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Effects of Cumulative Risk on Asthma Outcomes in Urban Children and Adolescents

Samantha A. Miadich
Virginia Commonwealth University

EFFECTS OF CUMULATIVE RISK ON ASTHMA OUTCOMES IN URBAN CHILDREN AND ADOLESCENTS

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

By: SAMANTHA A. MIADICH
B.A., University of Dayton, May 2008
M.A., Ball State University, July 2010

Director: Robin S. Everhart, Ph.D.
Assistant Professor
Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
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Abstract

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Pediatric asthma disproportionately affects racial/ethnic minority children and children living in low-income, urban areas. Many families living in low-income, urban areas experience a number of stressors that can place children/adolescents at risk for worse asthma outcomes. This study examined the impact of a cumulative risk model of stressors (e.g., ED visits, quick-relief medication use, lung function, asthma control, QOL) in urban children (7-12 years) with persistent asthma. This study further aimed to examine both the original cumulative risk model and an adolescent-specific cumulative risk model as predictors of asthma outcomes in a sample of 60 adolescents (13-17 years). Asthma-related caregiver support was examined as a potential buffer in the association between stress and asthma outcomes. Secondary data analyses were completed on sixty-one caregiver and child dyads (7-12 years old). Data were collected from a separate sample of 60 urban families of adolescents with asthma (13-17 years old). The two cohorts were also combined for analyses.

The original cumulative risk model developed for the younger children (7-12 years) was a predictor of child QOL in the younger cohort, and QOL and asthma control in the adolescent cohort. However, this finding in the younger cohort (7-12 years) was not supported in pooled

data analyses. The original cumulative risk model predicted QOL, asthma control, and quick-relief medication use in the combined cohort analyses (children 7-17 years). The adolescent-specific cumulative risk model was a significant predictor of adolescent QOL and asthma control. Asthma-related caregiver support was only a significant moderator of the association between cumulative risk and asthma control among adolescents. Child age did not moderate associations between cumulative risk and asthma outcomes in the combined cohort. Overall, findings suggest that the accumulation of stress can have a negative impact on asthma outcomes, especially for urban adolescents with asthma. Further research is needed to determine the most central sources of stress that urban school-aged children with asthma experience and to replicate findings for adolescent with asthma. The buffering role of asthma-related caregiver support in the association between cumulative stress and asthma outcomes needs to be examined further in children and adolescents with asthma.

Introduction

Pediatric asthma is a major public health concern that disproportionately affects minority children and children living in low-income, urban areas. Approximately 7 million children or every 1 in 11 children have asthma (Center for Disease Control [CDC], 2012). From 2001 to 2009, the greatest increase in asthma prevalence was in Black boys at an almost 50% increase (CDC, 2012). Black children are two times more likely to have asthma than White children (CDC, 2012). Black and Latino children are more frequently treated in the emergency department (ED) than White children (CDC, 2012). As will be described in detail, racial and ethnic minority children living in urban settings experience even higher rates of asthma morbidity. Moreover, costs of asthma care continues to rise; research is needed that addresses increased asthma morbidity, mortality, and healthcare utilization among low-income, urban children with asthma.

Asthma prevalence and mortality rates are higher among adolescents than younger children (CDC, 2006). The disparity in mortality rates between Black and White children has also increased (CDC, 2006). Poorly controlled asthma in children of all ages contributes to increased healthcare costs, more missed days of school, and overall poorer quality of life (QOL). Recent reports suggest that approximately 38.4% of children in the United States have uncontrolled asthma, with rates in Virginia being slightly higher than the national average of 39.7% (CDC, 2011). Despite efforts to reduce ED visits and hospitalizations, children still experience asthma attacks and ED use for asthma remains high (CDC, 2006). Therefore, factors outside routine care and medications are likely impacting child asthma care. Given the increased risk for poorer asthma outcomes among urban, racial/ethnic minority children, the current study included a predominately low-income, Black sample of families from the urban Richmond, Virginia area.

Ecobiodevelopmental and Toxic Stress Theoretical Frameworks

This document first discusses the ecobiodevelopmental and toxic stress frameworks that provide the theoretical background for examining multiple sources of stress among children and adolescents with asthma.

