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USING STRUCTURAL EQUATION MODELING TO UNDERSTAND THE ROLE OF THE
FAMILY IN PEDIATRIC ASTHMA CONTEXTS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science
at Virginia Commonwealth University

By: Nour Al Ghriwati
B.A., University of Maryland, Baltimore County, May 2014

Director: Marcia Winter, Ph. D.
Professor
Department of Psychology

Virginia Commonwealth University
Richmond, VA
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Abstract

USING STRUCTURAL EQUATION MODELING TO UNDERSTAND THE ROLE OF THE FAMILY IN PEDIATRIC ASTHMA CONTEXTS

By Nour Al Ghriwati, B.A.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University. Virginia Commonwealth University, 2015.

Virginia Commonwealth University, 2015

Major Director: Marcia Winter, PhD.
Assistant Professor
Department of Psychology

Family factors have long been associated with the psychosocial adjustment of children with chronic illnesses, such as asthma (Minuchin, 1975; Rapee, 1997). Research indicates that negative family factors may also contribute to child disease severity, via bio-behavioral mechanisms of effect (Wood et al., 2006); however, these pathways have yet to be examined with a comprehensive focus on more positive family factors. This study sought to examine whether factors such as family cohesion, problem solving abilities, and communication influence asthma symptom severity in children via their effects on child depression and anxiety symptoms. Using structural equation modeling, we identified significant indirect associations between family factors and child asthma severity via child depressive symptoms; however, these associations were not present in models mediated by child anxiety symptoms. Results highlight the importance of families in pediatric asthma settings. Findings suggest differential roles for anxiety and depression in their associations with child asthma severity.

Using Structural Equation Modeling to Understand the Role of Positive Family Factors in Pediatric Asthma Contexts

Asthma is the most prevalent chronic illness in children today, affecting one in ten children at any given time (Center for Disease Control, 2013). In 2014, more than seven million children in the United States had asthma, and these prevalence rates have more than doubled since 1980 (National Heart, Blood, and Lung Institute, 2014). Compared to children without asthma, children with asthma report greater social difficulties, limitations in their daily activities, and frustration from feeling left out (Juniper, 1997). Given the increasing magnitude of this problem over time, research has focused on identifying the factors that contribute to asthma's etiology and progression.

Initially, the etiology of asthmatic conditions was viewed as solely physiological or genetic in nature; researchers focused on an asthma disease model that did not consider the broader context beyond the child. Some linear models later began linking childhood experiences to emotions that result in illness, but this research still only focused on the sick child and failed to include familial influences (Graham, Rutter, Yule, & Pless, 1967). In 1975, Minuchin et al. presented one of the first models for psychosomatic child illnesses that incorporated the role of the family. Since then, research has expanded on this model and identified a number of ways in which families can influence a child's adjustment to his/her asthma diagnosis (Spagnola & Fiese, 2010; Wamboldt, Wamboldt, Gavin, Roesler, & Brugman, 1995; Wood et al., 2006). The utility in this line of research lies in its ability to understand how biopsychosocial processes may contribute to asthma severity in affected children.

The purpose of the proposed study is to evaluate how certain positive family characteristics, such as family communication, cohesion, and problem-solving skills, are associated with a child's adjustment to his/her asthma condition. Past studies have identified

various family and parent-child relationship characteristics that affect asthmatic children's internalizing symptoms and asthma severity (Fiese, Winter, Anbar, Howell, & Poltrock, 2008; Lim, Wood, Miller, & Simmens, 2011; Wood et al., 2007). However, existing studies focused on the effects of negative family characteristics, such as family dysfunction, conflict, and parental rejection, on child adaptation. Few studies to date have examined the effects of positive family factors on outcomes in child asthma contexts. Thus, this relationship in which positive family characteristics may influence an asthmatic child's emotional well-being, will be examined in the current study. Additionally, past studies have yielded mixed findings on whether children's emotional well-being directly impacts their asthma severity (Lim et al., 2011; Wamboldt et al, 1995). Therefore, this study will examine the differential effects of child depressive and anxiety symptoms on asthma symptom severity; asthma symptom severity will be defined via measures of subjective functional asthma severity, child quality of life, and objective pulmonary functioning. Finally, this study will examine the roles of child depression and anxiety symptoms in mediating the effect of family positive factors on child asthma severity.

Pediatric asthma will first be explained, followed by a description of family systems theory and the bio-psychosocial model of illness, which together provide the rationale for this study. Family communication, cohesion, and problem-solving abilities will be defined in terms of how they may influence child adaptation to asthma. Then, the literature that links child anxiety and depressive symptoms to asthma outcomes will be presented. The bio-behavioral family model, in conjunction with Miller's theory of autonomic dysregulation, will guide the proposed examination of family influences on children's emotional and physical well-being.

Pediatric Asthma

Asthma is a chronic condition marked by recurrent wheezing periods and sporadic symptom exacerbations (Wood, 2002). In 2014, pediatric asthma affected more than seven million children in the United States, with a disproportionate amount of them being low-income and urban. Thus, understanding the contextual processes that may influence asthma rates and severity is of high public health significance (National Heart, Blood, and Lung Institute, 2014).

Asthma is characterized by the inflammation and narrowing of pulmonary airways, which in turn restricts the amount of air that can travel through to the lungs. Inflammatory cells, such as eosinophils and mast cells, circulate through asthmatic children's airways and emit pro-inflammatory cytokines; these cytokines are responsible for subsequent mucus secretion and airway structural changes (Lemanske, 2002). As our understanding of asthma has progressed, an increased role for inflammatory disease mediators, such as leukotrienes produced by leukocytes, has been identified in the disease's pathology (Valacer, 2000).

There are three phases during an asthmatic episode: acute, marked by smooth muscle contraction and lung mucus secretion; chronic, marked by the flow of inflammatory cells into the airways; and irreversible, when airway remodeling has permanently occurred (Lemanske, 2002). Asthma symptom exacerbations can range in severity and may cause different levels of functional impairment in affected children (Wood, 2002).

Unlike with other chronic illnesses, the causes of asthma are not well understood; that notwithstanding, a number of risk factors have been consistently associated with asthma onset and maintenance of symptoms (Noutsios & Floros, 2014). First, a genetic disposition for atopy, a tendency to develop an allergic state, has been widely recognized as a risk factor for asthma; asthma tends to cluster in families, supporting the potential role of genetics in its pathology (Valacer, 2000). Second, sex has been implicated as a possible risk factor. For example, in

younger children, males are at increased risk to develop asthma, but with increasing age and hormonal variations, females become more susceptible to acquiring asthmatic symptoms (Noutsios & Floros, 2014). Third, environmental factors such as viral respiratory tract infections, exposure to allergens, and exposure to cigarette smoke have been associated with asthmatic episodes and may play a role in asthma etiology (Noutsios & Floros, 2014). Finally, individual factors such as poor diet and premature birth have also been associated with asthma onset and prognosis in children (Valacer, 2000).

While a child's psychological well-being is often overlooked by a physician, research suggests that untreated maladaptive emotions can result in poorer prognoses, highlighting possible links between children's emotional health and their asthma symptomatology (Peters & Fritz, 2011). It has also been proposed that family climate comprises a child's emotional and thus physical health; families with certain stressful profiles may have children with worsened asthma conditions (Wood et al., 2008). What is less clear is how different emotional states, such as anxiety or depression, may impact asthma severity in distinct ways (Wood et al., 2008).

Asthma from Ecological, Family Systems, and Biopsychosocial Perspectives

Past studies have identified various biological, psychological, and social factors that influence a child's asthma progression (Noutsios & Floros, 2014; Peters & Fritz, 2011; Wood et al., 2008). Theoretically, it is important to understand how a child's development is influenced by mutual interactions between the child, his/her immediate environments, and the larger social contexts within which these environments reside (Bronfenbrenner, 1977). Bronfenbrenner's ecological model of development posits that multiple contextual layers encircle the child and interact with one other to shape the child's growth (Bronfenbrenner, 1977). This model is an example of a general systems approach, which posits that the structure and organization of a

system affects the functioning of its individuals (Wood, 1994). According to Bronfenbrenner (1979), the primary context in which a child develops is the microsystem, which consists of immediate interactions with other people. The family is positioned in this microsystem, and thus, the bidirectional relationships between the child and his/her family must be examined to understand how they influence the child's psychological and physical development.

Focusing on the family aspect of the microsystem, Family Systems Theory (FST), proposed by Murray Bowen and colleagues during the latter half of the twentieth century, emphasizes the role of the family as an interconnected unit of functioning that reciprocally influences each of its member's behavior and development (Bowen, 1966). The family's united emotional well-being, what Bowen refers to as the "undifferentiated ego mass," explains why individuals cannot be understood in isolation without considering the overarching family dynamics; FST also explains how any change in the functioning of one family member subsequently affects the functioning of other members in the unit (Bowen, 1966; Bowen, 1974). One of the fundamental concepts of Bowen's theory is that of differentiation, explaining the degree to which each family member is able to separate his/her emotions from cognitions without being overly dependent on the family (Bowen, 1974). This degree of differentiation, or emotional maturity, is passed on across generations and characterizes family relationships; according to Bowen's theory, more adaptive, less enmeshed families tend to have individuals with optimal differentiation levels and better coping mechanisms in response to environmental stressors (Bowen, 1974). Conversely, families with decreased differentiation levels have members who have increased emotional difficulties, given that it is more difficult to establish emotional homeostasis in response to changes (Kerr & Bowen, 1988). Another pertinent characteristic proposed by FST is the nuclear family emotional environment, which explains how

family emotions, such as chronic anxiety, are passed on to the developing child through stressful, less constructive family interactions (Kerr & Bowen, 1988). According to Bowen, normally functioning families are those whose members are differentiated, minimally anxious, and able to sustain healthy relationships with others in the unit (Kerr & Bowen, 1988). The FST serves to be a very valuable theory for understanding how overall family functioning may significantly influence a child's emotional well-being and response to stressors such as asthma.

