Family Emotional Climate, Depression, Emotional Triggering of Asthma, and Disease Severity in Pediatric Asthma: Examination of Pathways of Effect

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Objectives  (a) To assess emotional triggering of pediatric asthma and ascertain its contribution to disease morbidity and functional status; (b) to test whether negative family emotional climate (NFEQ) is associated with depressive and/or anxious symptoms and emotional triggering of asthma attacks in the child.  Method  Children with asthma (N = 272, 56% male, age 7–17) and their primary caregivers answered together an Asthma Trigger Inventory (Ritz, Steptoe, Bobb, Harris, & Edwards, 2006). Children reported on anxious (STAIC) and depressive (CDI) symptoms and on asthma-related quality of life (PAQLQ). Parent(s) reported on their child’s internalizing (CBCL-I) and depressive symptoms (CDI-P). A clinician also rated the child’s depression using the structured CDRS-R. Asthma diagnosis was confirmed and disease severity rated according to NHLBI guidelines by an asthma clinician.  Results  Path analyses indicated that NFEQ was associated with depressive symptoms, which in turn were associated both directly and indirectly (by way of emotional triggering) with disease severity. Comparison of nested models indicated the possibility of differential roles and pathways for anxious versus depressive symptoms.  Conclusion  Findings elucidate possible pathways of effect by which family emotional climate and child depressive symptoms may influence pediatric asthma disease severity by way of potentiating emotional triggering of asthma.

Key words  anxiety; depression; family; pediatric asthma; stress.

Asthma is a complex clinical syndrome characterized by variable airflow obstruction, bronchial hyperresponsiveness (BHR), airway edema, and eosinophilic and lymphocytic inflammation. Triggers include allergens, infections, exercise, cold air, and emotions. Mechanisms of airway constriction include both immune/inflammatory pathways and cholinergic/vagal pathways (Middleton, Jr. et al., 1998). Asthma is one of the most common chronic diseases of childhood, affecting 4.8 million children in the United States (Adams & Marano, 1995). Among children with chronic medical conditions, asthma is the most common cause for hospitalization and school absence (Newacheck & Taylor, 1992). Asthma morbidity and mortality are increasing, despite new effective medical regimens (Weitzman, Gottmaker, Sobel, & Perrin, 1992).

Recent research has focused on both poor treatment adherence (Fiese & Wamboldt, 2001; McQuaid, Kopel, Klein, & Fritz, 2003) and stress and emotions (Miller & Wood, 2003; Wright, Rodriguez, & Cohen, 1998) as factors affecting asthma morbidity and mortality. Depressive emotional states have been particularly implicated (McQuaid, Kopel, & Nassau, 2001; Waxmonsky et al., 2006), although a few studies have also identified anxiety as a factor (Lehrer, Feldman, Giardino, Song, & Schmaling, 2002). It is possible that the effects of emotional distress on asthma are mediated in part by adherence, but psychobiologic pathways and mechanisms linking stress and emotions with disease activity have also been posited (Miller, 1987; Wright, Cohen, & Cohen, 2005). Research explicating such pathways has been limited by lack of theoretical models.
and appropriate methodology. Miller’s ANS–HPA–Immune Dysregulation Model of the influence of Emotions on Asthma addresses this limitation (Miller & Wood, 2003). This model proposes that emotionally dysregulated states of hopelessness/depression are accompanied by patterns of autonomic nervous system (ANS), hypothalamic, pituitary, adrenal (HPA) and immune/inflammatory dysregulation that promotes airway instability and compromise in asthma (Miller & Wood, 1994, 1997). Although the model proposes physiological processes by which emotionally dysregulated states may potentiate the effect of any trigger (allergen, infection, exercise, or stress), it is also hypothesized that being in an emotionally dysregulated state may directly affect emotional triggering of airway compromise.