Ecobiodevelopmental Framework. The ecobiodevelopmental framework, developed by the American Academy of Pediatrics, can be used to explain how early life experiences and environmental influences can affect a person's lifelong health (Shonkoff et al., 2012) and is also the basis of the toxic stress framework. The ecobiodevelopmental framework involves the overlap and interplay between biology (psychological adaptions and disruptions), ecology (societal and physical environment), and development and health (learning behavior and physical and mental well-being). This model focuses on nature and nurture as simultaneously interacting compared with previous models that place one against the other (Sameroff, 2010). In addition, this model suggests that there is an interaction between biology and ecology that influences development and health outcomes. The field of epigenetics has provided evidence for the ecobiodevelopmental framework (Shonkoff et al., 2012). Epigenetics studies how external factors affect gene expression without the altering of the actual DNA sequences, thus examining gene-environment interactions (Shonkoff et al., 2012).

Shonkoff and colleagues (2012) focus on the concept that positive early life experiences lay the groundwork for healthy brain development and the ability to learn and acquire new skills. Even prenatal experiences (e.g., maternal stress in utero) can affect development and health across the life span. The Adverse Childhood Experiences (ACES) study by Felitti and colleagues (1998) highlighted the impact that adversity in childhood can have on an individual's health. In their study, 9,508 adults completed a questionnaire that assessed multiple domains of adverse

childhood experiences including whether there was criminal behavior in the home, if their mother was treated violently, household members with mental illness or with substance abuse problems, and psychological, physical, and sexual abuse. Overall, Felitti and colleagues (1998) found that half the sample (52%) endorsed at least one adverse childhood experience and that 6.2% endorsed having experienced four or more adverse events. The authors found a dose response curve in the association between range of childhood adverse events and multiple risk factors for major causes of death (e.g., smoking, obesity, depression, no physical activity). Specific diseases including chronic lung disease were linked to the range of adverse events experienced by the individual. Findings from this study highlight how prevalent adverse childhood events are experienced and suggest the importance of including them and their impact on health outcomes in future studies.

In addition, Shonkoff and colleagues (2012) indicated that it is of the utmost importance for societies to invest in childhood development and fostering healthy children for the future. The authors point out that advances in developmental neuroscience, molecular biology, epigenetics, psychology, economics, and epidemiology are beginning to converge to explain the impact of early negative life events. In reviewing previous research, the authors concluded that early adverse experiences can lead to physiological changes in the stress response with lasting effects on the brain, cardiovascular, and immune systems, all of which are developing throughout childhood. These changes can carry over into adulthood and impair the person's health across their lifespan. Shonkoff and colleagues (2012) suggested that the ecobiodevelopmental framework takes into consideration that early life experiences impact disease courses later in life and can guide the development of more influential interventions.

Along with their report on the ecobiodevelopmental framework, the American Academy of Pediatrics in 2012 released a policy statement regarding the long-term impact of toxic stress and early adversity (Garner et al., 2012). This policy statement was to inform pediatric providers about the advances being made in understanding early adverse events and their effects on health. In addition, the policy statement advised that providers need to be a partner in identifying developmental concerns and aid in strategies to reduce the causes of toxic stress in children. The American Academy of Pediatrics (2012) stated that providers should adopt the ecobiodevelopmental framework as the means for understanding the social, economic, and behavioral components of health disparities. In addition, they stated that toxic stress and the trajectory of adverse childhood events should be incorporated in training for healthcare providers. Thus, this policy statement stressed the importance of this framework and the integration of toxic stress knowledge into health care.

Toxic Stress Framework. Children with asthma from low-income, urban families are more likely to experience higher levels of toxic stress, which can further compound a child's risk for poor asthma outcomes. The toxic stress literature builds from the ecobiodevelopmental framework previously discussed. Toxic stress is described as a continual activation of the stress response (multiple stressors) without a sufficient buffer against stress (e.g., caregiver support) in children (Johnson et al., 2013). Poverty, family violence, community violence, and single parent homes can create a toxic stress environment for children (American Academy of Pediatrics; Johnson et al., 2013); these factors are more commonly experienced by families living in low-income, urban areas. Given that pediatric asthma is managed within families, chronic stressors present in urban settings may make it difficult for caregivers to divert appropriate time and support to their child's asthma and its daily management (Bellin et al., 2014).

