptoms experienced over the past week. A score at or above 28 indicates clinically elevated levels of depressive symptoms. Alphas at Time 1 were 0.91 and 0.89 for the SB and comparison samples, respectively, indicating adequate internal consistency of the measure for this sample.

Child Depressive Symptoms

Youth depressive symptoms were assessed with child-report on the Children's Depression Inventory (CDI; Kovacs 1992). Respondents rated their degree of depressive symptomatology on this 27-item measure by choosing among three options representing three levels of symptom severity (with higher scores indicating a greater degree of depressive symptoms). The total score, which is based on five dimensions of depressive symptoms, was used in this study with the following clinical cutoff scores: 25 for boys and 23 for girls ages 7–12 and 28 for boys and 22 for girls ages 13–17. However, it is important to note that some have suggested recommended cutoff scores should be used with caution when screening for depression because they may yield a high number of false negatives (i.e., children with significant depressive symptoms will not be detected; Matthey and Petrovski 2002). Alphas at Time 1 were 0.81 and 0.80 for the SB and comparison samples, respectively, indicating adequate internal consistency of the measure for this sample.

Maternal Parenting Behaviors

Maternal acceptance, behavioral control, and psychological control were assessed with an abbreviated version of the Child Report of Parental Behavior Inventory (CRPBI; Schludermann and Schludermann 1970). The original CRPBI is a 108-item scale that assesses maternal and paternal parenting behaviors. Items are rated by respondents on a three-point scale: "not like", "somewhat like", or "a lot like" the parent. Three second-order factors (acceptance-rejection, firm control-lax control, and psychological control-psychological autonomy) are derived from items included in 18 first-order subscales. For our study, this measure was adapted for response by parents based on rewording procedures described in Schwarz et al. 1985. Because of time considerations, 44 items from the larger 108-item scale were administered, which included all items from the following first-order subscales: Acceptance (8 items) and Rejection (8 items, reverse scored) from the acceptance-rejection factor; Control (5 items), Enforcement (5 items), and Lax Discipline (5 items, reverse scored) from the firm control-lax control factor; and Intrusiveness (5 items) and Hostile Control (8 items) from the psychological control-psychological autonomy scale. For our study, a composite score based on mother and child report was created to yield three scales of parenting behavior: Maternal Acceptance, Maternal Behavioral Control, and Maternal Psychological Control. Alphas at Time 1 ranged from 0.60 to 0.78 across reporters for the SB group and from 0.55 to 0.83 for the comparison group.

Analysis Plan

Regression analyses were conducted to examine the association between maternal and youth depressive symptoms,

with three parenting behaviors as possible moderators or mediators. Prior to analyses, the continuous independent variables (maternal depressive symptoms and maternal parenting behaviors), were centered at the sample mean and interaction terms were created by multiplying the centered predictors (Aiken and West 1991). For each regression, data collected at Time 1 on the PPVT-R and SB status (SB or comparison group) were entered as covariates to statistically control for differences between groups and the variance associated with the PPVT-R. In addition, because prior analyses with this sample have found SES to be a significant predictor of adjustment (e.g., Holmbeck et al. 2003), Time 1 SES was entered as a covariate in all analyses.

The additive and moderator models were examined in the same set of multiple regression analyses. To test the additive model, the relation between two main effects (maternal depressive symptoms and maternal parenting behaviors) and child depressive symptoms was examined. To test the moderator model, interaction terms were included after the main effects to examine the potential moderating role of the parenting variables. A hierarchical-stepwise procedure was used, with blocks of variables entered using the forward selection technique. PPVT-R, SES, and SB status were entered in the first block; maternal depressive symptoms and the parenting variable were entered in the second block; three two-way interactions (maternal depressive symptoms X parenting style, maternal depressive symptoms X group, parenting style X group) were entered in the third block; and a three-way interaction (maternal depressive symptoms X parenting style X group) was entered in the fourth step. Separate analyses were conducted for each parenting variable at all five time points; therefore, fifteen regressions were run in total.