In 1994, Beatrice L. Wood introduced the bio-behavioral family model (BBFM), a developmental model that stems from other general system theories. By focusing on the family microsystem of the ecological model, Wood analyzes the interactions between the familial, psychological, and biological variables that may influence a child's physical well-being in the context of chronic illness. The BBFM posits that the family plays a crucial role not only in coping with stress, but in the actual disease severity of a child; the child, in turn, affects the family. The BBFM therefore builds on FST by extending the impact of family functioning to each member's physical health (Wood, 1994). There are three ways in which family members may influence each other's physical health: physically, through behavior mediation, and through bio-psychological pathways; the BBFM focuses on the latter, associating familial contributions with the exacerbation or buffering of a child's illness through their effects on the child's bio-behavioral reactivity (Wood, 1994).

Wood's BBFM has examined a number of family contributing factors to illness severity. Arguably the most important family characteristic proposed by the BBFM is interpersonal responsivity, or the intensity with which people in the family respond to one another as characterized by positive emotions to soothe the child or negative emotions that may adversely influence the child. Additionally, the family members' proximity, generational hierarchy,

triangulation around the child's illness, and inter-parental conflict are all factors that may also shape a child's disease severity (Wood, 1994). These factors are proposed to influence chronic illness' severity by affecting the child's psychological processes, such as anxiety and depression, which in turn changes a person's bio-behavioral reactivity (Wood, 1994). Bio-behavioral reactivity, or the ability to regulate emotions, describes the intensity of a response to an emotional stimulus; it influences physiological functioning and, thus, disease progression (Wood, 1994). Families characterized by more negative emotional climates are proposed to have children with greater internalizing symptoms and thus a poorer asthma prognosis. Thus, this study considers pediatric asthma from Biobehavioral Family Model (BBFM) and Family Systems Theory perspectives, by examining the role the family plays in influencing asthma severity via bio-behavioral processes.

The family plays an important role in children's development during middle childhood and the pre-adolescent years. Middle childhood is the time when children are developing a sense of logical thinking and understanding the world around them in a more comprehensive way (Sameroff & Haith, 1996). Additionally, children gain increased awareness of the self and their social environment (Sameroff & Haith, 1996). Children's emotional problems may also arise during this critical period, since children face several new challenging situations, like beginning elementary school, which may lower their self-esteem (Wigfield et al., 1997). In addition to recognizing their own capabilities, children begin gaining responsibilities in middle childhood and early adolescence; therefore, it is particularly important for the family to provide social support and communicate well at this time (Wigfield et al., 1997). Pre-adolescent years are the years preceding the shift from predominantly family influence to that of peers and the increased separation of the child from his family (Fulligni & Eccles, 1993). Thus, strong family

relationships are crucial to helping with difficult pubertal transitions and radical development shifts that occur in adolescence (Fulligni & Eccles, 1993). For these reasons, it is particularly important to understand how families influence children's psychological and physical well-being during middle childhood and early adolescent years.

Positive Family Factors

If, as proposed by the BBFM, negative family context results in negative effects on a child's psychological functioning by increasing anxious and depressive symptoms, the question becomes whether positive family contexts have the opposite effect. This study focuses on the effects of families' positive factors, including family communication, cohesion, and problem-solving abilities. This study is unique in looking at families through a more positive lens.

Several family factors may protect children with asthma from developing maladaptive patterns. Positive family factors consistently identified in developmental research include healthy family communication, cohesion, and problem-solving abilities (Delambo, Levers-Landis, Rosland, Heisler, & Piette, 2011; Reichenberg & Anders, 2005). In fact, at least one theory designates these three factors as the most important characteristics in determining the overall functionality of a family (Circumplex Model of Family Systems; Olson, Sprenkle, & Russell, 1979). More recent studies have also identified links between these factors and children's adaptation to asthma diagnoses; these studies are reviewed below, followed by a discussion of current gaps in the family asthma literature and this study's aims of expanding our knowledge on the effects of positive family factors in asthma settings.

Family communication. Communication is an important family characteristic that influences children's development and emotional well-being (Olson, 2000). Family communication refers to openness and direct forms of communication, and how family members

show genuine interest in and support for each other's statements throughout their interactions (Kaugars, Klinner, & Bender, 2004; Fiese, Winter, & Botti, 2011). More balanced families, with better communication patterns, have members who attentively listen, clearly discuss viewpoints, and share feelings with each other; research has demonstrated that children from these openly communicating families exhibit better socialization skills, decreased peer aggressive levels, and fewer social withdrawal symptoms than children from more restricted families (Anderson, 1986; Fitzpatrick et al., 1996; Olson, 2000). Most studies of child psychopathology have focused on characteristics of dyads, such as parent-child communication, and have found more open, validating communication styles to be associated with fewer child internalizing symptoms (Brown et al., 2007). However, it is crucial to examine the effects of communication of the entire family unit, as opposed to focusing on these dyads, on child depression and anxiety symptoms.

Family communication has also been associated with some enhanced asthma outcomes. For example, Fiese et al. (2011) found that positive family group communication during mealtime interactions significantly predicted children's quality of life. Mitchell, Powers, Byars, Dickstein, and Stark (2004) also show the importance of clear communication in predicting less frequent behavior problems in children with cystic fibrosis. Other studies have focused on negative aspects of family communication and have found that increased conflict, with less healthy family interactions, leads to compromised child mental health (Wood et al., 2000). To date, no research has evaluated the effects of family communication, in conjunction with other characteristics, on children's physical health via their influence on child internalizing symptoms. Therefore, this study aims to build on the preliminary research on family communication by analyzing its effects on child depressive and anxious states and on asthma severity.

Family cohesion. Another family factor that has been associated with child adaptation to asthma in previous studies is family cohesion. Family cohesion is a construct that, in past literature, was defined as “highly enmeshed families”; therefore, increased family cohesion was linked with poor child psychological outcomes (Minuchin, 1975). Today, family cohesion is a separate construct from family enmeshment and is a more positive term that characterizes the “appropriate, healthy, and positive interactions and support among family members”; it is the sense of togetherness or unity in a system (Lindahl & Malik, 2001). This new definition of family cohesion is inversely associated with adolescent emotional and behavioral problems (Barber & Buehler, 1996). For example, Lucia & Breslaw (2006) found that families characterized by mothers as highly cohesive had children with fewer teacher and parent-reported internalizing and attention symptoms; these protective effects remained stable five years later.

Caregiver-reported family cohesion can also be important for child emotional adjustment in pediatric asthma populations. In a sample of children with asthma, high family cohesion, as rated by primary caregivers via a self-report questionnaire, was associated with better child-reported global self-worth and fewer parent-reported child emotional and behavioral problems (Reichenberg & Anders, 2005). Similarly, Silva, Crespo, and Canavarro (2014) found that greater family cohesion and expressiveness were associated with improved child quality of life in asthmatic populations. These effects were also observed in children with kidney disease: families with higher reported levels of cohesion had fewer hospitalizations than did conflict-inflicted families (Soliday, Kool, & Lande, 2001).

Whereas previous findings offer widespread implications for family cohesion and its effects on child adjustment, it is important to note that cohesion measures may be biased when reported by primary caregivers; more objective measures of cohesion may overcome this

limitation by minimizing possible self-report biases. In addition, few studies have linked cohesion with objective indicators of child asthma severity, such as pulmonary spirometry results, and no studies to date have evaluated the mechanisms through which family cohesion, in conjunction with other family positive factors, is associated with improvements in child asthma symptoms via its influence on child internalizing symptoms. Thus, we incorporate these aims into the current study.

Family problem-solving. Family problem-solving abilities are also central to child adjustment to environmental stressors and family interventions. Family problem-solving characterizes the family's ability to solve any conflicts or disagreements that may arise (Herzer, Denson, Baldassano, & Hommel, 2011). It has also been described as the ability of families to adjust and change in response to environmental stressors to maintain optimal family functioning (Epstein et al., 1983; Olson, 2000). Research on family problem-solving skills has focused on families undergoing significant stressors, such as poverty or injuries, or chronic illnesses (Carlson, Gesten, McIver, DeClue, & Malone, 1994; Giallo & Gavidia-Payne, 2006). For example, Giallo and Gavidia-Payne (2006) found that family problem-solving skills significantly predicted children's adjustment to a siblings' disability status. Additionally, specific family problem-solving interventions with families experiencing multiple risk factors in their life have been identified as efficacious (Drummond, Fleming, & Kysela, 2005).

Family problem-solving (PS) abilities also affect child functioning in populations with chronic illnesses. In a study of children with inflammatory bowel disease, low levels of caregiver-reported family PS were found to predict adolescents' self-reports of depressive symptoms; however, family PS was not found to moderate the impact of illness severity on child depression in this population (Schuman, Graef, Janicke, Gray, & Hommel, 2013). Though

research on family problem-solving in asthma populations is limited, emerging studies have tested the efficacy of problem-solving skills training for families of children with asthma; problem-solving training provides families with the tools needed to tackle new challenges that may arise with an asthma diagnosis (Knafl et al., 2013). Knafl et al. (2013) found, compared to standard psychosocial care, problem-solving skills training was significantly better at increasing children's generic health-related quality of life. This suggests that problem-solving abilities could act as another protective factor for children with asthma; however, further research is needed on the effects of family PS on child mental and physical health in asthma populations.