Research has focused predominately on children experiencing toxic stress early in life and the development of negative health outcomes later in life as adults (Shonkoff et al., 2012). Development of health conditions is seen through dysregulation of the hypothalamic-pituitary adrenal (HPA) axis and the immune system due to prolonged exposure to stress without sufficient buffering that mitigates the negative impact of stress on the immune system (Shonkoff et al., 2012; Johnson et al., 2013). Dysregulation of the HPA axis and immune system can have a lasting impact on the individual, affecting their health throughout their life. The current study aimed to understand the impact of toxic stress on children already diagnosed with asthma.

As discussed by Johnson and colleagues (2013), prenatal stress may affect the development of the child and their immune system, which highlights the importance of early caregiving and its effect on development. The authors suggested that children who receive inadequate caregiving make adaptions (e.g. gene expression) to their caregiving environment for survival and the long-term effects of such adaptations may be detrimental (e.g., HPA dysregulation, impaired immune systems). Specifically, Johnson and colleagues (2013) stated, "Young children cared for by individuals who are available and responsive to their emotional and material needs develop immune systems that are better equipped to deal with initial exposures to infections and to keep dormant infections in check over time" (p. 323). The authors reported that caregiving, or the lack of, can have long-term effects on a child's immune system. Having someone who is a consistent source of nurturance can be a factor that leads to resilience in childhood. Along with Shonkoff and colleagues (2012), Johnson and colleagues (2013) stated that pediatricians could use their position to inform people (e.g., patients, policy makers) about toxic stress and the detrimental effects that can occur.

Shonkoff and colleagues (2012) reported that although educational interventions are important for child development, some interventions need to focus on reducing adversity. New strategies and interventions are needed to help minimize the number of adverse events children experience. The first step in this process is identifying the major sources of stress and negative events in a community. The current study assessed multiple stressors children and adolescents with asthma may experience in the urban Richmond, VA area. Stemming from the toxic stress framework, this study also investigated the buffering effect of caregiver asthma-related support on the association between cumulative risk and child/adolescent asthma outcomes.

Assessing Multiple Sources of Stress

Children and adolescents with asthma living in low income, urban areas may face a variety of stressors at multiple levels (e.g., individual, family, neighborhood/community), which can complicate their disease outcomes. Wright and Subramanian (2007) completed a review of the literature on asthma disparities. The authors reported that a large portion of research in this area is focused on individual-level risk factors and emphasized the need for a multi-level approach to better understand asthma disparities. Wright and Subramanian (2007) highlighted that rates of asthma are increasing the most among racial/ethnic minority children living in urban areas and among children in poverty. Additionally, asthma death rates have been increasing and these deaths are more centralized in low-SES and urban communities. The authors reported that racial/ethnic differences in asthma are independent of socioeconomic status (SES). However, not all studies are in agreement with this finding (Aligne, Auinger, Byrd, & Weitzman, 2000; Crain et al., 1994) and more research is needed that considers the contextual factors of asthma disparities.

In addition, Wright and Subramanian (2007) described the existing explanations for social disparities related to asthma. First, there are explanations related to exposures in the home and community, which include the hygiene hypothesis, research involving indoor allergens, and research involving air pollution and diesel particles. There are explanations for social disparities that also involve cigarette smoking and tobacco smoke exposure. Additionally, the authors called attention to the genetic factors related to asthma. However, understanding the genetic component of asthma will not help reduce disparities unless the social and environmental factors related to racial/ethnic and SES disparities are also understood. Therefore, Wright and Subramanian (2007) reported a need for a multilevel approach to understanding asthma disparities that included individual, family, and community factors. The authors highlighted the need to study the broader social contexts that might contribute to these disparities, which could include the community and neighborhood factors. The structure of the neighborhood, the neighborhood disadvantage level, and crime and violence are all possible influential factors that should be included in future work.

