Published in final edited form as:

J Fam Psychol. 2011 February ; 25(1): 137–146. doi:10.1037/a0022452.

Effects of Paternal and Maternal Depressive Symptoms on Child Internalizing Symptoms and Asthma Disease Activity: Mediation by Interparental Negativity and Parenting

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Abstract

This study tested a hypothesized model of the relationships among parental depressive symptoms, family process (interparental negativity and negative parenting behavior), child internalizing symptoms and asthma disease activity. One hundred and six children with asthma, aged 7 to 17, participated with their fathers and mothers. Parental depressive symptoms were assessed by self-report. Interparental and parenting behaviors were observed and rated during family discussion tasks. Child internalizing symptoms were assessed by self-report and by clinician interview and rating. Asthma disease activity was assessed according to National Heart Lung and Blood Institute guidelines. Results of structural equation modeling generally supported interparental negativity and negative parenting behavior as mediators linking parental depressive symptoms and child emotional and physical dysfunction. However, paternal and maternal depressive symptoms play their role through different pathways of negative family process.

Keywords

parental depressive symptoms; interparental negativity; negative parenting; child internalizing symptoms; asthma disease activity

The purpose of this study was to examine family process pathways by which parental depressive symptoms may influence child psychological and physical functioning. Previous research has documented associations among parental depression, family process and child

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internalizing symptoms (see Hammen, 2009; Kaslow, Jones, & Palin, 2005 for reviews), but few studies have examined the adverse effects of parental depression on child physical, as well as psychological, functioning. This is an important gap in the literature given current appreciation of the essential interaction between mind and body aspects of health and illness. Furthermore, although there is ample research indicating relations among subsets of these factors (see review below), to our knowledge there is no study that systematically investigates parental depression, family process, and child psychological and physical disorder in the same model. The goal of our line of research is to use a multi-level model, the Biobehavioral Family Model, to develop further understanding of the nature of the complex inter-relations of parental depressive symptoms, family relational stress and child physical and emotional compromise. We chose children with pediatric asthma as subjects of investigation because there is evidence for psychobiologic transmission of the effects of both family relational stress and child depression on asthma (Chen & Miller, 2007; Miller, Chen, & Cole, 2009), thus demonstrating plausible pathways of effect. We will review the current literature on parental depression, family process and child internalizing disorder, and the evidence for the impact of family relational stress on pediatric asthma. Then we will present a hypothetical model and rationale for a systemic study of the inter-relations of these factors in child internalizing symptoms and asthma.

Parental depression and family process in relation to child internalizing problems

Research indicates that children of depressed parents are at specific risk for internalizing problems such as depression and anxiety (Hammen, 2009). Evidence supporting the role of family process in relation to this effect is substantial (Davila, Stroud, & Starr, 2009; Kaczynski, Lindahl, Malik, & Laurenceau, 2006). Also there is considerable evidence that spousal depression and marital distress are complexly inter-related, with mutual causal effects and serious fallout for children (Davila et al., 2009; Joiner & Timmons, 2009). Specifically, studies found that parental depression was related to negative conflict strategies, which in turn, were linked to child internalizing symptoms (Du Rocher Schudlich, Papp, & Cummings, 2004). There is robust evidence from marital studies that conflictual interparental behavior is associated with child internalizing symptoms (Cummings & Davies, 2010). One theory is that compromised emotional security and parenting difficulties/attachment insecurity mediates this effect (Cummings & Davies, 2010). The “spillover” hypothesis posits that parental negative affect from interparental conflict carries over into parental interactions with children, including unresponsiveness, rejection, and hostile discipline which renders the child vulnerable to negative outcomes, including depression (Erel & Burman, 1995; Kaczynski et al., 2006). Studies also show a direct effect of interparental conflict on physiological dysregulation in children which is associated with internalizing symptoms and physical disease (El-Sheikh, 2005).

There is also evidence that parental depression affects child depression directly through negative parenting (Hammen, 2009). Meta-analytic reviews suggest that parental depression is strongly associated with negative, coercive parenting behavior (Kane & Garber, 2004a; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Moreover, meta-analytic reviews of the relation between parenting and childhood psychopathology indicate that negative parenting is consistently associated with child depression (McLeod, Weisz, & Wood, 2007) and modestly associated with anxiety (McLeod, Wood, & Weisz, 2007). Paternal depression is significantly related to both father-child conflict and child internalizing symptoms (Kane & Garber, 2004b). More recently the effects of both maternal and paternal depression on child depression are being examined in the same studies. The results overall support paternal as well as maternal effects, but maternal effects seem to be greater in most studies (Hammen, 2009).