Despite the long-standing clinical and empirical literature reporting emotional triggering of asthma (Lehrer, 1998), we found only one investigator specifically studying self-report of emotional triggering of asthma in daily life (Purcell, 1963, 1973). We found no systematic studies of emotional triggering of asthma in daily life for children. This research has likely been impeded by lack of reliable and valid instruments for assessing emotional triggering, and by lack of systematic theory as to possible pathways and mechanisms by which emotions could trigger asthma.

One possible pathway is by way of family relational dysfunction (Minuchin et al., 1975; Mrazek, Klinnert, Mrazek, & Macey, 1991; Purcell et al., 1969). In a review, Kaugars reported substantial evidence for family factors in asthma outcomes, positing both adherence and direct psychobiological pathways as mechanisms (Kaugars, Klinnert, & Bender, 2004). Previous findings in our laboratory have demonstrated that negative emotional climate in the family was related to self reported depressive symptoms in asthmatic children which, in turn, were associated with disease severity even after controlling for adherence (Wood et al., 2006b). Security of parent–child relationship appeared to partially mediate this effect. Although this study substantiated that particular family relational factors were implicated in asthma disease activity, the study did not assess whether the emotional distress engendered by the negative family emotional climate (NFEQ) was directly associated with emotional triggering of the child’s asthma.

Therefore, based on the literature and our previous findings, our study tested a conceptual model of pathways by which NFEQ would affect child depressive symptoms, anxiety, and emotional triggering, which would, in turn, predict asthma disease severity. Our previous findings (Wood et al., 2006b), and the models on which they are based (Miller & Wood, 2003; Wood, Klebba, & Miller, 2000), propose multiple pathways by which stress and emotions may influence child asthma. Specifically, we hypothesized direct and indirect pathways of association between NFEQ and emotional triggering of asthma. We posited that the indirect pathways would be by way of child anxious and depressive symptoms. We also hypothesized both direct and indirect pathways of association between depressive and anxious symptoms and disease severity, indirect by way of emotional triggering of asthma. (See Fig. 1 for conceptual model.)

The aim of this study was: (a) to assess emotional triggering and ascertain its contribution to disease morbidity and functional status by assessing associations between report of emotional triggering and asthma disease severity and quality of life; (b) to test whether stressful family emotional climate is associated with depressive and/or anxious symptoms and emotional triggering of asthma attacks in the child.

**Method**

**Subjects**

Children and their primary caregiver ($N = 272$, 56% male, age 7–17) were serially recruited from patients coming to a pediatric Emergency Department (ED) between the dates of June 2001 to March 2005 because of asthma symptoms. Inclusion criteria: (a) patient age of 7–17 years; (b) diagnosis of asthma; (c) at least one adult caregiver. Exclusion criteria: (a) patients actively treated for comorbid chronic medical conditions (allergies were allowed); (b) patients living in residential facilities.

The recruiter attempted face-to-face or telephone contact with all parents of patients who came to the ED because of asthma symptoms. Guardian consent was obtained and a demographic interview conducted. Of the
patients and families who were contacted, 59% agreed to be assessed for enrollment (n = 384) in the study. Of those who did not enroll, 32% were not eligible (n = 89) and 68% refused (n = 188). Seventy-one percent of the enrolled patients (n = 272) completed the study. The contacted versus enrolled and completed subjects were highly similar with respect to age (M = 11.4, SD = 2.9 vs. M = 11.5, SD = 2.9), gender (58% vs. 56% male), race (52% vs. 60% African American, 30% vs. 21% Caucasian, 18% vs. 19% others), and Medicaid status (18% vs. 18% Medicaid) (See Table I for demographics).