To explore the proposed mediator models, a series of multiple regression analyses were conducted according to the methods outlined by Baron and Kenny (1986) and Holmbeck (1997): (1) maternal depressive symptoms (the independent variable; IV) must be significantly associated with the parenting variable (the mediator), (2) the IV must be significantly associated with child depressive symptoms (the dependent variable; DV), (3) the mediator must be significantly associated with the DV, with the IV controlled, and (4) the impact of the IV on the DV must be less after controlling for the mediator. Mediator analyses were run for each parenting variable using three different combinations of time points: (1) Time 1, 2 and 3, (2) Time 2, 3, and 4, and (3) Time 3, 4, and 5. The IV was represented by the earliest time point, the proposed mediator by the middle time point, and the DV by the latest time point in each set of analyses. In addition, data obtained at the previous time point for the mediator and DV were included in the first step of the regression equation to statistically control for earlier waves of the mediator and DV, respectively (e.g., when the DV at Time 3 was examined, the first step of the regression equation included the DV at Time 2). In total, nine mediator models were examined.

Results

The mediator model was not supported for any of the parenting variables at any time point. For the SB group, earlier levels of maternal depressive symptoms were not related to later parenting behaviors or later levels of child depressive symptoms (i.e., the IV was not significantly related with the mediator or the DV) at any time point. For the comparison group, similar results were found at earlier time points (i.e., mediator models at Time 1, 2 and 3 and at Time 2, 3, and 4). Conversely, in the mediator model that examined Times 3, 4, and 5, maternal depressive symptoms at Time 3 were significantly related to child depressive symptoms at Time 5, after controlling for child depressive symptoms at Time 4, $\beta = 0.21$; $F(2, 56) = 20.25$, $P < 0.05$. However, maternal depressive symptoms at Time 3 were not significantly related to any of the three parenting behaviors at Time 4 (i.e., the IV was not significantly related to the mediator). In addition, after controlling for child depressive symptoms at Time 4, the three parenting behaviors at Time 4 were not significantly related to child depressive symptoms at Time 5 (i.e., the mediator was not significantly related to the DV). Therefore, the mediator model was not supported in any of the analyses. Due to the large number of regressions, results will only be presented for those analyses that examined the additive/cumulative risk and moderator models. In addition, the effect size for multiple regression (f^2, computed using the Free Effect Size Calculator for Multiple Regression created by Daniel Soper) is reported for each model to indicate the magnitude of the finding.

Findings for Covariates

As can be seen in Tables 1, 2, and 3, there was a significant main effect for SB Status at Time 1, $\beta = -0.23$; $F(1, 122) = 6.81$, $P < 0.05$, $f^2 = 0.06$, indicating that children with SB reported greater depressive symptoms than comparison children. SB Status was not a significant predictor of child depressive symptoms at any other time point. There was a significant negative main effect for SES at Time 2, $\beta = -0.18$; $F(1, 121) = 4.30$, $P < 0.05$; Time 3, $\beta = -0.20$; $F(1, 121) = 4.94$, $P < 0.05$; and Time 4, $\beta = -0.22$; $F(1, 121) = 5.72$, $P < 0.05$; lower SES was associated with greater child depressive symptoms. PPVT-R was not a significant predictor at any time point.

Parental Acceptance

Regression findings for associations between maternal depressive symptoms and maternal acceptance predicting child depressive symptoms are presented in Table 1. With the exception of Time 1, maternal acceptance yielded a significant main effect at all time points, indicating that higher levels of maternal acceptance were associated with lower levels of child depressive symptoms at Time 2, $\beta = -0.20$; $F(1, 119) = 4.98$, $P < 0.05$, $f^2 = 0.09$; Time 3, $\beta = -0.40$; $F(1, 119) = 22.48$, $P < 0.001$, $f^2 = 0.27$; Time 4, $\beta = -0.50$; $F(1, 119) = 40.36$, $P < 0.001$, $f^2 = 0.46$; and Time 5, $\beta = -0.48$; $F(1, 119) = 30.29$, $P < 0.001$. Contrary to hypotheses, maternal depressive symptoms were not associated with child depressive symptoms at the first four time points. However, at Time 5, maternal depressive symptoms were positively associated with child depressive symptoms, $\beta = 0.24$; $F(1, 118) = 7.41$, $P < 0.01$, $f^2 = 0.45$, even after the variance for maternal acceptance was accounted for. There were no significant two- or three-way interactions. Thus, a moderator model was not supported, but an additive/cumulative risk model was supported at Time 5.