Little is known about potential differential effects of paternal versus maternal depression on interparental negativity and their effects on parenting. However, Cummings and colleagues (2005, 2008) found that marital conflict mediated relations between maternal and paternal depressive symptoms and multiple problems in children. We found no studies that examined parental depressive symptoms, interparental negativity, parenting and child psychological and physical outcomes in the same model. Our study begins to close this gap in the literature.

Pediatric asthma and the effects of stressful family process

Asthma is a chronic disorder of the airways characterized by reversible airway obstruction, bronchial hyper-responsiveness, and airway inflammation leading to loss of air during acute attacks (National Heart Lung and Blood Institute, 2007). It is one of the most common illnesses of childhood, affecting nearly 9% of children in the U. S. (Federal Interagency Forum on Child and Family Statistics, 2010), and the prevalence of childhood asthma is increasing (Asher et al., 2006). Elevated levels of internalizing disorders are common in pediatric asthma (Katon, Lozano, Russo, McCauley, Richardson, & Bush, 2007), and these are associated with disease activity (McQuaid, Kopel, & Nassau, 2001). Moreover, research demonstrates psychobiologic pathways by which the emotional dysregulation of stress and child depression affects airway function in asthma (Chen & Miller, 2007; Miller, Wood, Lim, Ballow, & Hsu, 2009). Thus, for children with asthma, internalizing symptoms may be detrimental not only to psychological well-being, but also to physical function. Children with asthma might be particularly vulnerable to parental depressive symptoms and stressful family process because these processes may compromise physical functioning by way of the emotional and physiological dysregulation of internalizing psychological disorder. It is thus crucial to identify specific pathways by which family relational stress may aggravate internalizing symptoms in children with asthma.

The current study is part of a series of studies in a project aimed at understanding these pathways. Lim and colleagues (2008) published a study of children with asthma and their mothers (from one and two parent families) showing that maternal depressive symptoms predicted child internalizing symptoms mediated by negative parenting, and predicted disease activity mediated by child internalizing symptoms. The current study of two parent families extended that study by examining differential pathways of effect of paternal versus maternal depressive symptoms and by including interparental negativity as a mediator. About 40% of the mothers who participated in the current study were also examined in the first study.

The Biobehavioral Family Model

Our modeling derives from the BioBehavioral Family Model (BBFM) which emphasizes the interdependence of psychological and physical functioning in relation to family process and its influence on the child (Wood, Klebba, & Miller, 2000; Wood et al., 2008). The model rests upon assumptions of the interdependence of relational, emotional, and physiological processes consistent with the current “social neuroscience” paradigm (Cacioppo, Berntson, Ernst, & Ito, 2000). The BBFM posits, and findings for children with asthma support, that negative family emotional climate is associated with child internalizing symptoms which in turn affect child asthma disease activity through psychobiological pathways (Lim, Wood, & Miller, 2008; Miller et al., 2009; Wood et al., 2006, 2007, 2008). The current study extends the BBFM by examining pathways by which maternal and paternal depressive symptoms predict child internalizing symptoms mediated by interparental negativity and negative parenting (aspects of negative emotional climate), and by examining whether these

associations of family process with asthma disease activity are mediated by child internalizing symptoms. Figure 1 shows the specified pathways in the hypothesized model.

Method

Participants

Children were serially recruited from patients coming to a pediatric emergency department (ED) because of asthma symptoms. Inclusion criteria were (a) patient age 7-17 years, (b) diagnosis of asthma, and (c) English speaking. Exclusion criteria were (a) co-morbid chronic medical conditions (other than allergy), and (b) living in residential facilities. This study is a part of a larger research project examining stress, depression and asthma. A detailed description of recruitment methods has been reported elsewhere (e.g., Lim et al., 2008). Briefly, contact was established with approximately 60% of the potential participant pool. Some potential participants were not contacted because the study schedule was full, their sibling had been in the study, or they could not be reached. Around 88% of contacted families were eligible. Sixty-one percent of those eligible enrolled. Retention rate from visit 1 to visit 3 was about 81%. Among the families who completed the larger study, only two parent families were selected for the current investigation. Demographic data collected during enrollment indicated no significant difference in age, gender, insurance status, or disease activity between those who completed the study versus those who were eligible and did not complete the study.