**Measures**

The Asthma Trigger Inventory (ATI) (Ritz, Steptoe, Bobb, Harris, & Edwards, 2006) is a self-report measure for asthmatic adults, consisting of 32 items including various triggers. Patients rate on a 5-point scale (0 = never, 1 = rarely, 2 = sometimes, 3 = most of the time, 4 = always) how often the particular trigger was related to their asthma symptoms. This instrument yields scores on six trigger subscales: emotion/psychologic (e.g., angry, lonely, stress at home, tense, depressed, arguments, worried, and unhappy); animal allergens; pollen allergens; exercise; air pollution/irritants; and infections. Support for the reliability and validity of this instrument in a pediatric population is presented in a separate report (Wood et al., 2006a). The emotions trigger subscale score was used to index emotional/psychological triggering. The α coefficient for this scale in this study was .89.

The State–Trait Anxiety Inventory for Children (STAIC) is a 40-item child self-report anxiety measure with established reliability and validity (Spielberger, 1973). We used the trait subscale score to index anxiety (α coefficient is .78 for males and .81 for females) (Walker & Kaufman, 1984). The α coefficient for this study was .86.

The Children’s Depression Inventory (CDI) (Kovacs, 1985) is a child self-report of depressive symptoms with moderate to high internal consistency, test-retest reliability and validity (Twenge & Nolen-Hoeksema, 2002). The α coefficient for this study was .86.

The Children’s Depression Inventory-Parent (CDI-P) (Garber, 1984) was used to assess the convergence of parent report with child report of depression. The CDI-P is derived directly from the CDI and is scored identically. It has high test-retest reliability (.75), internal consistency (.74), and content validity, and is well correlated with the CDI (Wierzbicki, 1987). In this sample, the α coefficient was .88.

The Children’s Depression Rating Scale-Revised (CDRS-R) (Poznanski & Mokros, 1995), which is a clinician-guided measure of depressive symptoms, served as another convergence check for the CDI. The CDRS-R has demonstrated strong test–retest reliability (.80), internal consistency (α coefficient = .85) and inter-rater
reliability (.86) (Poznanski et al., 1984). Questions were presented verbally to children and parents by the interviewer. Children under the age of 12 were interviewed with their parent, and adolescents were interviewed separately to maximize the likelihood of accurate self-reporting. When symptom reports were discrepant between reporters, the clinician used both scores to compute a final composite score, with greater weight given to the reporter deemed by the clinician to be more accurate. Each item is rated 1–5 or 1–7, with 1 designating no difficulty with that symptom. The scale yields a total score of 17–113, with a score of 40 considered to be a strong indicator of an impairing depressive state. Half of CDRS-R interviews were completed by MD/PhD level clinician/researchers (J.W. and D.C.B.) skilled in the assessment of pediatric depression, and the remaining by a masters-level clinician. Inter-rater reliability (correlation between rater total scores) for this study was (.89), which is consistent with published values (.86). The \( \alpha \) coefficient (within-subject internal consistency of the interview items) for this study was .84.

The Child Behavior Checklist (CBCL) Behavior Problems Scale (Achenbach, 1991) is a 118-item questionnaire, completed by parents, which is used to assess behavioral functioning differentiated into individual internalizing and externalizing subscales. This scale has extensive reliability and validity data (Sattler, 2001). We used the Internalizing scale in this study to further examine relations between anxiety and depressive symptoms and the other variables. The \( \alpha \) coefficient of the Internalizing scale for this study was .84.

The Pediatric Asthma Quality of Life Questionnaire (PAQLQ) (Juniper, Guyatt, Willan, & Griffith, 1994) is a 23-item questionnaire that measures the physical, emotional, and social impairments experienced by children (7–17 years old) with asthma. Sample items are: How much did coughing bother you in the past week? How often did you feel you couldn’t keep up with others because of your asthma? The PAQLQ has been widely tested and is a reliable and valid instrument. The \( \alpha \) coefficient for this study was .92.