Given the above information from Wright and Subramanian (2007), a promising theoretical foundation for the current study includes Bronfenbrenner's Social-Ecological Theory (Bronfenbrenner, 1979). Previously discussed were the ecobiodevelopmental and toxic stress frameworks and how they explain negative health outcomes (American Academy of Pediatrics, 2012; Johnson et al., 2013). Bronfenbrenner's model is an established theory in developmental psychology that outlines how different levels of factors influence the developing child. This model provides the basis for why it is important to investigate the effects of multiple types of stressors on the individual child. Specifically, there are four levels within Bronfenbrenner's framework: the microsystem, the mesosystem, the exosystem, and the macrosystem. Each level can affect another in a bidirectional way to influence child development. At the center of this

model is the microsystem, which consists of the child's immediate environment. The microsystem may include immediate family members, peers, and school. Moving outward, next is the mesosystem, which consists of interactions between the child's different microsystems. For example, the child's parents and teachers may interact in regards to the child's asthma management and treatment. Next is the exosystem, which includes the neighborhood, community, and extended family influences on the child. This level may not include the child's direct involvement with any one person or group; however variables like community violence can still impact child asthma. Lastly, the outermost level is the macrosystem, which includes cultural beliefs and values and broader social contexts. Medication beliefs or complementary alternative medicine values held by the family can affect a child with asthma at this level in the model.

In Bronfenbrenner's model (1979) each level is a system that has an impact on the child at the center of the model. The influences are simultaneous and interact with one another to affect child development. Therefore, given the multi-level approach discussed by Wright and Subramanian (2007), as well as the adverse childhood experiences and toxic stress literature, Bronfenbrenner's theory is a strong initial foundation for studying the effects of multiple levels of stress on children and adolescents with asthma. All the frameworks discussed involve multiple components interacting with one another to affect development or health outcomes and are multi-systematic in nature. The current study assessed several stressors related to different levels (e.g., individual, family, neighborhood) of influence and how these stressors combined affect asthma outcomes.

Stressors Related to Asthma Outcomes in Children and Adolescents

In general, stress has been linked to asthma; higher levels of stress are associated with greater asthma morbidity (e.g., more symptoms, quick-relief medication use, ED visits). Neighborhood stressors including low socioeconomic status (SES) and community violence have been associated with greater asthma morbidity (e.g., hospitalizations, symptoms, severe episodes; Chen & Schreier, 2008). Family stressors, which include poor family functioning, poor relationship quality, caregiver stress, and caregiver mental health have also been associated with increased asthma morbidity (Chen & Schreier, 2008). In addition, the accumulation of multiple stressors has been linked to the development of asthma in children (Suglia et al., 2010). In the current study, a cumulative risk model of stress comprised of perceived discrimination, family functioning, caregiver stress, neighborhood stress, and poverty was tested; see Figure 1. These factors were selected based their relevance to a toxic stress framework in urban families and are embedded within the individual, family, and neighborhood levels. Each factor is described in the following section.