A total of 106 families participated. Children ranged in age from 7 to 17 ($M = 11.4$, $SD = 2.8$). Sixty-seven boys and 39 girls were included, with 50% African American, 32% European American, 10% Hispanics, and 8% other minority. About 36% were prescribed both controller (20% inhaled steroids, 16% other) plus rescue medication, and an additional 61% were prescribed rescue medication only. About 3% were prescribed no medication. Ninety-four percent of mothers were biological parents, and 6% were adoptive mothers, stepmothers, or grandmothers. Seventy-four percent of fathers were biological parents, 18% stepfathers, and 8% adoptive fathers or partners of the mother. The mean age of fathers and mothers was 42 years ($SD = 10.4$) and 39 years ($SD = 8.1$), respectively. Educational accomplishment of fathers and mothers (respectively) was: 14% and 18% less than high school, 48% and 33% high school graduate, 26% and 29% some college, 12% and 20% college graduate.

Measures

Demographics—Gender, age of child, ethnicity, family composition, parents' education, age of parents and insurance status were obtained by interview during enrollment.

Parental depressive symptoms—The Beck Depression Inventory II (Beck, Steer, & Brown, 1996) is a self-report instrument measuring depressive symptoms in adults with high reliability and established validity (Beck et al., 1996). It consists of 21 items rated 0 to 3 with higher scores reflecting greater depression. In this sample, internal consistencies were .87 for fathers and .90 for mothers.

Child depression—The Children's Depression Inventory (CDI; Kovacs, 1985) is a 27-item self-report of depressive symptoms with moderate to high internal consistency, test-retest reliability and established validity (Twenge & Nolen-Hoeksema, 2002). The items are rated 0 to 2 with higher scores indicating greater depression. The internal consistency for this study was .82. The Child Depression Rating Scale-Revised (CDRS-R; Poznanski & Mokros, 1995) is a 17-item clinician-guided interview measure of depressive symptoms. Each item is rated 1 to 5 or 1 to 7 with higher scores reflecting greater depression. The

CDRS-R has demonstrated good test-retest reliability, internal consistency and inter-rater reliability (Poznanski & Mokros, 1995). Questions were presented verbally to children and parents by the interviewer. Half of CDRS-R interviews were completed by MD/PhD level clinician/researchers skilled in the assessment of pediatric depression, and the remaining by a masters-level clinician, with the MD monitoring possible drift. The inter-rater reliability for this study was .91, and the internal consistency alpha coefficient was .81.

Child anxiety—The State-Trait Anxiety Inventory for Children (STAIC; Spielberger, 1973) is a 40-item self-report measure of anxious symptoms with established reliability and validity (Spielberger, 1973). The items are rated 1 to 3 with higher scores indicating greater anxiety. For this study the internal consistency of state anxiety was .79 and trait anxiety was .85.

Interparental behaviors—Interparental behaviors were observed during the laboratory-based Family Process Assessment Protocol (FPAP; Wood et al., 2008), and rated by trained raters using the Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). Behaviors rated as “hostile” were hostile, angry, critical, disapproving, and/or rejecting behavior toward another person, and “warm” as expressions of liking, appreciation, praise, care, affection, concern, or support. Each behavior rated has a specific code definition, with IFIRS ranging from 1 (no evidence of the behavior) to 9 (high frequency/intensity of the behavior). Interparental behaviors were computed as hostility scores minus warmth scores between parents (averaged across tasks) to reflect the combined effect of both negative and positive effects of behaviors between parents. Data analyses in our previous studies (e.g. Wood et al., 2008) indicate that the combination of negative and positive effects predicted outcomes better than either alone. This observation makes sense given that it is the overall impact of the combination of negativity and positivity in family relational process that has effect on individuals in the family. Higher scores indicate greater tendency toward negativity. A detailed description of training for raters and the scoring process for hostility and warmth have been reported elsewhere (Wood et al., 2008). Average intraclass correlation coefficients were .75 for hostility and .87 for warmth.

Parenting—Parenting behaviors of mothers and fathers during the FPAP were also rated using the IFIRS. Negative parenting behavior was indexed by an average of the rating scores for hostility, intrusiveness, and neglect/distancing, and positive parenting by an average of scores for warmth, child-centeredness and listener responsiveness. Each behavior rated has a specific code definition, with IFIRS ranging from 1 (no evidence of the behavior) to 9 (high frequency/intensity of the behavior). All scores were averaged across the tasks. Thus, the negative parenting or positive parenting scores were ranged from 1 to 9. The total score for parenting behavior was computed as the average negative minus the average positive parenting scores to reflect the overall effect of both negative and positive effects of parenting. Higher scores indicate greater tendency toward negative parenting. A detailed description of the training process has been reported elsewhere (Lim et al., 2008). Average intraclass correlation coefficients were .82 for positive parenting, .79 for negative parenting of mothers and .82 for positive parenting, .83 for negative parenting of fathers.