Parental Behavioral Control

Table 2 presents regression findings for associations between maternal depressive symptoms and maternal behavioral control predicting child depressive symptoms. Maternal behavioral control yielded a significant main effect at Time 2, $\beta = 0.18$; $F(1, 119) = 4.23$, $P < 0.05$, $f^2 = 0.08$; Time 3, $\beta = 0.22$; $F(1, 119) = 6.26$, $P < 0.05$; and Time 5, $\beta = 0.19$; $F(1, 118) = 4.14$, $P < 0.05$, indicating that increased levels of maternal behavioral control are associated with higher levels of child depressive symptoms. In addition, maternal depressive symptoms were positively associated with child depressive symptoms at Time 3, $\beta = 0.23$; $F(1, 118) = 6.96$, $P < 0.01$, $f^2 = 0.19$ and Time 5, $\beta = 0.33$; $F(1, 119) = 11.61$, $P < 0.01$, $f^2 = 0.20$. Once again, there were no significant two- or three-way interactions. Thus, a

Table 1 Maternal acceptance and depressive symptoms predicting child depressive symptoms: cross-sectional analyses

Step		R	$R^2\Delta$	β	FΔ	f^2
			Time 1			
1	SB status	0.23	0.05	−0.23	6.81*	0.06
			Time 2			
1	Time 1 SES	0.18	0.03	−0.18	4.30*	
2	SB status	0.21	0.01	−0.17	1.12	
3	Time 1 PPVT-R	0.21	0.00	−0.04	0.14	
4	Time 2 acceptance	0.28	0.04	−0.20	4.98*	0.09
			Time 3			
1	Time 1 SES	0.20	0.04	−0.20	4.94*	
2	SB status	0.25	0.02	0.15	2.78	
3	Time 1 PPVT-R	0.25	0.00	0.05	0.27	
4	Time 3 acceptance	0.46	0.15	−0.40	22.48***	0.27
			Time 4			
1	Time 1 SES	0.22	0.05	−0.22	5.72*	
2	SB status	0.26	0.02	0.14	2.15	
3	Time 1 PPVT-R	0.26	0.00	−0.04	0.16	
4	Time 4 acceptance	0.56	0.25	−0.50	40.36***	0.46
			Time 5			
1	Time 1 PPVT-R	0.14	0.02	0.14	1.92	
2	Time 1 SES	0.16	0.01	−0.09	0.80	
3	SB status	0.16	0.00	0.01	0.00	
4	Time 5 acceptance	0.51	0.23	−0.48	30.29***	
5	Time 5 mother depressive symptoms	0.56	0.05	0.24	7.41**	0.45

Cross-sectional regression results for predictors of child depressive symptoms from maternal depressive symptoms and acceptance, including tests of additive/cumulative and moderator models. The qualitative descriptors for f^2 are: 0.02 = small, 0.15 = medium, 0.35 = large

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

Table 2 Maternal behavioral control and depressive symptoms predicting child depressive symptoms: cross-sectional analyses

Step		R	$R^2\Delta$	β	FΔ	f^2
			Time 1			
1	SB status	0.23	0.05	−0.23	6.81*	0.06
			Time 2			
1	Time 1 SES	0.18	0.03	−0.18	4.30*	
2	SB status	0.21	0.01	−0.09	1.12	
3	Time 1 PPVT-R	0.21	0.00	−0.04	0.14	
4	Time 2 behavioral control	0.27	0.03	0.18	4.23*	0.08
			Time 3			
1	Time 1 SES	0.20	0.04	−0.20	4.94*	
2	SB status	0.25	0.02	0.15	2.77	
3	Time 1 PPVT-R	0.25	0.00	0.05	0.27	
4	Time 3 behavioral control	0.33	0.05	0.22	6.26*	
5	Time 3 mother depressive symptoms	0.40	0.05	0.23	6.96**	0.19
			Time 4			
1	Time 1 SES	0.22	0.05	−0.22	5.72*	0.05
			Time 5			
1	Time 1 PPVT-R	0.14	0.02	0.14	1.92	
2	Time 1 SES	0.16	0.01	−0.09	0.80	
3	SB status	0.16	0.00	0.01	0.01	
4	Time 5 mother depressive symptoms	0.36	0.10	0.33	11.61**	
5	Time 5 behavioral control	0.41	0.04	0.19	4.14*	0.20

Cross-sectional regression results for predictors of child depressive symptoms from maternal depressive symptoms and behavioral control, including tests of additive/cumulative and moderator models. The qualitative descriptors for f^2 are: 0.02 = small, 0.15 = medium, 0.35 = large

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

moderator model was not supported, but an additive/cumulative risk model was supported at Times 3 and 5.