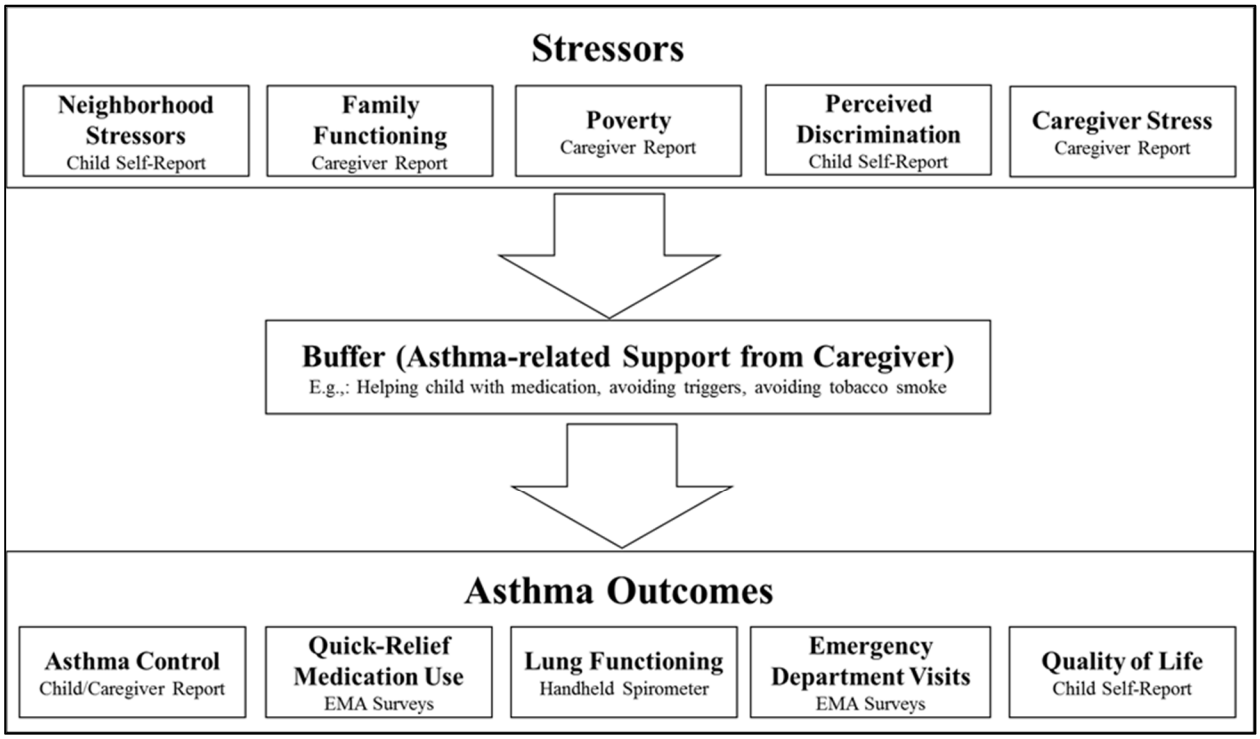

Figure 1. *Toxic stress theoretical framework of the current study. (American Academy of Pediatrics; Johnson et al., 2013).*

Individual Factors: Perceived Discrimination. Discrimination can be harmful to the health of all individuals. A meta-analysis conducted by Schmitt, Branscombe, Postmes, and Garcia (2014) found larger effect sizes for studies that investigated the role of perceived discrimination on psychological well-being in children compared to adults, such that perceived discrimination was associated with worse psychological well-being. Their findings were in concordance with another meta-analysis conducted by Lee and Ahn (2013) that found a greater impact of racial discrimination on psychological distress in children compared with adults. In the asthma literature, experiences with racism has been linked to adult-onset asthma among Black women (Coogan et al., 2014). The authors suggested the stress associated with discrimination was linked to the women developing asthma in adulthood.

Limited research has focused on discrimination in the pediatric asthma literature, and much of that research has focused on caregiver perceived discrimination. Koinis-Mitchell and colleagues (2007) assessed caregiver perceived discrimination, which was included in their cumulative risk model to predict asthma morbidity. Perceived discrimination was described as a psychological factor that could influence the management of a child's asthma within a family (Koinis-Mitchell et al., 2007). Discrimination can be a source of stress, especially among racial/ethnic minority families living in low-income urban areas.

Similarly, Astell-Burt and colleagues (2013) found an association between asthma prevalence and racial discrimination in a group of adolescents across all ethnic groups (i.e., White UK, Black Caribbean, Nigerian and Ghanaian, Other African, Indian, Pakistani and Bangladeshi, and mixed White/Black Caribbean). In the first wave of data collection adolescents

were 11 to 13 years old and in the second wave they were 14 to 16 years of age. Additionally, the authors found that racial discrimination exacerbated the association between pollution and risk of asthma; therefore perceived discrimination heightened the association between pollution and the risk of having asthma. The authors discussed that the psychosocial stress of perceived discrimination may be an underlying factor in this interaction. Findings suggest that interventions to reduce discrimination would not only have societal benefits, but also health benefits (Astell-Burt et al., 2013).

More recently, Thakur and colleagues (2017) specifically examined the association between perceived discrimination and asthma morbidity among Black and Latino youth. Participants were youth from two large ongoing studies from the continental United States (US) and Puerto Rico. Over 3,500 youth with and without asthma aged 8 to 21 years were included. Racial discrimination among Black youth was associated with increased odds of asthma prevalence and poorer asthma control. Thakur and colleagues (2017) also found a dose-response association between discrimination and asthma control: as the severity of perceived discrimination increased, so did poor asthma control. For Mexican American and other Latino youth, the association between perceived discrimination and asthma control depended on SES, with the association being stronger for youth from lower SES backgrounds. Findings suggest that perceived discrimination has negative consequences on health outcomes, specifically asthma control among youth. Thus, the current study included child perceived racial discrimination in the cumulative risk model predicting asthma outcomes.