Asthma disease activity—The National Heart, Lung and Blood Institute Criteria for asthma disease activity (National Heart Lung and Blood Institute, 2007) was used to establish diagnosis and characterize level of disease. The disease activity rating is based upon configuration of objective measures of pulmonary function along with frequency of daytime and nighttime symptoms. Forced Expiration Volume in 1 second (FEV1) was used to evaluate pulmonary function. FEV1 was measured using a portable spirometer (Nelcor Puritan Spirometer “Renaissance” #220) that met the American Thoracic Society

performance standards. FEV1 scores were averaged across the three non-medicated research visits, approximately 1-week apart. A trained pulmonary function technician conducted the spirometric maneuvers. The frequencies of daytime and nighttime symptoms were rated by the research nurse or asthma specialist during the asthma clinical interview with the child and parent(s). The range of disease activity was 1 (mild intermittent) to 4 (severe persistent).

Medication adherence—Medications prescribed were determined by parent and patient report during the demographic survey, and convergent information was obtained during the asthma clinical evaluation by physician interview of parent and patient and by chart evaluation. Adherence to medication as prescribed was assessed by a telephone interview (Bender, Rankin, Tran, & Wamboldt, 2008), conducted before each of 3 research visits, to collect information on medication taken (i.e., medication name, dose, time) during the 24 hours preceding the call. Both controller medications and rescue medications were monitored according to how consistently they were used as prescribed. Adherence was rated by two independent raters (the research nurse and a PhD-level clinician) using a four-point scale (1-very poor, 2-fair, 3-good, 4-very good), and then consensed if there was a discrepancy. The inter-rater reliability before consensing was .95.

Procedures

The study was approved by the CYIRB of the Women and Children's Hospital of Buffalo. Consent (parents) and assent (children) were obtained, and a demographic interview was conducted. Demographic and adherence data were obtained by telephone interview at enrollment and before each visit. As part of a larger study on stress and asthma, there were three research visits, the first occurring about 4 weeks after the ED visit (to allow asthma symptoms to stabilize), and the subsequent two occurring at about 1 week intervals. At research visit 1 and 2, a clinical asthma evaluation, including pulmonary function tests, was performed by the research nurse and the asthma specialist at the Lung Center and the Asthma Clinic. At research visit 3, the pulmonary function tests were performed again and self-report questionnaires and a clinical depression interview were conducted. Next, the child and family gathered in a living room styled laboratory setting to participate in the family interaction protocol. Interparental behaviors and parenting behaviors of mothers and fathers were observed during this laboratory-based Family Process Assessment Protocol (FPAP; Wood et al., 2008), a structured set of interaction tasks. Four of the most emotionally evocative of the six 5-minute FPAP tasks were used: loss, parent-child conflict, parent-parent conflict, and cohesion. We selected these tasks because they elicit a broad range of emotionally evoked interactions thus enhancing the ecological validity and generalizability of the family interaction ratings. Following the protocol there was a debriefing and payment for participation.

Data Analyses

Pearson correlation analyses were used to test bivariate associations among the study variables. Structural Equation Modeling (SEM) was used to test the direct and indirect pathways posited. Square root transformations were used to achieve approximate normality as needed for those analyses. SEM analyses were conducted with AMOS 7.0 using maximum likelihood estimation. All SEM models were examined for model fit using widely used statistical indices: chi-squares, IFI, CFI and RMSEA. There were less than 3% missing data in three study variables (i.e. maternal depressive symptoms, expert-rate child depressive symptoms, medication adherence), and 9% missing data in the observation variables. The patterns of parameter estimates and fit measures were similar with or without cases with missing data.

Considering the potential moderating role of child gender in models of depression and family process (Davies & Lindsey, 2001; Davies & Windle, 1997), preliminary SEM analyses were conducted to determine whether child gender moderated the hypothesized pathways in the model using multi-group analysis (Hong, Malik, & Lee, 2003). The hypothesized model was not significantly different according to gender. Thus, we performed subsequent analyses using the full sample. The sample size was not sufficient to test moderating effects of child race, and there were no associations between age and the study variables, so age was not tested as a moderator.