Family gaps in the literature. Although the effects of several family factors on child adjustment to asthma have been examined, there are several gaps in the literature that this study hopes to address. Firstly, past studies have examined family characteristics separately; this study aims to combine important family characteristics, such as cohesion, communication, and problem solving, to provide a more comprehensive, global picture of family climate in asthma contexts. Second, this study focuses on family positive factors as opposed to solely focusing on negative factors that may affect asthma outcomes. Finally, the study aims to understand how these family factors can influence children's asthma severity via their effects on children's internalizing symptoms. With this information, we hope to provide a number of targets for family interventions within pediatric asthma contexts.

Child Internalizing Symptoms

Children with asthma exhibit greater rates of internalizing – but not externalizing - symptoms in comparison to their same-aged peers without asthma (Alati et al., 2005). Studies in the past have focused on the effect of asthma severity on child emotional difficulties (Wamboldt, Fritz, Mansell, McQuaid, & Klein, 1998), whereas other research has taken the opposite

approach, focusing on how increased levels of internalizing symptoms may exacerbate asthma symptoms in children and adolescents (Baron et al., 1992; Miller & Wood, 1997, Peters & Fritz, 2011). Initial work on this directionality of effect focused on anxiety symptoms in predicting asthma severity (Butz & Alexander, 1993). The BBFM for asthma, however, posits that although depressive symptoms do play a role in mediating the effects of negative family environments on asthma severity, anxiety symptoms do not (Miller & Wood, 1997). This study hopes to advance this knowledge of the differential effects of anxiety and depression on asthma severity by separately analyzing how each factor mediates the relationship between positive family factors and child asthma symptoms. Associations between anxiety and depressive symptoms and asthma severity are presented below.

Anxiety. Children with asthma experience a significantly higher prevalence of anxiety symptoms in comparison with the non-asthmatic population (Bussing, Berket, & Kelleher, 1996). Anxiety symptoms include those of worry and fear, and an elevation of these symptoms is associated with the development of other emotional difficulties later on (Pine et al., 1998). A review by Katon, Richardson, Lozan, and McCauley (2004) indicated that nearly one-third of children with asthma meet the criteria for a diagnosis of an anxiety disorder. Additionally, children with asthma are nearly twice as likely to be diagnosed with an anxiety disorder as children who are unaffected by asthma (Peters & Fritz, 2010). The impairment caused by anxiety, its association with other emotional difficulties, and its increased prevalence in the asthmatic population makes it particularly important to study.

The relationship between anxiety and family factors is well-established in current pediatric literature. In their review on the literature that links family variables to child anxiety symptoms, Bogels and Brechman-Toussaint (2006) found that high parental conflict, poor family

communication, and increased family enmeshment were all associated with elevated child anxiety symptoms. Additionally, greater child anxiety has been linked to increased parental rejection and control (Rapee, 1997). Studies have also identified the positive effects of family positive factors on child anxiety symptoms. For example, in a study that evaluated the mediating outcomes of cognitive behavioral treatment on child anxiety, children from families with high cohesion and low parental stress showed the greatest reduction in their anxiety symptoms (Victor, Bernat, Bernstein, & Layne, 2007). The importance of a positive family environment becomes apparent in family cognitive behavioral therapies that successfully reduce child and parent anxiety by decreasing parenting over-control and rejection in families (Bogels & Siqueland, 2006).

While the link between the family environment and childhood anxiety seems clear, evidence concerning anxiety and asthma severity is less so. There are mixed findings for the magnitude and directionality of the relationship between anxiety symptoms and child asthma severity. For example, a study by Wamboldt et al. (1998) found no significant variations in anxiety symptoms among children based on differences in their asthma symptom severity. Also, anxious individuals have failed to exhibit objective changes in their pulmonary airways in comparison with those with fewer anxiety symptoms; nevertheless, these individuals have reported increased shortness of breath (Steptoe & Vogele, 1992). Similarly, in a population of children with asthma, heightened anxiety levels were not associated with the emotional triggering of asthma; however, these anxiety symptoms were associated with general asthma severity ratings (Wood et al., 2007). Additionally, although an association between anxiety symptoms and asthma has been identified, this relationship seems to disappear when controlling for child depressive symptoms (Wood et al., 2006). Conversely, Lehrer et al. (2002) report a

significant association between increased asthma severity and anxiety disorder symptoms. Conflicting findings make it difficult to conclude whether anxiety symptoms directly exacerbate asthma symptom severity; therefore, this study aims to examine the nature of the relationship between anxiety and child asthma by testing anxiety as a potential mediator of family positive factors' effects on asthma severity.

Depression. The relationship between child depressive symptoms and asthma severity is more clearly established. Depressive symptoms include feelings of persistent sadness, helplessness, irritability, loss of interest, sleep and eating difficulties, and difficulty concentrating (National Institute of Mental Health, 2013). Children with asthma are twice as likely to develop depressive disorders compared with children who do not have asthma (Katon et al., 2007). Additionally, past studies have found significant associations between asthma severity and child depressive symptoms (McQuaid, Kopel, & Nassau, 2001; Waxmonsky et al., 2006). Researchers have proposed that depressive symptoms may exacerbate children's asthma conditions via poor medication adherence and/or via autonomic nervous system biases (Miller & Wood, 1997). These psychophysiological pathways of effect will be described below, but first, we present current research that links family factors to asthma severity via child depressive symptoms.

Depressive symptoms may mediate the effects of certain family factors on child asthma severity (Wood et al., 2006). Initially, researchers focused on the effect of certain emotional states, such as depression, on pulmonary functioning in children with asthma and found that elevated levels of hopelessness led to poor pulmonary functioning (Miller & Wood, 1997). Wood et al. (2006) then identified certain family factors, such as negative family climate, triangulation, and parent-parent relationship quality, that indirectly influence asthma severity via increasing depressive symptoms; these resulting depressive symptoms directly predicted asthma

disease severity, even after controlling for child anxiety symptoms. Child depressive symptoms, partly predicted by negative family emotional climates, are significantly related to the emotional triggering of asthma; this triggering of asthma is in turn associated with poorer objective measures of lung functioning (Wood et al., 2007). Miller's Autonomic Dysregulation Model of the BBFM attempts to justify the presence of these relationships.

Miller's Autonomic Dysregulation Model of the BBFM

According to the BBFM, child bio-behavioral reactivity, characterized by emotional processes, plays a crucial role in linking psychological factors to child illness severity (Wood et al., 2000). Persistent states, such as anxiety or depression, can be indicative of bio-behavioral reactivity; the reason depression is proposed to influence asthma severity to a greater degree than anxiety is that depressive conditions are more involved in parasympathetic (vagal) activation of the autonomic nervous system (ANS); conversely, anxiety states are involved in sympathetic activation of the ANS (Wood et al., 2000).

The ANS is the division of the nervous system that controls involuntary bodily processes, and because it innervates the smooth muscles that surround the pulmonary airways, it plays a role in regulating lung activity (McCorry, 2007). The ANS is divided into two distinct categories: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). Sympathetic activation of the nervous system is associated with emergency states that require "flight-or-flight" responses, whereas parasympathetic activation occurs during times of rest in order to regulate basic bodily functions (McCorry, 2007).

Parasympathetic nerves are principally associated with the airway's smooth muscle control and airway mucus secretion via their release of the neurotransmitter acetylcholine (ACH; Van der Velden & Hulsmann, 1999). Released ACH binds on the airway's smooth muscle

receptors to constrict bronchioles, secrete mucus, and dilate the surrounding blood vessels (Van der Velden & Hulsmann, 1999). The resulting narrowed airways and elevated mucus levels decrease the air volume that can travel through to the lungs and thus worsen asthma conditions (Miller & Wood, 1997). Sympathetic nerves play a considerably smaller role in asthma severity, since these nerves do not extend to the smooth muscles of the pulmonary airways; sympathetic nerves may only indirectly affect asthma severity via control of surrounding blood vessels, but this pathway of effect is still unclear (Van der Velden & Hulsmann, 1999).

In the late 1990s, Miller introduced the autonomic (ANS) dysregulation model of childhood asthma, which posits that depression may exacerbate asthma symptoms via “cholinergic and vagal biases” (Miller & Wood, 1997). A depressive state causes increased ACH production in the central nervous system, which subsequently causes bronchoconstriction in children with asthma (Miller & Wood, 1997). Research suggests that children with depression have predominantly vagal or parasympathetic activation of the nervous system in response to emotional stressors. Conversely, children who are not depressed respond with increased sympathetic arousal to these same stressors (Miller, Wood, Lim, Ballow, & Hsu, 2009). This tendency to show parasympathetic reactivity to stressors may increase airway resistance in children with asthma and depressive symptoms (Miller et al., 2009). Additionally, children in heightened depressive states have shown significantly higher airway resistance than children who are not depressed (Miller et al., 2009). Therefore, children who have more depressive symptoms may have increased asthma severity due to their heightened cholinergic states, vagal bias, and increased airway resistance.

Miller’s ANS dysregulation model of childhood asthma guides our mediation analysis, whereby family factors affect child asthma severity via child depressive symptoms. We

additionally test child anxiety as a mediator in this study, given the inconclusive evidence of its effects on a children's pulmonary functioning; though exploratory in nature, a non-significant mediation pathway via anxiety symptoms will provide support for Miller's explanation of depression having a unique influence on asthma severity. Conversely, a significant mediation pathway via anxiety symptoms may indicate the presence of other bio-behavioral mechanisms of effect in pediatric asthma settings.