Parental Psychological Control

Regression findings for associations between maternal depressive symptoms and maternal psychological control predicting child depressive symptoms are presented in Table 3. Maternal psychological control yielded a significant main effect at Time 3, $\beta = 0.37$; $F(1, 119) = 17.70$, $P < 0.001, f^2 = 0.23$; Time 4, $\beta = 0.33$; $F(1, 119) = 12.93$, $p < 0.001$, $f^2 = 0.19$; and Time 5, $\beta = 0.47$; $F(1, 119) = 25.01, P < 0.001$, indicating that increased levels of maternal psychological control are associated with increased child depressive symptoms. Maternal depressive symptoms were positively associated with child depressive symptoms only at Time 5, $\beta = 0.24$; $F(1, 118) = 7.11$, $P < 0.01$, $f^2 = 0.38$. In addition, a significant two-way interaction between psychological control and maternal depressive symptoms was found at Time 2, $\beta = 0.23$; $F(1, 117) = 6.87$, $P < 0.05$, $f^2 = 0.11$. When this interaction was explored further, the simple slopes for associations between maternal depressive symptoms and child depressive symptoms under conditions of low and high psychological control were both statistically significant, $F(5, 121) = 2.56$, $P < 0.05$ (F-value was identical in both conditions), and in a negative direction ($\beta = -2.18$ and $\beta = -1.68$, respectively). In other words, higher levels of maternal depressive symptoms were associated with lower levels of child depressive symptoms under both low and high levels of psychological control, which runs contrary to the hypotheses. Overall, however, results support an additive/cumulative risk model for maternal depressive symptoms and psychological control predicting child depressive symptoms at Time 5.

Discussion

This study examined the extent to which three parenting behaviors (acceptance, behavioral control, psychological control) influence relations between child and maternal depressive symptoms in youth with SB and a comparison group of typically developing youth. To explore potential mechanisms through which parenting behaviors impact the relation between parent and youth depressive symptoms, three models were tested: (1) an additive/cumulative risk model, (2) a moderator model, and (3) a mediator model (see Fig. 1). In general, results did not support the moderator or

Table 3 Maternal psychological control and depressive symptoms predicting child depressive symptoms: cross-sectional analyses

Step		R	$R^2\Delta$	β	FΔ	f^2
			Time 1			
1	SB status	0.23	0.05	−0.23	6.81*	0.06
			Time 2			
1	Time 1 SES	0.18	0.03	−0.18	4.30*	
2	SB status	0.21	0.01	−0.09	1.12	
3	Time 1 PPVT-R	0.21	0.00	−0.04	0.14	
4	Time 2 psychological control	0.22	0.00	0.07	0.43	
5	Time 2 mother depressive symptoms	0.22	0.00	0.01	0.01	
6	Psych control X maternal depressive symptoms	0.31	0.05	0.23	6.87*	0.11
			Time 3			
1	Time 1 SES	0.20	0.04	−0.20	4.94*	
2	SB status	0.25	0.02	0.15	2.77	
3	Time 1 PPVT-R	0.25	0.00	0.05	0.27	
4	Time 3 psychological control	0.43	0.12	0.37	17.70***	0.23
			Time 4			
1	Time 1 SES	0.22	0.05	−0.22	5.72*	
2	SB status	0.26	0.02	0.14	2.15	
3	Time 1 PPVT-R	0.26	0.00	−0.04	0.16	
4	Time 4 psychological control	0.40	0.10	0.33	12.93***	0.19
			Time 5			
1	Time 1 PPVT-R	0.14	0.02	0.14	1.92	
2	Time 1 SES	0.16	0.01	−0.09	0.80	
3	SB status	0.16	0.00	0.01	0.01	
4	Time 5 psychological control	0.47	0.20	0.47	25.01***	
5	Time 5 mother depressive symptoms	0.53	0.05	0.24	7.11**	0.38

Cross-sectional regression results for predictors of child depressive symptoms from maternal depressive symptoms and psychological control, including tests of additive/cumulative and moderator models. The qualitative descriptors for f^2 are: 0.02 = small, 0.15 = medium, 0.35 = large