A model building approach is used to test the hypothesized model, which allows testing essential pathways (e.g. child internalizing symptoms to asthma disease activity) first and then including further family process mediating pathways. First, a baseline model was estimated in which only the direct paths from parental depressive symptoms to child internalizing symptoms and the path from child internalizing symptoms to asthma disease activity were included, while all other path coefficients were assumed to be zero. Second, an initial mediating model was estimated in which the indirect paths from parental depressive symptoms to child internalizing symptoms through interparental negativity and parenting behaviors were added to the baseline model. In the last step, the hypothesized model was estimated in which the indirect paths from interparental negativity to child internalizing symptoms through parenting were added to test the “spill-over” effect of interparental negativity on parenting behaviors.

Medication adherence was correlated with all predictors and with asthma disease activity. It is already well established in the literature that child and family dysfunction are associated with poor adherence in asthma. The purpose of our research program is to examine hypothesized direct family-psycho-biologic links. Therefore, in our study medication adherence was treated as a control variable.

Results

Descriptive Statistics and Correlations among Study Variables

Descriptive statistics and bivariate correlations of the study variables are presented in Table 1. About 11% of fathers and 20% of mothers showed depressive symptoms above the clinical cutoff for moderate to severe depression (scores > 19), 11% of fathers and 9% of mothers evidenced mild depressive symptoms (scores of 14 to 19), and 77% of fathers and 71% of mothers showed minimal depressive symptoms (scores of 0 to 13). About 16% of the children scored above a clinical cutoff (score of 13) on the CDI, and 10% scored in the clinical range of the CDRS-R (score of 40 or above). Among family process variables, about 15% of mothers and 20% of fathers showed more negative behaviors compared to positive behaviors in parenting. About 38% of parents showed more negative behaviors compared to positive behaviors in interparental behaviors. For disease activity, participants were characterized as: 15% mild intermittent, 17% mild persistent, 38% moderate persistent and 30% severe persistent. The distribution of medication adherence was as follows: 19% very poor, 13% fair, 24% good and 44% very good.

Testing the Hypothesized Model

First, the measurement model for child internalizing symptoms was examined. The measurement model fit the data well, $\chi^2(2, N=106) = 2.01, p = .37$, IFI = 1.0, CFI = 1.0, RMSEA = .007, with the high fit indices partially a function of the model being near saturation. Factor loadings for each manifest indicator of the latent construct were all positive and significant at $p < .001$ (standardized loadings were .37 to .93, weak at the low

end and excellent at the high end). Residual error variances of indicators were decided, a priori, to be allowed to correlate for depression or for anxiety in the model.

The following three steps were used to test which pattern of direct and indirect paths best represented the data. Although not represented in the figure to simplify presentation, medication adherence was included as a control variable in all models. Table 2 shows the path added and tested for each of the model-building steps. As shown in that table, the significance test of each step beyond the baseline model is based on the change in chi-square model fit statistics.

In the first step, a baseline model was estimated in which the measurement model, the direct paths from parental depressive symptoms to child internalizing symptoms and the path from child internalizing symptoms to asthma disease activity were included, but all other path coefficients were assumed to be zero. This model was used as a basis on which to compare the fit of the nested model to be tested in the second step. The baseline model, $\chi^2(39, N=106) = 99.64, p < .001, IFI = .73, CFI = .70, RMSEA = .12$, showed poor fit to the data.

In the second step, the initial mediating model was estimated. In the model, the paths from parental depressive symptoms to interparental negativity and parenting behaviors of both parents were added to the baseline model, along with the paths from interparental negativity and parenting to child internalizing symptoms, to test the indirect effect of parental depressive symptoms on child internalizing symptoms via interparental negativity and parenting. This model, $\chi^2(32, N=106) = 57.50, p = .004, IFI = .89, CFI = .87, RMSEA = .09$, showed that the indirect path from maternal depressive symptoms to child internalizing symptoms via mothers' parenting behaviors was significant, and in turn, child internalizing symptoms predicted asthma disease activity ($\beta = .28, p < .05$). Paternal depressive symptoms were linked to fathers' parenting ($\beta = .29, p < .001$) as well as to interparental negativity ($\beta = .32, p < .01$), but they were not linked to child internalizing symptoms either directly or indirectly through interparental negativity or his parenting. This model fit the data significantly better than the baseline model did, $\Delta\chi^2(7, N=106) = 42.14, p < .0001$.

In the last step, the hypothesized model was estimated including the paths from interparental negativity to parenting behaviors of both parents to test the spillover effect of interparental negativity on parenting (See Figure 2). The model provided a strong fit to the data, $\chi^2(30, N=106) = 36.18, p = .20, IFI = .97, CFI = .97, RMSEA = .04$, and resulted in a significant improvement in fit, $\Delta\chi^2(2, N=106) = 21.32, p < .0001$, compared with the previous model. The hypothesized model revealed that interparental negativity was linked to father's negative parenting ($\beta = .41, p < .001$) as well as mother's negative parenting ($\beta = .38, p < .001$), supporting the spillover hypothesis.