Covariates

When examining the effects of family factors on child asthma severity, it is essential to account for the influence of confounding variables that are known to affect family functioning or child health indicators. Variables associated with child and family adjustment to pediatric asthma in past studies include child age, gender, and medication adherence (Almqvist, Worm, & Laynaert, 2008; Skobellov, Spivey, Clair, Schofstell, 1992).

Child age. Children experience variations in asthma severity and psychosocial well-being as they grow older; for example, hospital admissions for asthma-related emergencies in the first two years of life are greater than at any other age (Skobellov et al., 1992). Additionally, recent research has found that younger children with chronic conditions, such as asthma or obesity, report better health-related quality of life in comparison with adolescents and young adults (Moreira et al., 2013). Age also has been found to affect children's emotional development. Although no gender differences exist in children before age eleven, women become twice as likely to develop significant depressive symptoms after puberty (Angold & Rutter, 1992). Additionally, teacher reports of children's internalizing symptoms seem to increase in severity as children grow older (Snyder et al., 2009). These influences of child age on various aspects of child well-being make it imperative to control for age in our models.

Child gender. Child gender also has significant effects on family adaptation and child health in asthma situations. Gender's effects on asthma status in children seems to be age-dependent. Although males are more likely to have greater asthma severity at young ages, asthma status is more severe in females after puberty (Almqvist et al., 2008). Furthermore, in a study of children and adolescents with asthma, females reported significantly lower levels of health-related quality of life than their male counterparts (Warschburger et al., 2004). Child gender also influences the development of mental health issues; as stated above, females develop anxiety and depressive symptoms at a greater prevalence than males after puberty (Angold & Rutter, 1992). Finally, child gender has been significantly associated with family functioning in the contexts of childhood asthma and diabetes, with families with female children reporting greater levels of cohesion (Holden, Chmielewski, Nelson, Kager, & Foltz, 1997).

Medication adherence. Children with asthma are typically prescribed two to three daily doses of medication to minimize airway inflammation and prevent asthma symptom exacerbations (McQuaid, Kopel, Klein, & Fritz, 2003). Non-adherence to asthma medication has been consistently associated with increased levels of asthma severity on both objective and self-report measures (McQuaid et al., 2003). Furthermore, negative family factors have significant effects on child asthma outcomes via their influence on medication non-adherence; for example, children from highly conflicted families with elevated psychosocial symptoms reported significantly reduced adherence to medication in comparison with more cohesive families (Kaugars et al., 2004). Finally, children who report increased anxiety and depression symptoms are significantly more likely to be non-adherent with their asthma medication than their healthy counterparts (Lehrer et al., 2002).

Importance of Multi-Rater Multi-Method (MRMM) Designs

Obtaining measures from multiple informants and via multiple methods, referred to as a MRMM design, is important to ensure comprehensive depictions of each construct of interest in research. Having data from multiple sources yields more information about each patient's psychopathology and minimizes biases that may result from single indicator measurements; additionally, it is accepted that combining this data results in particularly valuable assessment and treatment information (Achenbach, 2006). Furthermore, using multiple ratings minimizes biases that may result from separate ratings of youth problems and offers unique views of the child (Achenbach, 2006). Discrepancies do exist between raters, and many have argued that these variations are simply due to the different environments in which each rater examines the child (Smith, 2007). Others argue that these discrepancies may be due to social desirability biases and rater characteristics and may skew accurate evaluations of problems (Smith, 2007). There is a general consensus, however, that although there may be different ways of combining rater information, one must obtain MTMM measurements whenever possible (Smith, 2007).

Young children, under twelve years old, report internalizing symptoms consistent with those of their caregivers; additionally, there are strong correlations between caregivers' reports of child internalizing symptoms (Tarren-Sweeney, Hazell, & Carr, 2004; Grietens et al., 2004). In addition, teacher ratings, though not highly correlated with parent reports, provide information about child behavior in a unique setting and may add on to parent reports of these symptoms (Smith, 2007). Therefore, including ratings from both caregivers and teachers into our measures of child anxiety and depression will provide us with a more complete picture of children's emotional problems in the most salient contexts of development for young children.

Similarly, by including multi-rater, multi-method measurements of asthma severity, we obtain a comprehensive picture of children's asthma severity in our sample. In the proposed

study, we include both subjective (parent and child) and objective reports of asthma severity. Subjective reports are uniquely suited to capturing the pragmatic effect of asthma on children, including activity limitations, exercise impairment, and school absenteeism (Cowen, Wakefield, & Cloutier, 2007). Meanwhile, objective measures of lung function can best capture the physical aspect of lung functioning, via Forced Expiratory Volume (FEV1). By combining these measures, we are adhering to the National Heart, Lung, and Blood Institute's guidelines (2002) for the determination of asthma severity in this population.

Statement of the Problem

Regardless of the enhancement of medical practices and procedures available today, asthma prevalence continues to increase throughout the world, with roughly 25.7 billion people being affected in 2010 (Akinbami et al., 2012). This increase in asthma diagnoses, in addition to our inadequate knowledge of all of asthma's specific causes, makes its etiology a particularly salient topic of research (Noutsios & Floros, 2014). While the impact of negative family factors has been discussed and the literature points to a number of deleterious effects of these factors on asthma outcomes, no study to date has evaluated these relationships via a positive lens (Wood et al., 2007). Understanding how particular family factors cohere to protect children from increased asthma severity would allow for more comprehensive family asthma interventions. Additionally, understanding the differential effects of child anxiety and depression on asthma symptom severity provides insight on how to screen patients who are at higher risk for worsened asthma morbidity. Finally, determining whether different reporters' ratings load on to child constructs of anxiety and depression provides insight into how certain entities may view the child in similar or different ways.

The purpose of the present study is to examine the relationship between family positive factors, child emotional well-being, and child asthma severity. First, we hypothesize a significant negative relationship between family positive factors (i.e., cohesion, problem-solving abilities, and communication) and child anxiety and depressive symptoms, such that positive family factors will predict lower levels of anxiety and depression in children. If supported, these results will help shed light on the importance of family functioning to children's emotional well-being in threatening pediatric asthma contexts. We will also examine the predictive relationships of child anxiety and depressive symptoms on child asthma symptomology in order to understand whether these symptoms relate to asthma in distinct ways. Finally, the examination of child depressive and anxiety symptoms as mediators of the effects of family positive factors on child asthma status will allow us to understand potential mechanisms by which families may ultimately influence a child's asthma status. A clearer understanding of this mediation model could allow clinicians to focus on particular family skills in their interventions with pediatric asthma; additionally, identifying different mediating pathways may allow clinicians to focus on depressive or anxiety symptoms in attempts to minimize child asthma morbidity.

Hypotheses

- 1) Greater parental positive factors, such as cohesion, communication, and problem-solving abilities will negatively predict child asthma severity.
- 2) Greater parental positive factors will be significantly associated with lower anxiety and depressive symptoms.
- 3) Child depressive symptoms will be positively related to child asthma severity.
- 4) After controlling for child age, gender, and medication adherence, depressive symptoms will mediate the effects of family positive factors on child asthma

severity, with increased family positive factors predicting decreased asthma severity via a decrease in depressive symptoms.

- 5) In concordance with Miller's ANS dysregulation model, we hypothesize that child anxiety symptoms will not significantly predict child asthma severity.
- 6) In concordance with Miller's ANS dysregulation model, child anxiety symptoms will not significantly mediate the effects of family positive factors on child asthma severity.

Methods

Participants

Data were drawn from a larger study, the *Family Life and Asthma Project*, which examined the effects of asthma on child and family functioning. Participants were 215 children, ages five to twelve years old ($M = 7.86$, $SD = 2.18$) with an asthma diagnosis, and their families. Families were recruited at an ambulatory clinic of a hospital, a pediatric pulmonary clinic, and at various urban pediatric practices. Inclusion criteria included a confirmed asthma diagnosis for at least one year, prescription of asthma controller medication for at least six months, and absence of any other chronic illness associated with a daily medical regimen.

Procedure

After approval was obtained from the Institutional Review Board for this study, families were approached at their clinic visits and asked if they were interested in taking part in a study regarding the effects of pediatric asthma on families. Interested families were invited to a university laboratory setting, where caregivers' written consent and children's verbal assent were obtained and a number of child and caregiver measures were completed. Additionally, children and their families were videotaped completing multiple interactive activities, and these videos

were later coded by trained rating teams. Caregiver permission was also obtained to contact each child's teacher, who completed questionnaires for child behavioral and emotional problems at school via mail. Statistical analyses of these data will be conducted using Mplus Version 7.0 and SPSS version 22.0.

Measures

Family cohesion. During the initial lab visit, children and their caregivers were asked to complete a fifteen-minute interactive activity, in which they were asked to draw a "family crest." These interactions were videotaped and later coded via the System for Coding Interactions and Family Functioning (SCIFF; Lindahl & Malik, 2001). The cohesiveness or cohesion scale in this coding system represents the sense of mutual appreciation and unity between family members as they work together toward a common goal. Two coders separately provided a rating on a scale of 1-5 (1 = very low, 2 = low, 3 = moderate, 4 = high, and 5 = very high), and consensus was reached for each rating. Low cohesion identified families in which members appeared disengaged and disconnected from one another. Conversely, highly cohesive families had members who appeared united and comfortable among each other. Interrater reliability in our sample, obtained from 45 families with 12 of them having fathers present, was adequate ($\alpha = .82$).