Family Factors: Family Functioning and Caregiver Stress. Familial factors (e.g., family functioning, parent-child conflict) can also be a source of stress for children and adolescents with asthma. Given that pediatric asthma is managed within families, family

functioning may be an especially important predictor of child asthma outcomes. For instance, poor family functioning can affect the family's ability to manage a child's asthma and hinder adherence to treatment (Fiese & Everhart, 2006). Family daily management of asthma care, which includes ensuring the child took his/her medications properly, setting doctor appointments, and arranging for transportation, when needed, to attend appointments is essential. When there is less family cohesion and more turmoil, asthma management might decline, which can compromise the child's health (Fiese & Everhart, 2006).

In children with chronic health conditions, better family functioning has been related to positive psychological adjustment and well-being (Drotar, 1997). Specifically, greater family cohesion and supportive family relationships have been related to better psychological adjustment in children with a chronic illness (Drotar, 1997). Poorer family functioning has been found among families of children with asthma as compared with a control group of families of children without asthma (Ozkaya, Cetin, Ugurad, Samancı, 2010). Family stressors, including poor family functioning and quality of relationships, have been associated with increased asthma morbidity and poorer QOL in children with asthma (Chen & Schreier, 2008; Kaugars, Klinnert, & Bender, 2004). In addition, higher levels of cohesion and expressiveness, used to assess family functioning, have been related to better QOL in children with asthma (Crespo, Carona, Silva, Canavarro & Dattilio, 2011).

Wood, Miller, and Lehman (2015) conducted a review of the literature to enhance understanding of the intricate interplay between family variables and child asthma. The authors first described an overarching framework in which family relations/functioning and the child's asthma had a bidirectional association. Thus, not only do family relations/functioning affect the child's asthma, the child's asthma can also affect family functioning (as well as caregivers'

physical and mental health). Asthma itself can be seen as a stressor and impact the family. Family stress may affect child asthma by impairing disease management or though psychobiologic pathways. The psychobiologic pathways include gene-environment interactions, which are mediated by epigenetics, and transmission of vulnerability to stress from parent to child (Wood et al., 2015). However, inconsistent definition and measurement of parental stress, and the fact that it has been studied independent of family and social stressors, are current limitations to this line of research.

Wood and colleagues (2015) discussed that social factors, including poverty and disadvantaged social status, may influence asthma health disparities, even prenatally. In their review, Wood and colleagues discussed a socio-psycho-biological model developed by Wright (2011) that incorporated the effects of social influences on asthma. However, they stated there was a missing piece of the model in that it did not account for the effects of family functioning on the mother and child. Therefore, Wood and colleagues (2015) proposed the integration of the biobehavioral family model (BBFM; Wood, 1993; Wood et al., 2008) with Wright's (2011) model.

The BBFM is a multilevel systemic model that takes into account the child, the family, and parent-child relationships (Wood, 1993; Wood et al., 2008). This model can account for both protective and stressful effects, and posits that family variables can buffer effects of external stressors. There are four dimensions to the BBFM, which include: family emotional climate, parental relationship quality, parent-child relational security, and biobehavioral reactivity. Wood and colleagues (2015) specified that biobehavioral reactivity is the central dimension that links family emotional processes to biological and disease processes. Specifically, the authors defined biobehavioral reactivity as, "the degree or intensity with which an individual responds

physiologically, emotionally, and behaviorally to emotional challenge" (p. 382). Therefore, the integrated model (Wright-BBFM Integrated Heuristic Model) would be able to more fully explain the intricacies of the multilevel influences on the development of asthma. Specifically, the integrated model poses family functioning as a moderator between external stressors and physiological stress responses in the mother and child (e.g., HPA axis activity, immune function).