The hypothesized model results also were consistent with the following mediated processes: Maternal depressive symptoms increased the probability of negative parenting behaviors ($\beta = .27, p < .01$) which lead to more child internalizing symptoms ($\beta = .32, p < .05$) which, in turn, predicted asthma disease activity ($\beta = .28, p < .05$). Paternal depressive symptoms had a similar but weaker effect ($\beta = .20, p < .05$), with the link between negative parenting and child internalizing symptoms not reaching statistical significance ($\beta = .19, ns$). Paternal depressive symptoms were, however, linked to interparental negativity ($\beta = .32, p < .01$) (maternal depressive symptoms were not) which predicted child internalizing through negative parenting of mother ($\beta = .38, p < .001$ and $\beta = .32, p < .05$ respectively). Said another way, maternal negative parenting predicted child internalizing and subsequent asthma disease activity, however maternal negative parenting was predicted by both maternal depressive symptoms and interparental negativity, and the latter was primarily affected by paternal depressive symptoms. Finally, all these effects cascaded to a significant

indirect effect of maternal negative parenting on child asthma disease activity through child internalizing symptoms, which is further supported with the Sobel test ($z' = 1.45, p < .05$). Direct effects of parental depressive symptoms and interparental negativity on child internalizing symptoms were not observed.

Although the hypothesized model was found to have a strong fit to the data, it is possible that the inter-relations of family relational process variables and asthma disease activity could also be described by different models. Thus, a more inclusive model was estimated to examine the plausibility for pathways for the observed relations in this study. The direct paths from family relational process variables (i.e. parenting and interparental negativity) to child asthma disease activity were added to the hypothesized model and tested. The paths from interparental negativity and negative parenting of mother to asthma disease activity were not significant, but the path from negative parenting of father to asthma disease activity was significant ($\beta = .27, p < .05$), and the model resulted in a significant improvement in fit, $\Delta\chi^2(3)=11.72, p < .01$, compared with the hypothesized model.

Discussion

This study investigated the impact of parental depressive symptoms on psychological and physical functioning in children with asthma, focusing on family process as a potential mediator. Based on the BBFM and previous results, we examined specific multiple dimensions of family relational process within one comprehensive model, allowing for a simultaneous test of the unique contributions of fathers and mothers to child dysfunction. Results supported the role of family process as an intervening mechanism linking parental depressive symptoms to child dysfunction. These findings are consistent with the BBFM (Wood et al., 2006) which proposes that stressful family relational process may evoke emotion dysregulation (e.g., internalizing symptoms) in the child, accompanied by physiological dysregulation, which in certain stress-sensitive diseases such as asthma potentiates disease activity. Specifically, negative family emotional climate predicted child internalizing symptoms, which in turn predicted asthma disease activity. The direct psychobiologic mechanisms transmitting the effect of internalizing problems were not tested in this study, but have received support from another study in our lab (Miller et al., 2009). Moreover, the current findings extend the BBFM by finding evidence that parental depressive symptoms are direct contributors to interparental negativity and negative parenting, and indirect contributors to child psychological and physical dysfunction. It is noteworthy that although adherence was associated with disease activity in the bivariate analysis, when adherence was controlled for in the model, the relational stress pathways predicting asthma remained significant, consistent with current literature demonstrating direct psychobiological effects of stress and depression on asthma (Chen & Miller, 2007; Miller et al., 2009). The findings indicate the importance of addressing family relational stress and child internalizing symptoms when treating children with asthma.

Findings are also consistent with other studies showing family relational process as mediating the effect of parental depressive symptoms on child psychological functioning (Davila, Stroud, & Starr, 2009; Hammen, 2009), including marital conflict (Cummings & Davies, 2010) and negative parenting (Hammen, 2009; Lovejoy et al., 2000). This suggests the importance of addressing both interparental negativity as well as negative parenting when intervening with families of children with asthma.