The Family Expressiveness Questionnaire (FEQ) (Halberstadt, 1986) characterizes negative and positive emotional expressiveness in a family and was used to operationalize “negative emotional climate.” The questionnaire consists of 40 written scenarios involving emotional expression. Mothers rated on a scale of 1 (not at all frequently) to 9 (very frequently) to indicate how often each scenario occurs in their families. Sample items are: Expressing anger at someone else’s carelessness; trying to cheer up someone who is sad. Reliability and validity have been established on a wide range of families (Halberstadt, Cassidy, Stifter, Parke, & Fox, 1993). The \( \alpha \) coefficient for FEQ in this study was .90. Following the method in our previous studies, we subtracted the positive from the negative emotion subscale scores in order to obtain an index of emotional balance in the family. Larger scores, therefore, indicate an imbalance towards NFEQ (Wood et al., 2006b).

The National Heart, Lung and Blood Institute (NHLBI) Criteria for asthma disease severity (NHLBI, 2003) were used to establish diagnosis and characterize level of disease. The disease severity rating is based upon configuration of objective measures of pulmonary function (FEV1) (average of three measures, ∼1 week apart, with medication withheld) along with frequency of daytime and nighttime symptoms. Disease severity was determined by the research nurse T.S. and asthma specialist M.B. with independent assignment (blind) by a masters-level health clinician (81% perfect agreement; 91% agreement within one step, \( \kappa = .75 \)). All discrepancies were reconciled by reviewing additional clinical material from charts, and consensus scores were used in analyses.

**Procedure**

The study was approved by the IRB of the children’s hospital. Guardian consent and child assent were obtained. Children and families were compensated $150 for their time and effort. After enrollment, children received an asthma diagnostic evaluation (visits 1 and 2) free of charge and, with consent, results were sent to the child’s physician. The protocol involved three hospital visits, the first one between 4 and 6 weeks after the ED admission, to allow for stabilization of the asthma exacerbation. The subsequent two visits occurred at about 1 week intervals (variation 1–2 weeks).

Visit 1: Clinical asthma evaluation (Lung Center and Asthma Clinic, 3–4 h). Pulmonary function tests (FEV1) were performed by the research nurse. Next, the research nurse administered the ATI to the child or adolescent and parent together. If the child and parent disagreed on an answer, the interviewer used her best nursing judgment to choose which answer was likely the most accurate. The disagreement rate was <2%. A clinical asthma evaluation, including physical exam, allergy testing, history of disease and current symptoms, and medication history (parent and child interviewed together),
was conducted by the nurse, T.S., under supervision of our co-investigator asthma specialist, M.B.

Visit 2: *Pulmonary function testing* (Lung Center, 1–2 h). Spirometry (FEV1) was conducted a second time to assess airway function.

Visit 3: *Pulmonary function and psychological testing* (Child and Family Asthma Study Laboratory, 3 h). Spirometry (FEV1) was repeated a third time to assess airway function. Then, apart from one another in order to maintain confidentiality, parents answered the CDI-P, CBCL-I, and PAQLQ. A clinical CDRS-R depression interview was conducted. In the afternoon, the children and families participated in a family interaction protocol. Findings from this aspect of the larger study will be reported elsewhere.

Data Analyses
Descriptive statistics characterized the sample. Pearson and Spearman correlation analyses tested associations. Path analyses tested the direct and indirect pathways posited. Using the maximum-likelihood program, AMOS, path coefficients were estimated simultaneously, and an estimate of the overall fit of the model to the data was generated. Maximum likelihood estimation procedures were used, and standardized parameter estimates are presented. The goodness of fit of the models was examined by using the normed fit index (NFI) and the root mean square error of approximation (RMSEA). The NFI varies between 0 and 1.0, and a value of 1.0 indicates perfect fit, with values close to .95 indicating a good fit. The RMSEA is bounded by zero and will take on that value when a model exactly reproduces a set of observed data. Values of < .05 indicate a good fit. The chi-square difference test was used to compare the fit of successive nested models.