Kaugars, Klinnert, and Bender (2004) discussed two potential pathways in which family functioning may affect asthma outcomes: 1) through managing the child's asthma and 2) through physiological factors including the HPA axis and immune system, the autonomic nervous system, and symptom perception. First, family conflict can affect the family's ability to properly manage the child's asthma. In addition, poorer mental health in caregivers has been related to poor adherence in children with asthma (Bartlett et al., 2001). More conflict and more impaired psychological functioning may hinder how compliant families are to the child's medical regimen for their asthma. Family disorganization and disagreement over who is responsible for the child's asthma care can also impact asthma outcomes. Secondly, there are several possible physiological mechanisms in which family functioning may impact pediatric asthma outcomes. Emotional factors related to family functioning may impact asthma outcomes through the HPA axis and inflammation, the autonomic nervous system functioning, or through the accuracy with which the individual can perceive their symptoms. Family functioning may be an integral variable impacting disease outcomes in children with asthma.

Caregiver mental health and perceived stress can also impact child asthma outcomes. . Caregiver functioning and parent-child interactions within the family can affect disease outcomes in a bidirectional manner. Parent psychological functioning is one aspect that can

impact family functioning; poorer parental psychological functioning has been associated with worse asthma morbidity in children (Kaugars et al., 2004). Caregivers from inner-cities with poorer psychological functioning (e.g., worse mental health, more depressive symptoms) reported taking their children with asthma to the emergency department more frequently. Also, greater asthma symptoms were reported among children with caregivers with poorer psychological functioning. Additionally, higher levels of caregiver perceived stress and depressive symptoms have been associated with increased asthma functional morbidity (e.g., symptoms, activity limitations due to asthma) among children with asthma (Clawson, Borrelli, McQuaid, & Dunsiger, 2016). Maternal perceived stress has also been associated with recurrent wheeze among young children with asthma (Ramratnam et al., 2016). Therefore, findings suggest the stress that caregivers experience may negatively impact the care a child with asthma receives and lead to worse health outcomes.

In a review paper, Chen and Schreier (2008) discussed the literature on the impact the social environment may have on asthma. The authors defined the social environment as, "the connections a person has to a larger social community" (Chen & Schreier, 2008, p. 650) and divided the social environment into three levels: the family level, the peer level, and the neighborhood level, with the family being the most proximal level of influence. The authors argued that although other factors including environmental exposures and viral infections have been well established in the literature as contributing factors to asthma, the impact of social factors is less clear.

The quality of familial relationships has been linked to asthma morbidity and mortality (Chen and Schreier, 2008). Greater family dysfunction and more parent-child conflict have been related to poorer asthma outcomes, including more hospitalizations and even death from asthma

in previous research (Chen, Chim, Strunk, & Miller, 2007; Kaugars et al., 2004; Strunk, Mrazek, Fuhrmann, & LaBrecque, 1985). Different characteristics of parents including parent mental health (e.g., parental depression) and parental stress have been associated with poorer asthma outcomes. A longitudinal study by Weil and colleagues (1999) found children with caregivers who had mental health problems at baseline were approximately twice as likely to be hospitalized due to asthma at the 9 month follow-up assessment. Parental stress and mental health problems may affect the support a child receives, which can affect child asthma outcomes. For instance, Chen and colleagues (2007) found children who experience more asthma symptoms and worse daily lung function reported receiving less support from their parents. Chen and Schreier (2008) discussed that early experiences with family members may affect the biology of the child (e.g., stress responses), which may increase the child's vulnerability to developing asthma. In addition, familial relationships may impact the way the child perceives stress, the way they learn to cope, and/or the health behaviors they acquire, which if maladaptive, may be associated with worse asthma morbidity.

Given the importance of familial factors on child asthma outcomes, the current study incorporated factors at the level of the family. Specifically, general family functioning was assessed and included in the cumulative risk models. In addition, caregiver perceived stress was included given that caregiver mental health and stress can affect family functioning, which has implications for how a child's asthma is managed daily. Among adolescents, caregiver-child conflict was also assessed. As will be described later, relationships with parents has been reported as a main source of stress for adolescents.