Results of the current study suggest different pathways of effect for mothers versus fathers. Maternal depressive symptoms appeared to affect child internalizing symptoms by contributing to mother's negative parenting behaviors, whereas paternal depressive symptoms appeared to contribute to greater interparental negativity, which contributed to

maternal negative parenting, which in turn, contributed to child internalizing symptoms. Mothers' negative parenting, but not fathers', was directly linked to child internalizing symptoms, which in turn were associated with child asthma disease activity. The discrepancy in effect of maternal vs. paternal negative parenting is consistent with previous meta-analytic findings (Connell & Goodman, 2002). One possible explanation is that school-aged children tend to spend more time with their mothers than their fathers, with mothers taking the primary care-taking role, and thus children are more susceptible to maternal negative parenting (Parke, 2002; Pleck, 1997). A second possibility is that the aspects of parenting we measured (hostility, intrusiveness, and neglect/distancing versus warmth, child-centeredness and listener responsiveness) may not be the father-child parenting factors which most importantly influence child internalizing symptoms.

Paternal, but not maternal, depressive symptoms were significantly linked to interparental negativity, suggesting that interparental negativity may be more strongly impacted by paternal depressive symptoms compared to maternal depressive symptoms. This finding is convergent with others' observations that paternal depression is related to greater angry and hostile communication which may provoke interparental conflict, whereas depressed mothers are typically tired and withdrawn (Du Rocher Schudlich, Papp, & Cummings, 2004). Therefore, fathers' depressive symptoms may play a more prominent role in shaping overt interparental negativity. In any case, these findings, taken together, support the importance of including fathers in family interventions and of resisting the often more convenient approach of intervening solely with the mother and child.

Results of this study support the spillover hypothesis (Erel & Burman, 1995). Interparental negativity was consistently associated with negative parenting. No direct link of interparental negativity to child internalizing symptoms was found, which is consistent with other findings (Du Rocher Schudlich & Cummings, 2003; Franck & Buehler, 2007; Kaczynski et al., 2006). However other studies do show child emotion dysregulation and physiological stress responses to enactment of marital conflict in laboratory situations which have implications for future child psychological and physical compromise (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; El-Sheikh, 2005). This is particularly evident in cases of marital violence (El-Sheikh, Cummings, Kouros, Elmore-Staton, & Buckhalt, 2008). Therefore interparental negativity should not be ruled out as a potentially important direct influence on child function through pathways of chronic stress/arousal due to exposure to interparental conflict. Taken together, these findings indicate that family-informed interventions should seek to lower any potential effect interparental negativity on the child.

It is also noteworthy that the spillover from interparental negativity to mother's parenting behaviors served as an intervening mechanism linking paternal depressive symptoms to child internalizing symptoms. This suggests an indirect, but important, contribution of fathers to child psychological and physiological disorder. Fathers thus should not be overlooked as an important source of emotional impact on children, indirect as well as direct. Given the paucity of empirical data, clinicians should be open to discovering aspects of paternal effects, both positive and negative, on child psychological and physical well being.

It is of note that the finding of no direct effect of maternal depressive symptoms on child internalizing symptoms is discrepant from our previous study focusing on maternal depression in relation to child internalizing symptoms and asthma. In the former study (Lim et al., 2008) we found direct as well as indirect effects (through parenting). However in the former study 62% of the sample was single maternal parent families. It is possible that when there is only one depressed parent there may be a stronger direct effect on the child through

the child's worrying about the parent, through insecurity of attachment, and other possible mechanisms which are not buffered by the presence of a second parent.

In exploratory analysis, a direct pathway from negative parenting of father to child asthma disease activity was found, which implies that the negative parenting of father may affect child asthma disease activity through other as yet unknown mediators, rather than through child internalizing symptoms. This analysis was post hoc, and not predicted by the BBFM, so the findings must be interpreted with caution, particularly since adding new pathways moved the model further towards saturation, and thus potentially spurious findings. Nonetheless these exploratory findings point to additional paternal and maternal differentiation of effects on child well being. They bear future replication, along with examination of potential mechanisms to explain these pathways. Taken together, the current investigation and the exploratory findings underscore the importance of stress management in families of children with asthma who are challenged by interparental negativity and parenting compromise. Findings from our studies, including the one reported herein, strongly suggest the need for family based intervention in treating troubled children with asthma.

Strengths and Limitations

To our knowledge there are no other studies examining the impact of both paternal and maternal depressive symptoms on child psychological and physical function through interparental negativity and negative parenting in a comprehensive multi-dimensional model including both psychological and physiological disease outcomes. Our study also examined unique pathways for fathers and mothers. Furthermore, our study focused on the emotional tone of interparental negativity, rather than on intensity, frequency or resolution. This is to date an understudied aspect of interparental relational process. Of additional importance is fact that the variable “interparental negativity” was operationalized as a compiled hostility and warmth score, which takes into account the balance of hostility and warmth. We believe this conceptualization has more ecological validity than separate single concepts of hostility and warmth because family patterns of interaction are typically mixes of negative and positive interaction, and it is the overall effect of the balance of one over the other that ultimately determines the emotional climate of a family and its effects on the individual family members.