Family Assessment Device. The Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983) is a self-report scale based on the McMaster Model of Family Functioning (MMFF), and it assesses the six family domains of problem solving, communication, roles, affective responsiveness, affective involvement, and behavior control. The FAD provides 60 statements, for which respondents are asked to indicate how well each statement describes their own family on a 4-point Likert scale. Scores for each of the six subscales are obtained by adding

together the respondent's ratings and dividing by the number of items. Higher scores are indicative of worse family functioning. For this particular study, scales of family problem-solving ability and communication will be used. The FAD has demonstrated adequate test-retest reliability (Cronbach's alpha = .66 to .76) and high internal consistency ($\alpha = .72$ to .92) (Miller et al., 1985). In this study, the FAD displayed acceptable internal consistency for the total score ($\alpha = .75$), problem-solving score ($\alpha = .67$), and communication score ($\alpha = .65$).

Child depression and anxiety symptoms. Child depression and anxiety symptoms were assessed via primary caregiver, other caregiver, and teacher reports:

Child Behavior Checklist. To assess levels of child anxiety and depressive symptoms, primary and other caregivers were each asked to individually complete the 118-item Child Behavior Checklist (CBCL; Achenbach & Edlbrock, 1983). The CBCL is a widely used measure of child behavior to be completed by parents or surrogates. It consists of two versions, one for children eighteen months to five years old and another for children six to eighteen years old. Each item on the CBCL describes a particular behavioral or emotional problem, which respondents are asked to rate on a three-point Likert scale (0=absent, 1= occurs sometimes, 2=occurs often). For this particular study, the DSM-oriented scales of affective problems and anxiety problems will be used; both scales have shown high levels of test-retest reliabilities ($\alpha = .82$ and .72 respectively) and significant cross-informant agreements (Pearson's $r = .69$ and .66 respectively) in past studies (Achenbach & Rescorla, 2001). T-scores greater than or equal to 70 on the CBCL scales correspond to clinical levels of child psychopathology.

Teacher-Report Form. The Teacher Report Form (TRF; Achenbach & Rescorla, 2001) is an equivalent form to the CBCL to be completed by children's teachers. The TRF has demonstrated similar psychometric properties to the CBCL, with acceptable test-retest (.6-.96)

and internal consistency scores (.72-.92). Additionally, significant cross-informant agreement on both the affective and the anxiety domains has been evidenced when comparing TRF scores to CBCL scores (Achenbach & Rescorla, 2001).

Pulmonary functioning. Child lung functioning was objectively measured via a PDS 313100-WSO KOKO Spirometer, which yielded measurements of forced vital capacity (FVC), forced expiratory flow in one second (FEV₁) and forced expiratory flow for 25 to 27 percent of vital capacity (FEV₂₅₋₇₅; see Raymond et al., 2012). Children exhaled three times into the spirometer, and the results from the test with the highest sum of FEV₁ and FVC were recorded. Additionally, children repeated spirometry testing after ten minutes of being administered Albuterol via an Aerochamber. After assessing all spirometry results, a board-certified pediatric pulmonologist classified asthma severity according to the guidelines set forth by the National Heart, Lung, and Blood Institute (NHLBI, 2007) and provided a rating of child pulmonary functioning on a 4-point Likert scale (1 = slight or normal, 2 = mild, 3 = moderate, 4 = severe), with higher scores indicating worse overall lung functioning.

Functional Severity Scale. Primary caregivers reported their perceived levels of their child's asthma severity via the six-item Functional Severity Scale (FSS; Rosier et al., 1994). Respondents were asked to report the severity levels of a variety of child asthma symptoms for the past year, including child wheezing, night wakening, speech limitations, and activity limitations. Consistent with previous research, a total severity score was calculated by combining ratings across the six items. Scores on the Functional Severity Scale have been significantly associated with school absences, functional impairments, medical care visits, and medication usage in a large sample of children with asthma; goodness of fit statistics indicate that the scale

accurately described 97% of the children in the initial scale development sample (Rosier et al., 1994). The FSS had adequate internal reliability in this sample ($\alpha = .71$)

Asthma quality of life. The Pediatric Asthma Quality of Life Questionnaire (PAQLQ; Juniper et al., 1993) was used to assess children's quality of life. The PAQLQ is a 23-item self-report measure on which children seven to seventeen years old report their levels of impairment regarding each problem on a seven-point Likert scale, with a score of one indicating all the time /extremely bothered and seven representing none of the time/not at all bothered. For the children aged five to seven in our sample, we used the Pictorial Version of the PAQLQ, on which children respond to each situation using a picture of a thermometer ranging from empty to full to rate how much a symptom bothered them in the past week; scores on this version are reverse-scored (Everhart & Fiese, 2009). The questionnaire has three domains: activity limitations, symptoms, and emotional functioning; for this particular study, we used the children's PAQLQ scores on the symptom domain of the scale in order to capture how children are being affected by asthma symptoms such as wheezing, coughing, or nocturnal awakening. Elevated scores on the PAQLQ scales indicate better quality of life in these children. Past studies have demonstrated good psychometric properties of the PAQLQ and have confirmed it as a valid way to evaluate and discriminate child quality of life differences (Juniper et al., 1993). This sample exhibited good internal consistencies for the symptom subscale, with Cronbach's alphas of .86 for the PAQLQ and .83 for the Pictorial version.

Demographics. Child demographics, such as age and gender, were reported by the caregiver as part of a demographics questionnaire. Caregivers also reported on their own sociodemographic variables.

Asthma medication adherence. Each child's adherence to prescribed asthma medication was tracked over a six-week period via either an electronic recording device (MDIlog-II) or telephone diaries for oral controller medication. Adherence was calculated as the number of doses taken each day divided by the prescribed dosages for that day, averaged over a six-week period. Both the MDIlog and telephone diaries have been found to provide satisfactory adherence measurements (Bender et al., 2000; Rapoff, 1999).

Analyses

Preliminary descriptive analyses. Initial data checking assessed the presence of variable normality, multivariate outliers, and linearity. These assumptions were evaluated via the examination of skewness and kurtosis values and Mardia's normalized estimates (Bentler, 2005). Similarly, the assumptions of residual normality, linearity, and homoscedasticity were tested by examining each variable's residual scatterplot. Finally, tolerance values and predictor correlations identified whether multi-collinearity existed in the dataset.

Confirmatory factor analysis. For each model, confirmatory factor analysis (CFA) was initially used to identify whether our hypothesized model fit the data appropriately, using Mplus version 7.31 (Muthen & Muthen, 2014). According to the CFA, indices with better fit indicated that our estimated covariances from the hypothesized model were similar to the observed covariances in our sample. These CFAs tested the following models: 1) all indicators for constructs of family positive factors, child depression, and child asthma severity 2) all indicators for constructs of family positive factors, child asthma, and child asthma severity. Following the recommendations of Hu and Bentler (1999), a combination of the Chi-Square, Root Mean Square Error of Approximation (RMSEA), and two incremental fit indices were used to assess model fit, as follows. The chi-square goodness-of-fit statistic and the RMSEA index were used to

evaluate our absolute model fit. A non-significant chi-square index and an RMSEA less than or equal to .05 represented an appropriate-fitting model (Jackson, Gillaspay, & Purc-Stephenson, 2009). The chi-square statistic is widely used and at moderate sample sizes, identifies models that do not represent the data well (Kline, 2005). The RMSEA is a similar index, but provides additional advantages of having a confidence interval, being sensitive to number of estimated parameters, and being less vulnerable to sample size variations (Hooper, Coughlan & Mullen, 2008). Additionally, the comparative fit index (CFI), an ancillary index score, verified the degree of model fit demonstrated by the absolute fit indices. This score is different from an absolute fit index in that it compares the model fit to a baseline model where none of the variables are related; the CFI is currently one of the most trusted ancillary index scores since it is least resistant to changes in sample size (Hooper et al., 2008). Finally, the Tucker Lewis Index (TLI) provided an indication of model fit, as it compares the tested model to a baseline model that is resistant to sample size changes (Bentler & Bonett, 1980). A CFI and TLI greater than or equal to .90 supported the strength of each hypothesized model (Bentler, 1990). By evaluating absolute and ancillary indexes of fit together, a more reliable, conservative estimate of model fit is obtained (Hu and Bentler, 1999). To determine whether each variable accurately represented its intended construct, all factor loadings and their significance were evaluated. All standardized parameter estimates were reported, and non-significant indicators were removed to provide a more parsimonious model. Maximum Log-Likelihood (ML) estimation methods were used for confirming model fit and for structural equation modeling analyses.

Structural equation modeling (SEM). Initial confirmatory factor analyses were followed by SEM analyses for each of the hypothesized mediation models (see Figure 4 and 5). The four previously mentioned indexes of model fit - chi-square, RMSEA, CFI, and TLI - were

evaluated to determine the appropriateness of fit to the sample data. Modifications specified in modification indices were only made if they were also theoretically justifiable, per recommendations by MacCallum (1995). All parameter estimates were calculated, along with corresponding standard errors, error variances, and significance levels (McDonald & Ho, 2002). The proportion of variance in child asthma severity accounted for by positive family factors and child depression or anxiety symptoms indicated the degree to which each model's predictors contribute to child lung functioning. Covariates were included in each proposed model to identify their relationship to each of the latent constructs; all covariates significantly related to at least one construct were retained and controlled for in the model. Finally, bootstrapping analyses, with 5000 samples, provided a 95 % confidence interval for the effect of each mediation pathway; a confidence interval that did not contain zero was indicative of significant mediation (Cheung & Lau, 2007).