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

mediator models. However, parenting behavior was significantly related to child depressive symptoms at most time points and maternal depressive symptoms were linked with child depressive symptoms in mid-adolescence. Therefore, an additive/cumulative risk model was supported at Time 5 for all parenting behaviors as well as at Time 3 for behavioral control. A significant group effect was only found at the first time point, suggesting that although preadolescents with SB are at greater risk of experiencing depressive symptoms when compared to their same-age peers, this increased risk is not found as youth reach adolescence. It is also important to note that with the exception of this group difference, findings were similar across groups in analyses examining the additive/cumulative risk model. In other words, when compared to their same-age peers, youth with SB did not experience an increased risk of developing depressive symptoms if their mother exhibited depressive symptoms or less adaptive parenting behaviors.

Overall, the impact of maternal acceptance and psychological control on child depressive symptoms was consistent with the hypotheses for this study and findings from previous research. With the exception of the first data collection time point, a significant main effect for maternal acceptance was found at all time points. The relation between maternal acceptance and child depressive symptoms was negative at all time points, indicating that youth with mothers who exhibited lower levels of acceptance experienced more depressive symptoms. This finding is not surprising given the large body of work supporting a positive relation between maternal acceptance and child adjustment (Brennan et al. 2003; Holmbeck et al. 2002; Jaser et al. 2008). Psychological control yielded similar findings, although in the opposite direction. Youth with mothers who exhibited higher levels of psychological control experienced greater depressive symptoms at the three later time points. This is also consistent with previous research suggesting that parental psychological control is associated with an increased risk of internalizing problems and adjustment difficulties (Brennan et al. 2003; Holmbeck et al. 2002). Finally, a significant interaction between

maternal depressive symptoms and psychological control emerged at Time 2. When this interaction was explored, the simple slopes for both low and high psychological control were significantly different from zero and in a negative direction, indicating that higher levels of maternal depressive symptoms were associated with lower levels of child depressive symptoms under conditions of both low and high levels of psychological control. This finding was unexpected and contradicts the main effect findings. Given the number of analyses conducted in this study, this finding should be interpreted with caution because it is possible that the finding is spurious. Future research should continue to explore the impact of maternal depressive symptoms and psychological control on youth depressive symptoms to better understand the relation between these variables.

A significant main effect for behavioral control was found at Time 2, Time 3, and Time 5 and results indicated that youth with mothers who exhibited higher levels of behavioral control experienced greater depressive symptoms. This suggests that although some researchers theorize that behavioral control is associated with positive adjustment in general (Holmbeck et al. 2002; McKee et al. 2008), this association might not hold across all domains of functioning; that is, greater behavioral control might actually be a risk factor for developing internalizing problems. In addition, some have suggested that aspects of behavioral control might be perceived as being similar to psychological control for some groups of youth, particularly older adolescents (Butler et al. 2007). Additional research on the impact of parental behavioral control on youth functioning, and particularly on the development of internalizing problems, is warranted to further explore this possibility.

Taken together, findings from the additive/cumulative risk model suggest that the differential influence of maternal parenting behavior and maternal depressive symptoms on child depressive symptoms is not uniform across development. Beginning in childhood, it appears that maternal parenting behavior has a significant impact on child development such that less adaptive parenting places a child at risk for developing depressive symptoms. However, in mid-adolescence, maternal depressive symptoms become an additional risk factor for the development of adolescent depressive symptoms. One possible explanation for this finding is that the negative impact of maternal depressive symptoms may take longer to accumulate because it is experienced less directly by the child, whereas parenting behaviors are experienced more directly and thus have a more immediate effect. In addition, adolescence is a developmental period associated with an increased risk of depressive symptomatology, and it is possible that the impact of maternal depressive symptoms does not become salient until depressive symptoms emerge in the child. The developmental stage of the child may also impact the degree to which the child models the affective state of his or her mother, such that older youth who are more aware of their mother's depressive symptoms are more likely to exhibit depressive symptoms themselves. Additional longitudinal research is needed to better understand differences in how these maternal risk factors are associated with the development of depressive symptomatology in children over the course of development.