The conceptual model (Fig. 1) was tested in three steps using nested models to assess whether anxiety and depression predicted disease severity differentially. In the first step, a Baseline Model was estimated in which all the paths from both depression and anxiety to ATI emotional triggers and disease severity were constrained to be zero but all other paths were included. This allowed a basis on which to compare the improved fit of the model to the data by successively including depression and anxiety. In step 2, a depression-only model was estimated where paths from anxiety to ATI emotional triggers and disease severity were constrained to be zero. In the final step, the hypothesized model, including both anxiety and depression pathways, was estimated and compared with the Baseline Model and the Depression Model.

Results
Child Anxiety, Depression, and Emotional Triggering of Asthma
Self-report of anxious symptoms (STAIC-T) and of depressed symptoms (CDI) were correlated with the ATI emotional trigger score \(r = .20, p < .01; \ r = .30, p < .001\), respectively. The associations of anxiety and depressive symptoms with emotional triggering of asthma were corroborated by correlations between the following: ATI emotional trigger scores and parent report of internalizing problems (CBCL-I; \(r = .25, p < .001\)); parent report of child depressive symptoms (CDI-P; \(r = .35, p < .001\); and clinician-based rating of depressive symptoms (CDRS-R; \(r = .42, p < .001\)). As hypothesized, the ATI emotional trigger score was correlated with both asthma disease severity \(r = .23, p < .001\) and with asthma-related quality of life (PAQLQ; \(r = -.41, p < .001\)). NFEQ was correlated with child depressive symptoms (CDI; \(r = .18, p < .01\)). This relationship was corroborated by correlations between NFEQ and parent report of child depressive symptoms (CDI-P; \(r = .14, p < .05\), and a trend toward association between NFEQ and clinician rating of depression (CDRS-R; \(r = .15, p = .06\)). NFEQ was not related to child anxiety (STAIC-T) nor to disease severity or pediatric asthma-related quality of life (PAQLQ) (See Table II for descriptive statistics).

<table>
<thead>
<tr>
<th>Table II. Variable Mean and Standard Deviation</th>
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<tbody>
<tr>
<td>Variable</td>
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<tr>
<td>ATI emotions subscale</td>
</tr>
<tr>
<td>State–Trait Anxiety Inventory for Children</td>
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<tr>
<td>Child Depression</td>
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<tr>
<td>CBCL internalizing problems</td>
</tr>
<tr>
<td>CDI parent about patient</td>
</tr>
<tr>
<td>Children’s Depression Rating Scale-Revised</td>
</tr>
<tr>
<td>Disease severity</td>
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<tr>
<td>Pediatric Asthma Quality of Life Questionnaire</td>
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<tr>
<td>Negative Family Emotional Climate (NFEQ)</td>
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Family Climate, Depression/Anxiety, Emotional Trigger, Asthma Disease Severity Path Analysis Results

We carried out a three-step procedure to test whether anxiety and depressive symptoms predicted disease severity differentially. Compared with the Baseline Model, the Depression Model had a better fit [delta chi-square(2) = 39.95, p < .001], indicating an improved model and significant association between child depressive symptoms and emotional triggering (β = .30, p < .001) and disease severity (β = .25, p < .001) (Table III). Higher depression scores were associated with higher emotional trigger scores as well as greater disease severity. Next, in the Depression and Anxiety Model, pathways from both anxiety and depression to emotional triggering and disease severity were freely estimated, and compared with the Depression and the Baseline Models. The fit of the full model indicated significant improvement in fit [delta chi-square(2) = 10.84, p < .01], indicating that adding anxiety symptoms improved the model (Table III). This analysis revealed an additional significant association between child anxiety and disease severity (β = .17, p < .01) and a trend towards association between child anxiety and emotional triggering (β = .11, p = .08). In addition, the pathway indicating higher depression scores in association with higher emotional trigger scores and greater disease activity remained significant. Thus the best model appeared to be the full model including both anxious and depressive symptoms (Fig. 2).