Results

Preliminary analysis of missing data values identified the absence of 18.1 % of teacher and 7 % of primary caregiver ratings of child emotional and behavioral functioning; however, there were no particular patterns of missingness detected. Therefore, Full Information Maximum Likelihood (FIML) was used as an estimation method to replace missing data on variables of interest. FIML is a modern technique that uses all case values, in addition to estimates of standard errors, to estimate the most likely value of a missing case; FIML provides more accurate, less biased estimates than mean replacement, listwise deletion, and other methods of dealing with missingness (Little, 2003). Approximately 50 % of the other caregiver reports of child behavior were missing; thus, we excluded other caregiver reports in our models rather than estimate such a large number of missing cases.

Mardia's normalized estimates ($m_{ij} > 5, p < .001$) detected significant multivariate non-normality in the data (Bentler, 2005). To account for this non-normality, Maximum Likelihood Robust (MLR) estimation methods, often used in CFA and SEM to correct for sample univariate and multivariate non-normality, were used (Byrne, 2006; Muthen, 2011). Monte-Carlo experiments have found no significant differences in results when comparing MLR estimates of samples with high skewness and kurtosis; therefore, MLR estimates are considered robust to violations of normality assumptions (Enders, 2001).

The final sample consisted of 136 males and 79 females from diverse caregiver-reported racial backgrounds (53% White, 31% African-American, 3% Hispanic, 0.5% Asian American, and 12.5% other). Families varied with regards to their socioeconomic status, with Hollingshead index scores ranging from 8.00 to 66.00 ($M = 38.58, SD = 16.34$). On average, families had moderate levels of cohesion, communication, and problem solving abilities, with means and standard deviations of 3.49 ($SD = 1.10$), 1.99 ($SD = 0.36$), and 1.91 ($SD = 0.36$), respectively. Mean child anxiety and depression scores fell in the non-clinical range for both parent and teacher reports. Children varied with regard to objective asthma severity, with 22 % having mild asthma, 30 % mild persistent, 37 % moderate persistent, and 11 % severe asthma. Children had adequate overall medication adherence, as they took approximately of 72 % of their asthma medications as prescribed. Descriptive statistics for all family, psychological, and asthma severity variables are presented in Table 1 and bivariate correlations are presented in Table 2.

Table 1.

Descriptive Statistics for Family, Psychological, and Asthma Variables

Variable	<i>N</i>	<i>M (SD)</i>	Range
Child age	215	7.86 (2.18)	5-12
Medication adherence	180	0.72 (0.31)	0-1
Family cohesion	208	3.49 (1.10)	1-5
Family problem-solving ^a	208	1.91 (0.36)	1-4
Family communication ^a	208	1.99 (0.36)	1-4
Child anxiety (PC report) ^b	200	55.15 (7.02)	50-86
Child anxiety (Teacher report) ^c	177	54.30 (6.66)	50-91
Child depression (PC report) ^b	200	55.02 (7.24)	50-88
Child depression (Teacher report) ^c	177	55.37 (7.16)	50-80
Asthma severity/Lung dysfunction	207	1.95 (.99)	1-4
Functional Severity Scale (PC report)	215	8.57 (4.77)	0-23
Symptom-related QOL (Child report) ^d	210	0.00 (1.00)	-2.63-1.84

Note. ^aFamily Assessment Device scale scores: higher scores indicate poorer family functioning. ^b Primary caregiver ratings on Child Behavior Checklist (CBCL): higher scores indicate poorer functioning. ^cTeacher Rating Form(TRF): higher scores indicate poorer functioning. ^d Centered child-report on Pediatric Asthma Quality of Life Questionnaire (PAQLQ): higher scores indicate better symptom-related quality of life (QoL_{Sx}).

Table 2.

Correlations among Family, Psychological, and Asthma Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Cohesion	--												
2. Problem solving ^a	-.16*	--											
3. Communication ^a	-.16*	.39**	--										
4. Anxiety (PC) ^b	-.19**	.03	.05	--									
5. Anxiety (TRF) ^c	-.11	-.01	-.10	.23**	--								
6. Depression (PC) ^b	-.16*	.16*	.09	.66**	.05	--							
7. Depression (TRF) ^c	-.09	.10	.11	.18*	.45**	.18*	--						
8. Asthma severity	-.12	.03	.05	.08	.23**	.09	.34**	--					
9. FSS	-.16*	-.07	.02	.15*	.003	.19**	.18*	.16*	--				
10. Symptom QoL ^d	.03	.09	.04	.04	.03	-.01	-.20**	-.27**	-.12 ⁺	--			
11. Child age	.06	-.04	.03	-.10	-.03	-.04	-.08	.02	-.12	.05	--		
12. Child sex	-.09	.07	.09	.10	-.09	.19**	-.04	-.03	.06	.09	-.03	--	
13. Med. adherence	.13	-.04	.03	-.04	-.06	-.04	-.20*	-.24	-.17*	.17*	-.02	.01	--

Note. ^aFamily Assessment Device scale scores: higher scores indicate worse family functioning. ^bAchenbach Child Behavior Checklist (CBCL): higher scores indicate poorer functioning. ^c Achenbach Teacher Rating Form (TRF): higher scores indicate poorer functioning. ^d Child-report on Pediatric Asthma Quality of Life Questionnaire (PAQLQ): higher scores indicate better symptom-related quality of life (QoLSx).

⁺ $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Depression Pathway Model

Confirmatory Factor Analyses were used to evaluate the hypothesized depression model's fit to the observed sample values (see Figure 1). Results indicated that this model appropriately fit the sample data ($\chi^2 [17, n = 215] = 22.34, p = .17$; CFI = .943; RMSEA = .038; TLI = .906). Additionally, all indicators significantly predicted their hypothesized latent constructs at the $p \leq .01$ level. No modification indices greater than the assigned 4.0 value were suggested; therefore, no changes were made to the theorized depression model. According to the CFA model, statistically significant associations were present between family positive factors and child depression symptoms ($r = -.33, p = .048$) and between child depression symptoms and asthma severity ($r = .83, p < .001$). However, no significant relationship existed between the latent constructs of family factors and child asthma severity ($r = -.03, ns$; see Figure 1 for CFA results).

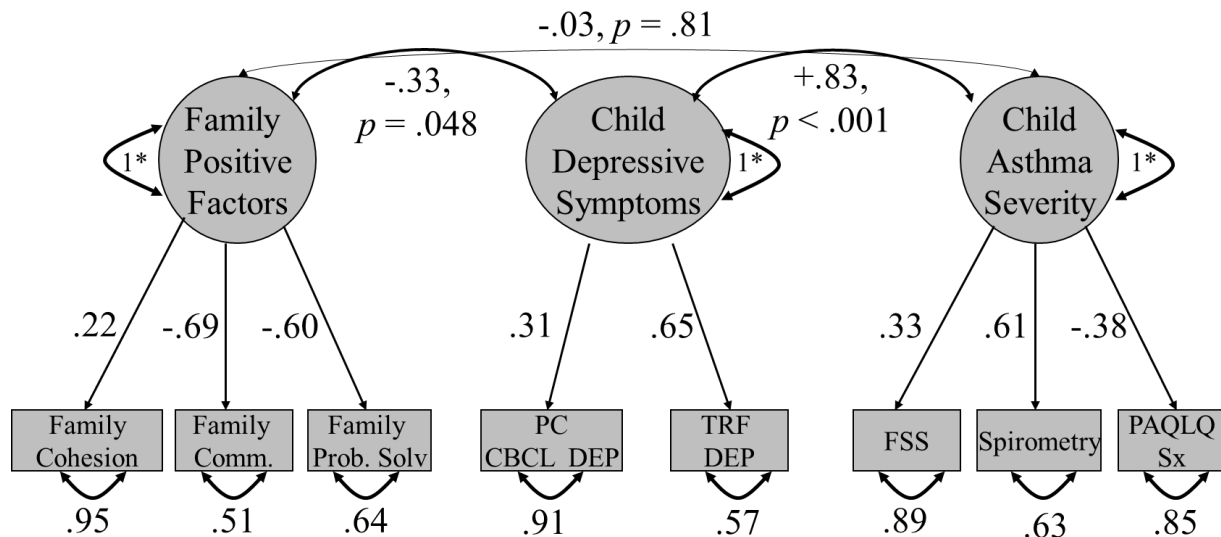


Figure 1. Confirmatory factor analysis for the model containing family positive factors, child depressive symptoms, and child asthma severity; $\chi^2 (17, n=215) = 22.34, p = .17$; RMSEA = .038_(.000-.077); CFI = 0.94; TLI/NNFI = 0.90; All loadings are significant at $p < .01$;

Note. Family Comm. And Family Prob. Solv. = Family communication and problem solving scales on Family Assessment Device; PC CBCL DEP = Primary caregiver reported depression syndrome score of the Child Behavior Checklist; TRF DEP = Teacher Report Form depression score; FSS = Functional Symptom Severity; PAQLQ Sx = Child ratings on Pediatric Asthma Quality of Life Symptom Domain.

The depression pathway model was then tested with SEM, using 5000 bootstraps to provide a 95 % confidence interval of any indirect effects. After including covariates of child sex, age, and medication adherence in the model, only adherence was significantly associated with the latent variables of depression and asthma severity; therefore, we controlled for the effects of medication adherence in these pathways. The model fit our data well, ($\chi^2 [23, n = 215] = 25.60, p = .31; CFI = .975; RMSEA = .023; TLI = .962$). Our model explained 9.8 % of the variance in child depression and 75.5 % of the variance in child asthma symptoms. Family positive factors were inversely associated with child depression ($\beta = -.31, p = .048$) but were not directly associated with child asthma severity ($\beta = -.27, ns$). Child depression was significantly associated with child asthma severity ($\beta = .75, p = .03$) and significantly mediated the path between family positive factors and child asthma severity; Point estimate of $-.29, 95\% CI = [-5.038, -0.011]$; see Figure 2 for depression mediation results).