Several limitations of this study must be acknowledged. The sample was recruited from an inner-city emergency department, so our findings may not be generalizable beyond high risk populations. The findings of this study are based on multi-informant, multimethod data collection, but the occurrence of observed behaviors during laboratory-based family tasks may not capture the complete set of effects of interparental and parenting behaviors. The design of the current study can neither prove causal direction nor rule out alternative models, including a model in which child psychological and physical dysfunction contribute to parental depressive symptoms and negative family process. It is most likely that the directions of effect are bi-directional. Longitudinal or intervention designs are indicated to examine directions of causal influence. The sample size was insufficient to examine multiple demographic moderating effects such as ethnicity and SES. Thus the findings require replication with a larger sample.

Despite the limitations in this study, we conclude that our heuristic model may serve as a prototype for integrative theory-building that includes parent risk factor-family process-mind-body interactions in a unified model. Furthermore, the results of this study contribute to our understanding of the unique pathways by which paternal and maternal depressive symptoms are linked to child psychological and physical dysfunction through negativity in family process.

Acknowledgments

This study was supported by grants from the National Institute of Mental Health provided to Dr. Miller (5R01MH058761) and Dr. Wood (5R01MH064154). Preparation of this manuscript was also supported in part by a grant from College of Education, Korea University to Dr. Lim.

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Figure 1.
Hypothesized model of the pathways of effect of paternal and maternal depressive symptoms on child internalizing symptoms and asthma disease activity.

Figure 2.

Structural equation model showing the hypothesized direct and indirect pathways from parental depressive symptoms on child internalizing symptoms and asthma disease activity. $\chi^2(30, N=106) = 36.18, p = .20, IFI = .97, CFI = .97, RMSEA = .04$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1
Descriptive Statistics and Bivariate Correlations among the Study Variables

Variable	M (SD)	1	2	3	4	5	6	7	8	9	10
1. BDI_M	11.03 (8.79)										
2. BDI_F	9.02 (6.94)	.35***									
3. NPM	-.80 (.96)	.31**	.11								
4. NPF	-.60 (.97)	.18+	.25*	.53***							
5. Interparental Negativity	-.05 (1.02)	.19+	.30**	.42***	.46***						
6. CDI	6.83 (5.52)	.06	.17+	.24*	.24*	.08					
7. CDRS	28.08 (7.73)	.18	.23*	.23*	.19+	.06	.62***				
8. STAI_C_T	28.12 (4.71)	-.02	.03	.06	.05	.04	.38***	.24*			
9. STAI_C_S	34.29 (7.18)	.04	.15	.23*	.23*	.14	.56***	.42***	.15		
10. Disease activity	2.83 (1.03)	.23*	.12	.33**	.35***	.19+	.14	.002	.01	.25*	
11. Medication adherence	2.92 (1.16)	-.03	-.10	-.14	-.01	-.11	-.003	.14	-.01	-.09	-.26**

Note. BDI_M (maternal depressive symptoms), BDI_F (paternal depressive symptoms), NPM (negative parenting of mother), NPF (negative parenting of father), CDI (child-report depression), CDRS (expert-rate child depression), STAI_C_T (child-report trait anxiety), STAI_C_S (child-report state anxiety).

*
 $p < .05$.

**
 $p < .01$.

 $p < .001$.

Table 2
Evaluation of Structural Equation Models from the Baseline to the Hypothesized Model

Model	Path to be added	Path coefficient	χ^2	df	$\Delta\chi^2$
Baseline	Paternal depressive symptoms to child internalizing symptoms	.21	99.64	39	
	Maternal depressive symptoms to child internalizing symptoms	.12			
	Child internalizing symptoms to asthma disease activity	.21			
Initial Mediating Model	Paternal depressive symptoms to father's parenting	.29***	57.49	32	29.75***
	Father's parenting to child internalizing symptoms	.19			
	Maternal depressive symptoms to mother's parenting	.31***			
	Mother's parenting to child internalizing symptoms	.32*			
	Paternal depressive symptoms to interparental negativity	.32**			
	Maternal depressive symptoms to interparental negativity	.07			
	Interparental negativity to child internalizing symptoms	-.11			
Hypothesized Model	Interparental negativity to father's parenting	.41***	36.18	30	21.32***
	Interparental negativity to mother's parenting	.38*			

*
 $p < .05$.

**
 $p < .01$.

 $p < .001$.