The results for the Depression and Anxiety Model confirmed all pathways except for the direct pathway between NFEQ and emotional triggering, and between NFEQ and child anxiety. In addition, the indirect pathway between child anxiety and disease severity was not confirmed because of a lack of significant relation between child anxiety and emotional triggering. The test

Table III. Goodness of Fit Summary for the Path Model

<table>
<thead>
<tr>
<th>Model</th>
<th>Chi-square</th>
<th>df</th>
<th>p</th>
<th>NFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline Model</td>
<td>50.91</td>
<td>5</td>
<td>.000</td>
<td>.52</td>
<td>.18</td>
</tr>
<tr>
<td>Depression Model</td>
<td>10.96</td>
<td>3</td>
<td>.012</td>
<td>.90</td>
<td>.10</td>
</tr>
<tr>
<td>Depression plus Anxiety Model</td>
<td>.12</td>
<td>1</td>
<td>.720</td>
<td>.99</td>
<td>.00</td>
</tr>
</tbody>
</table>

NFI, normed fit index; RMSEA, root mean square error of approximation.
Depression Model: Paths from anxiety to emotional triggers and disease severity constrained to zero.
Depression plus Anxiety Model: Paths from depression and anxiety to emotional triggers and disease severity were freely estimated.

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Figure 2. Path model of family effects on emotional triggers and disease severity: Depression plus anxiety model. *p < .05, **p < .01, ***p < .001.
of alternative nested models to identify distinct effects of depressive symptoms and anxiety indicated that both depressive and anxious symptoms may play a role in disease severity, but there is a suggestion that only depression may influence disease severity by way of emotional triggering.

Discussion

This study demonstrated that child depressive symptoms as measured by self-report, maternal report, and clinician rating are associated with reported emotional triggering of asthma. Furthermore, the clinical significance of emotional triggering was evidenced by the association observed between the ATI emotional trigger score and objective (NHLBI) ratings of disease severity, along with poorer asthma-related quality of life. The finding that depressive symptoms were associated with disease severity is consistent with a large literature (McQuaid et al., 2001), and with previous findings from our laboratory (Waxmonsly et al., 2006; Wood et al., 2006b). However, to our knowledge, this is the first time emotional triggering of asthma has been specifically assessed in association with anxiety or depression and with disease and quality of life outcomes. This design permitted the examination of one potential psychobiological pathway by which depression may influence asthma disease morbidity.

The path model testing the depression and anxiety pathways in the same model was a viable and significantly better model than the model with depression pathways alone. However, the marginal significance of the anxiety to emotional triggering pathway raises the question as to whether depressive and anxious symptoms play differential roles in disease severity in pediatric asthma. Although there is abundant evidence that anxiety and depressive symptoms co-occur, it is possible that each symptom type has differential psychobiological effects in the realm of autonomic patterning, which may have significance for airway function in asthma (Miller & Wood, 2003). These findings, taken into consideration with theories and empirical tests of autonomic mechanisms of the effect of emotions in asthma (Lehrer et al., 1996; Miller & Wood, 2003), indicate the importance of further investigation of both depressive and anxiety symptoms in the same studies in order to elucidate the pathways and mechanisms by which each type of symptom may influence asthma disease.

There are alternative explanations of these findings. One is that it is possible that being depressed causes a child to subjectively attribute to emotional triggering those attacks that are actually caused by other triggers. The finding that the parent report CDI-P and clinician-based CDRS-R ratings were also correlated with the ATI emotional trigger score argues against this as an explanation.

Theoretical Implications

The current study is consistent with the ANS–HPA–Immune Dysregulation Model (Miller & Wood, 2003) which posits that depressive emotional states promote asthma disease activity by way of the accompanying physiological dysregulation which potentiates the effect of any asthma trigger. However, the current findings, while consistent with the original model, extend the model by indicating that depressed states may also directly exacerbate the effects of emotions and stress as asthma triggers (i.e., the greater the depression, the greater the emotional reactivity, and the greater the emotional reactivity, the greater the frequency or intensity of emotional triggering).