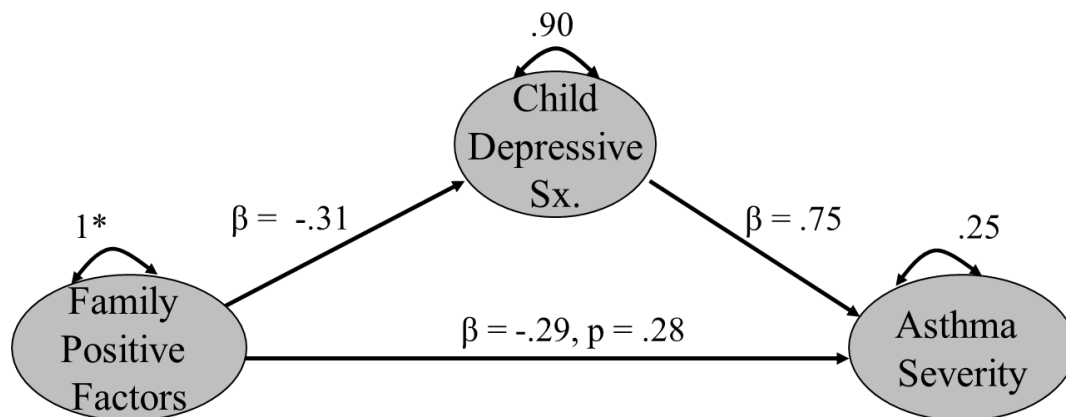


Figure 2. Structural equation model of the significant indirect relationship between family positive factors and asthma severity via child depressive symptoms; $\chi^2(23, n=215) = 23.60, p = .32; RMSEA = .02_{(.000-.062)}$ CFI = 0.97; TLI/NNFI = 0.96; Values reported are all standardized; Ab = $-.29; 95\% CI (-5.038, -0.011)$.

Anxiety Pathway Model

A confirmatory factor analysis, with the inclusion of family factors, child anxiety, and child asthma severity as latent constructs, indicated that the model adequately fit the sample data ($\chi^2 [17, n = 215] = 22.50, p = .17$; CFI = .927; RMSEA = .039; TLI = .870). All indicators significantly predicted their hypothesized latent constructs at the $p < .05$ level. However, none of the latent constructs were significantly associated with each other. This model is presented in Figure 3.

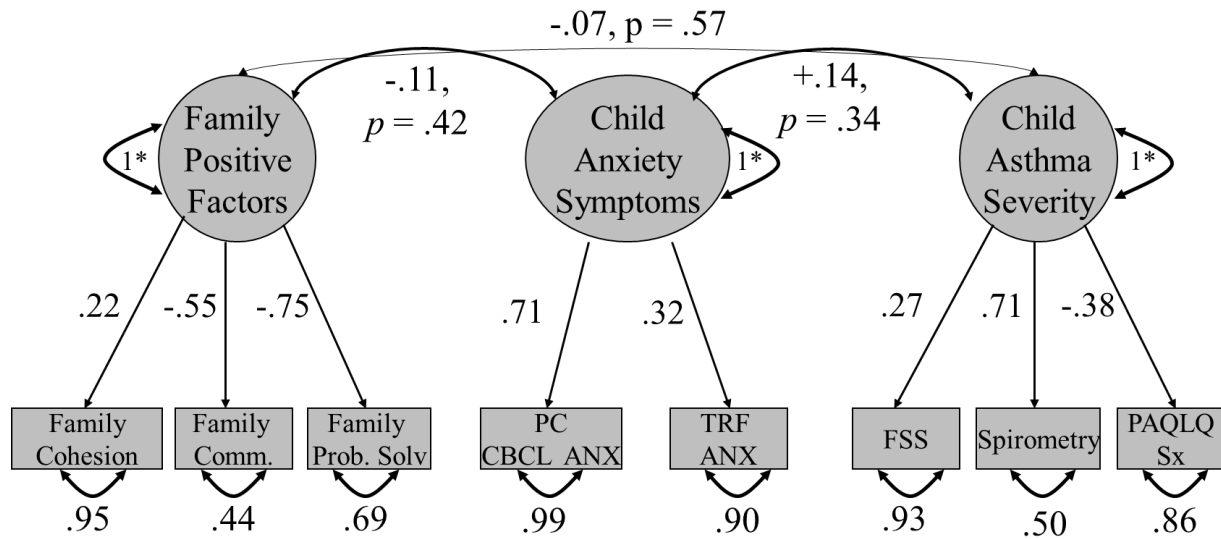


Figure 3. Confirmatory factor analysis for the model containing family positive factors, child anxiety symptoms, and child asthma severity $\chi^2 (17, n=215) = 22.50, p = .17$; RMSEA = .04_(.000-.079); CFI = 0.92; TLI/NNFI = 0.87; All loadings are significant at $p < .05$;

Note. Family Comm. And Family Prob. Solv. = Family communication and problem solving scales on Family Assessment Device; PC CBCL ANX = Primary caregiver reported anxiety syndrome score of the Child Behavior Checklist; TRF ANX = Teacher Report Form anxiety score; FSS = Functional Symptom Severity; PAQLQ Sx = Child ratings on Pediatric Asthma Quality of Life Symptom Domain.

Structural equation modeling did not support the anxiety hypothesized mediation pathway, with no direct associations between child anxiety and family factors ($\beta = -.11, ns$) nor between child asthma severity and family factors ($\beta = -.06, ns$). There were also no direct

associations between child anxiety and asthma symptom severity ($\beta = .14, ns$). Bootstrapping analyses, with 5000 samples, confirmed the absence of a significant mediation pathway, with a point estimate of $-.01$ and 95 % CI of -0.289 to 0.026 .

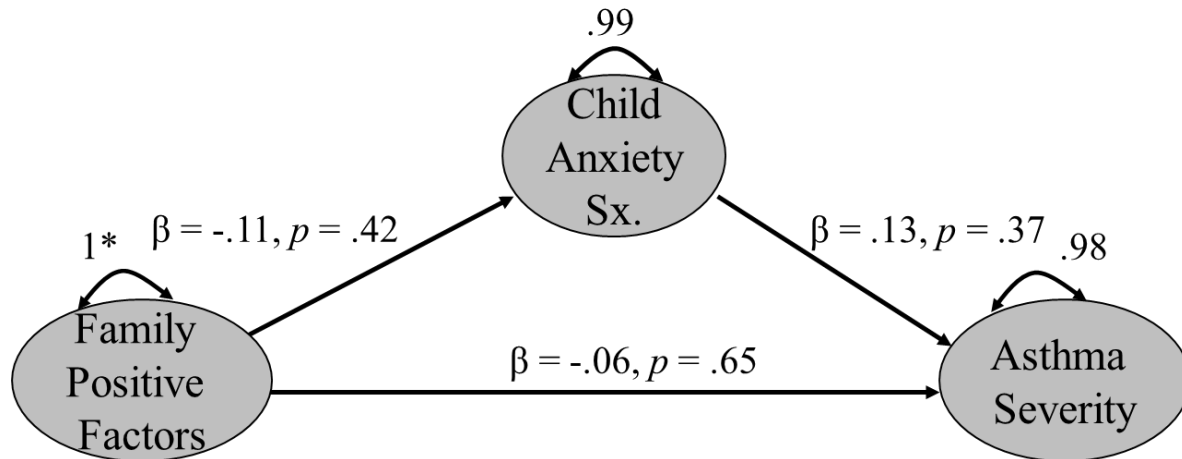


Figure 4. Structural equation model of the non-significant indirect relationship between family positive factors and asthma severity via child anxiety symptoms; $\chi^2(23, n=215) = 23.60, p = .32$; RMSEA = $.04$ ($.000-.075$) CFI = 0.96 ; TLI/NNFI = 0.94 ; Values reported are all standardized; Ab = $-.01$; 95 % CI ($-0.289, 0.026$).

Discussion

The current study examined associations between the positive family factors of communication, cohesion, and problem-solving abilities and child asthma outcomes. Child asthma outcomes encompassed measures of objective lung functioning, parental perceptions of child symptom severity, and child self-reported asthma quality of life. This study also examined the relationship of family factors and child internalizing symptoms (i.e., depression and anxiety). The bio-behavioral link between these internalizing symptoms and child asthma severity was also analyzed. Finally, child anxiety and depression symptoms were tested as potential mediators of the relationship between positive family factors and asthma outcomes.

Family Factors, Child Depression, and Asthma

Family functioning, represented by cohesion, communication, and problem solving abilities, was inversely related to child depression symptoms, confirming the important role families play for children's psychological and physiological adjustment to chronic health conditions (Reichenberg & Anders, 2005). These findings replicate, in a pediatric asthma population, links between family characteristics and child depression that have been found in general child literature (Chariello & Orvaschel, 1995). The way families openly communicate, unite in solving particular problems, and change in the face of threat to optimize family functioning are important for the way a child feels in the face of his/her asthma condition. As they begin to gain more responsibilities, children rely on their families to help them adjust to developmental shifts and asthma management changes (Kaugars et al., 2004; Wigfield et al., 1997). Thus, it is crucial for families to communicate well and provide children with extra support during this period (Fulligni & Eccles, 1993; Wigfield et al., 1997). A positive family environment in middle childhood may reduce the likelihood of developing clinical depression symptoms later on (Keiner et al., 1987). Therefore, it is important for physicians and pulmonologists to be cognizant of and emphasize these associations to families to prevent the high prevalence rate of depression symptoms in pediatric asthma populations.