A mediator model was not supported at any time point. Specifically, earlier levels of maternal depressive symptoms were not related to later maternal parenting behavior or levels of child depressive symptoms in the SB or comparison samples. Although Time 3 maternal depressive symptoms were related to Time 5 child depressive symptoms in the comparison group, the complete mediator model was not supported because earlier maternal depressive symptoms were not related to later parenting behavior, which were not related to later child depressive symptoms. This suggests that, for this sample, maternal depressive symptoms did not significantly impact maternal parenting behaviors. Mediator analyses are conducted longitudinally and control for earlier waves of data; therefore, it is possible that the null findings are due to stability of these variables over time, thereby leaving little variance in the dependent variables (i.e., parenting behavior and child depressive symptoms) after controlling for earlier levels of those variables.

A moderator model was also not supported at any time point. With the exception of one interaction effect at Time 2, the interaction between maternal depressive symptoms and maternal parenting behavior was not significant for any of the parenting behaviors. Greater statistical power is required to detect interaction effects than is required to detect main effects (Aiken and West 1991). In addition, few mothers and children in this sample reported clinically elevated levels of depressive symptoms. Therefore, it is possible that interaction effects were not detected because: (1) the sample size was insufficient to achieve ample power and (2) there was limited variance in the independent and dependent variables. Additional research with larger sample sizes is warranted to further examine the possible moderating role parenting behaviors might exert on the relation between maternal and youth depressive symptoms.

The design of this study had several strengths. First, it examined the relation between maternal depressive symptoms, maternal parenting behaviors, and child depressive symptoms longitudinally, beginning when participants were between the ages of eight and nine (Time 1), through sixteen and seventeen years of age (Time 5). Data were analyzed separately at each time point to determine if the relation between variables changed over time. Second, this study included two groups, SB and comparison youth

(matched on demographic characteristics). Thus, this study allows for important comparisons to be made between youth with SB and their same-aged typically developing peers. Finally, multiple respondents were included such that mothers reported on their depressive symptoms, children reported on their depressive symptoms, and both mothers and children reported on mothers' parenting behaviors. The use of multi-informant data is particularly important because mothers with greater depressive symptoms are more likely to express negative affect and view their child's behavior more negatively than mothers with lower levels of depressive symptoms (Lovejoy et al. 2000; McKee et al. 2008).

Although this study had several strengths, it also has many potential limitations. First, a relatively small number of participants reported clinically elevated levels of depressive symptoms. Across the five time points, the number of mothers reporting clinically elevated levels of depressive symptoms in the SB group ranged from zero to two (0–3.3%) and from one to three (1.5–4.5%) in the comparison group. The number of youth reporting clinically elevated levels of depressive symptoms ranged from 1 to 3 (1.5–6.0%) in the SB group and 0 to 3 (0–5.1%) in the comparison group. Thus, results of this study may not generalize to populations experiencing clinical levels of depressive symptoms. Second, we chose not to include fathers in analyses because the number of fathers who completed measures at later time points (T5 $n = 33$ and 44 in SB and comparison groups, respectively) was too few to yield adequate power for the statistical analyses. Many have recognized the importance of including fathers in psychological research (Phares 1992) and future research should explore the extent to which paternal parenting behaviors impact the relation between parental depressive symptoms, parenting behaviors, and child depressive symptoms. Third, although the prevalence of SB is relatively high in Latino populations (Lary and Edmonds 1996), the current sample did not include a representative number of Latino participants. Future studies should include greater numbers of Latino families. Fourth, although some youth with SB experience profound cognitive impairment, only those youth who were able to provide assent and reliably complete measures were included in this study. Therefore, the findings of this study may not necessarily reflect maternal-child relations in cases of extreme disability. Fifth, as mentioned earlier, reduced statistical power may have undermined our ability to detect interaction effects. In addition, the limited sample size restricted us from including other potentially meaningful covariates (e.g., strength of peer relationships) in the analyses. Finally, given the large number of analyses, some findings may have emerged by chance.

The findings of this study have important implications for clinicians and professionals who work with families with a child who has SB as well as families who have typically developing offspring. Results highlight the important impact parenting behaviors have on child psychological functioning beginning in pre-adolescence and continuing through mid-adolescence. Thus, interventions for families should aim to enhance and foster a warm, accepting relationship between the mother and child. Further, they should provide parents who exhibit high levels of behavioral and psychological control with alternative strategies (e.g., rewards, verbal praise) to more effectively manage child behavior, while encouraging children to develop their own independence and autonomy. Finally, the fact that maternal depressive symptoms emerged as an additional risk factor in mid-adolescence suggests family-based interventions would be especially effective for adolescents whose mothers are experiencing depressive symptoms.

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