The disconfirmation of a pathway by which NFEQ directly affects emotional triggering of asthma is intriguing in the face of other literature finding associations between family factors and asthma disease outcomes (Kaugars et al., 2004; Wood et al., 2006a). However, the measure of NFEQ used in this study is a self-report of only one person, the mother, and her report might be highly influenced by her own emotional state. Alternatively, it is quite possible that the overall negativity in the family is not what matters, but rather negativity directed toward the child in question. Findings from an earlier study indicate that negative emotional climate may influence the child’s depression by way of insecurity of the parent–child relationship (Wood et al., 2006b), a pathway which is yet to be explored with respect to emotional triggering.

Strengths and Limitations

One strength of this research is that it tests multilevel factors (family, emotions, and disease) via multiple informants and objective measures of disease. However, family emotional climate was assessed in a limited way. The FEQ is a global scale, characterizing the family as a whole, and it is a self-report measure from only one family member. Furthermore, the ATI is still limited by its nature as a self-report instrument, despite support for its validity indicated by associations between the ATI emotions subscale, depressive symptoms, disease severity,
Another strength of this research is that anxiety and depressive symptoms are differentiated and measured in the same study, whereas usually they are studied separately or globally as internalizing symptoms. The value of this distinction was corroborated by the differential relations between anxiety and depressive symptoms suggested in the path models. It is a limitation that anxiety was only measured by self-report, whereas depression was measured by self-report, parent report, and clinician interview/rating. In addition, because we had only the child self-report of anxiety, we were limited in the path analyses to using only the child self-report of depression in order to make our comparison of the anxiety versus depression pathways more comparable. Further research with multiple informant measures is indicated.

The cross sectional nature of this study is a limitation which precludes interpretations of direction of effect. Because the design of this study is cross-sectional, it is impossible to rule out the possibility that increased disease activity may cause the child to have more depressive symptoms and to attribute asthma attacks to emotions. Indeed, it is likely that the psychobiological effects in asthma are bi-directional or even multidirectional. Currently, we are collecting laboratory-based family interaction data which is rated for a variety of positive and negative emotional interactions among family members. This data is collected in concert with autonomic nervous system and cortisol measures, and with pulmonary function tests throughout the family interactions. This will allow for testing direct effects of family emotional interchange on physiology and airway function in children with asthma. This experimental approach is necessary in order to clarify directions of effect in the associations reported above and elsewhere.

It is a strength of the study that we have both males and females in this sample, as well as a wide range of socioeconomic status and racial grouping. However, the current sample size is too small to support systematic observation of the influence of gender, race, minority status, and SES on the effects under investigation. Therefore caution must be taken in generalizing the findings to any subgroup in this sample, pending further investigation.

Recruitment from the ED allowed the study to capture a sample with a wide range of asthma disease severity, mild to severe. However, ED recruitment may differentially tap an asthma population whose asthma is more severe or more poorly managed than in the general population and hence limit external validity of the findings.

Clinical Implications

Given asthma’s significant morbidity and potential mortality, identification of treatable conditions that may worsen asthma activity, such as depression or anxiety, is of significant clinical value. Furthermore, the observations that depressive symptoms may potentiate emotional triggering of asthma, and that emotional triggering predicts disease severity, suggest the importance of specifically interviewing the child and parent with respect to emotional triggers of the child’s asthma, as well as for signs of depressive or anxious states. The CDI and the ATI are systematic measures designed primarily for research purposes, but they can guide the clinician towards important avenues for inquiry. If signs of depression or reports of emotional triggering are discovered by the medical health provider, it is important to refer the child and family for further assessment and intervention for emotional problems and/or family distress.

Conflict of Interest: None declared.

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