Additionally, there was a significant direct association between child depression symptoms and child asthma severity. Children with greater depression symptoms had a greater likelihood of having a poorer asthma prognosis. The way in which depression is thought to influence an individual's asthma severity is through its effect on the autonomic nervous system (Miller & Wood, 1997). When children are depressed, they are more likely to respond to environmental stressors via parasympathetic nervous system's activation; consequently, with the domination of the PNS, they are more likely to have constricted airways and greater mucus

secretion (Van der Velden & Hulsmann, 1999). It is important to bear in mind these bio-behavioral associations when treating children with asthma; it may be beneficial for children with these comorbid mental health symptoms to receive evidence-based therapy in addition to their asthma medication regimen.

Whereas family functioning did not directly relate to child asthma symptoms, depression acted as a mechanism through which these variables indirectly related to each other in our sample. Children from families who were more open, cooperative, and flexible had less depression symptoms; in turn, these fewer depression symptoms were associated with better asthma status. This path model held true even after controlling for the influences of child age, sex, and medication adherence. Findings from this study support the Bio-behavioral Family Model (BBFM), which posits that a family influences asthma severity via its effects on a child's bio-behavioral reactivity (Wood et al., 2006). Children may feel confused and insecure if the family is not united or lacks effective communication methods; consequently, their internalizing symptoms may influence the progression of their disease. These findings extend past research by jointly assessing family's effects on subjective and objective measures of asthma severity; additionally, findings emphasize the importance of studying families comprehensively when examining relationships to child emotional and physical well-being.

Family Factors, Child Anxiety, and Asthma

Better family functioning is associated with fewer child anxiety symptoms in general child literature (Bogels & Brechman-Toussaint, 2006). Negative family emotional climate has also been linked to child anxiety in pediatric asthma contexts (Wood et al., 2006). However, we did not find a significant relationship between positive family factors and asthma symptoms in our sample. In this study, families who were more cohesive, openly communicative, and

adaptable did not have children who were less anxious. This lack of a significant relationship may be explained by several study factors. Firstly, a large proportion of the children in our sample did not report elevated anxiety symptoms in comparison to their same-aged peers. While robust SEM estimation methods (i.e., MLR) continue to provide accurate relationship statistics regardless of skewness, the narrow variability in anxiety symptoms across our small sample may be limiting our findings; this variability limits the anxiety pathway to a greater extent than the depression pathway as it is hypothesized to have a smaller effect on child asthma severity (Wood et al., 2006). Additionally, older children are better reporters of their anxiety symptoms than their caregivers and teachers; given the absence of child ratings of their own psychopathology in our sample, we may have an underrepresentation of symptoms experienced. Finally, it may be that children with asthma who experience poor family functioning develop specific types of anxiety, such as separation anxiety or panic attacks, that may not be accurately captured in an overall measure of child anxiety (Fiese et al., 2010; Goodwin, Fergusson, & Horwood, 2004). It is important to examine whether these associations would exist for different types of anxiety in child asthma settings.

This study also considered the relationship between child anxiety symptoms and asthma severity. As hypothesized, there was no direct link between child anxiety and asthma severity. Results indicate that anxiety, being primarily associated with the sympathetic nervous system, may not be associated with asthma severity to the same extent as depression. Again, these findings must be interpreted with caution, given the positively skewed nature of anxiety symptoms in our population. However, our findings concur with past studies that have failed to identify a relationship between anxiety and objective measures of asthma severity (Stephoe & Vogelee, 1992; Wamboldt et al., 1998). It may be that children with anxiety symptoms report

increased symptoms of asthma severity, but the associations fail to exist when these symptoms are paired with objective measures of lung functioning. Anxiety somatic symptoms, such as shortness of breath, may mimic those of asthma symptoms and allow individuals to misattribute their bodily responses to their asthma condition (Peters & Fritz, 2011). Results highlight the importance of incorporating objective measures in studies evaluating pediatric asthma severity.

The mediation role of child anxiety in the relationship between family factors and child asthma symptoms was also examined. Again, as hypothesized in Miller's ANS dysregulation theory, asthma did not play a significant role in linking family functioning to child asthma severity. This non-significant relationship may be associated with the restricted range of anxiety symptoms in this population. Future studies should evaluate whether associations between family factors, asthma, and anxiety exist for children diagnosed with anxiety disorders; whereas slightly elevated symptoms may be manageable, when these anxiety symptoms increase in severity, they may play a more significant role in children's asthma progression. The lack of significant mediation effects could also be due to the failure to examine anxiety symptoms of different disorders as mediators; for example, the way in which separation anxiety symptoms affects children with asthma may be different from that in which social anxiety symptoms operate. Additionally, because we only have parent and teacher reports of child anxiety, these perceptions may be somewhat different from the child's own experiences of his/her emotional well-being. Future studies should try to replicate these findings with the incorporation of child reports of anxiety. Finally, studies should examine the effects of parental depression and asthma symptoms on these associations; because parental symptoms may influence child symptomology and family functioning, it is important to see how these relationships exist at different levels of parental psychopathology (Feldman et al., 2013).

Research thus far has focused on examining the interrelationships among family factors, internalizing symptoms, and asthma severity; however, few studies have considered separate pathways for child anxiety and depressive symptoms in these associations (McQuaid et al., 2001). Our findings suggest that there are different roles for these internalizing symptoms in linking the relationship between family factors and child pulmonary functioning. Our findings are consistent with past literature examining the associations between family conflict and child outcomes; whereas depression symptoms did mediate the relationship between family functioning and child asthma symptoms, anxiety symptoms did not (Wood et al., 2006).

Strengths

To our knowledge, this is the first study that seeks to examine the bio-behavioral pathways of influence for the positive family factors of communication, cohesion, and problem-solving abilities. Given the importance of these characteristics for the optimal functioning of a family unit, understanding their links to asthma psychological and physical outcomes is crucial (Olson, 2000). Additionally, our study examined these mediational pathways using a multi-informant, multi-method comprehensive approach, allowing for the integration of information from multiple sources. For example, the combination of observational methods and subjective ratings of family functioning offers a more accurate view of the family and minimizes biases that may result from single measures; similarly, incorporating parent and child perceptions, in addition to spirometry results, into asthma severity measures allows a complete view of a child's illness. Finally, our study examines the distinct mediation pathways of anxiety and depression in the relationship between family functioning and disease progression. Our findings provide insight into how these internalizing symptoms may influence disease progression in different ways.

Limitations

This study's findings must be taken with caution, in light of some methodological limitations. Firstly, this study cannot infer causality or rule out other models, given its cross-sectional design. For example, our findings can also be explained by an alternative model in which child asthma severity influences family functioning via its effects on child depressive symptoms; without a longitudinal design, this unidirectional pathway of effect cannot be established. Additionally, while we used a multi-rater, multi-method design, we may not have completely captured the true nature of children's internalizing symptoms without obtaining their own subjective ratings. Also, the study's sample size did not provide adequate statistical power for us to control for anxiety symptoms in our depression mediation model. Without doing so, we cannot infer whether depression predicts child asthma symptoms over and above the effects of anxiety symptoms. Findings of this study require replication with a larger, more diverse sample to ensure greater variability in internalizing symptoms, family functioning, and asthma severity.

Recommendations and Future Directions

The current study's findings offer preliminary evidence for the relationship between positive family factors (i.e., communication, cohesion, and problem solving abilities) and child asthma severity via child depressive symptoms; these associations hold true even when controlling for adherence in the model. In the future, studies should further examine the psychobiological mechanism of effect, for example by measuring differences in acetylcholine (Ach) among these children. By incorporating these biological markers into a longitudinal design, directionality and mechanisms of effect can be established. Future studies should also include child ratings of internalizing symptoms in addition to those of their primary caregivers and teachers; as children are the most accurate reporters of their internalizing symptoms. Once

this directionality is established, studies should focus on developing, implementing, and testing family-focused treatments for children with worsened asthma symptoms; these treatments should have a particular emphasis on enhancing communication, increasing family unity, and building problem solving skills.

As for practical considerations, we suggest incorporating screening tools in pediatric asthma settings to help identify children with elevated depressive symptoms or struggling families. Administration of short questionnaires at routine pediatric visits could help identify children at elevated risk for worsened asthma symptoms. Additionally, we recommend providing family therapy services in conjunction with psychosocial interventions for children at risk for increased asthma morbidity. Allowing families to build upon their communication, cohesion, and problem-solving skills may help their children's psychological and physical asthma outcomes. It may also be helpful to provide psychoeducation to help children and their families accurately distinguish between symptoms of anxiety and of asthma; doing so may challenge their cognitive beliefs of their children's symptoms being solely attributable to their poor asthma condition. Finally, pediatricians are encouraged to share knowledge pertaining to bio-behavioral associations with families in order to show how environmental stressors can ultimately affect children's asthma progression.

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Vita

Nour Al Ghriwati was born on September 11, 1991, in Damascus, Syria. She received her Bachelor of Arts in biological sciences and psychology from the University of Maryland, Baltimore County, and she graduated summa cum laude in 2014. Prior to graduating, Nour spent one year as an undergraduate research assistant at UMBC's Pediatric Psychology lab and at Johns Hopkins Bayview Medical Center. She began the Developmental/Clinical Doctoral program at Virginia Commonwealth University in August 2014.