Neighborhood/Environmental Factors: Neighborhood Stress and Poverty. The neighborhood and environment can also impact a child's asthma. Chen and Schreier (2008)

described the neighborhood level as the broadest level of an individual's social network, which includes influences from SES and exposure to violence. Although SES may have more tangible impacts on a variety of factors, including access to care, it can also impact a family's position within the community and how they may perceive themselves. Research has shown that lower SES is associated with poorer asthma outcomes (e.g., more symptoms, greater number of ED visits and hospitalizations; Chen & Schreier, 2008). Previous research has shown that neighborhood factors, including exposure to violence and higher crime rates, have been associated with worse asthma morbidity (Chen et al., 2007; Wright et al., 2004). Chen and Schreier (2008) suggested one reason why neighborhood factors affect a child's asthma may be due to the stress related to living in a low SES and/or high violence area. Limited resources (both material and coping mechanisms) and high demands may contribute to this stress. High demands can cause conflict in the family or unpredictability in schedules, in that children cannot rely on a typical routine; this can cause stress for children and impact their asthma, as well as create situations for inconsistent asthma care (Chen and Schreier, 2008). Additionally, these environmental effects can change behaviors or there can be different norms in low SES neighborhoods (e.g., smoking is more prevalent and acceptable). Chen and Schreier (2008) also discussed the biological mechanisms that may underlie these associations, specifically inflammation. Social influences at both the neighborhood and family level may cause stress, which can impact the child's inflammatory processes.

Suglia and colleagues (2010) sought to examine more closely the role of the physical environment of the home as a stressor and how the home environment in conjunction with other stressors can impact asthma outcomes. The authors specifically focused on three factors that define housing quality: housing deterioration, housing disarray, and housing hardship. The

authors used a public dataset, the Fragile Families and Child Well-Being Study, which follows a birth cohort sample from 20 cities around the U.S. Families completed a baseline session after delivery and follow-up assessments at 12 and 36 months. Suglia and colleagues' (2010) study was based upon the 2,013 families that completed the full 36 month assessment. The 36 month follow-up assessment was conducted at the families' homes and interviewers assessed the indoor housing conditions. Additionally, mothers were asked about housing hardships. Intimate partner violence was assessed at all three time points. At the 36 month follow-up, mothers were asked if their child had been diagnosed with asthma and if the child had an attack in the previous 12 months. Of the 2,013 children included in this study 52% were male and 54% were Black, 27% Hispanic, and 19% White. Ten percent of the children had been diagnosed with asthma and 17% of children had been exposed to maternal intimate partner violence at some point in their life. With respect to housing, 15% of children lived in deteriorated housing, 24% of families were experiencing housing hardships, and 35% of children lived in disarrayed housing.

Suglia and colleagues (2010) found an association between exposure to intimate partner violence and childhood asthma, which they stated was consistent with previous research. In addition, the authors found that housing disarray was associated with asthma prevalence, and this association was independent of intimate partner violence or any other confounder. Additionally, the authors found that children who had mothers experiencing intimate partner violence along with either housing disarray, housing deterioration, or housing hardships had greater odds of having asthma. Thus, for families experiencing intimate partner violence, poor housing conditions may worsen the stress of intimate partner violence on childhood asthma. The authors discussed two ways in which the findings could be interpreted. First, they discussed that housing conditions may be an indicator of environmental factors that are associated with childhood

asthma, such as dust or cockroaches. Second, they discussed that housing quality can be a direct source of stress rather than a proxy for other environmental stressors as mentioned in their first explanation. In the second explanation, housing quality was a direct source of stress that would affect the neuroendocrine and immune system, which impacts the development of asthma. The authors called for a need to focus on both social and environmental factors in intervention work with children with asthma.

In a longitudinal study, Bellin and colleagues (2014) investigated the effects of protective factors and community risk on healthcare utilization and asthma morbidity. Specifically, the authors examined the effects of social cohesion, informal social control, and community violence on asthma outcomes. Participants included 300 children ages 3 to 10 years old diagnosed with asthma and their primary caregivers that were part of a larger randomized control trial. Participants were recruited from two hospitals when the child was discharged from the emergency department. Caregivers were randomized into one of two groups: either a standard asthma education control or the behavioral/educational intervention group. Participants were assessed at baseline, 6 months, and 12 months. Caregivers were administered measures that assessed their neighborhood (i.e., an informal social control scale and social cohesion scale) and violence exposure. Additionally, caregivers reported on their child's asthma symptoms and healthcare utilization.

Of the 300 children, 59% were male and 96% were Black. Ninety-two percent of the caregivers were biological mothers. Fifty-four percent of caregivers were unemployed and 50% of households had an income of less than $10,000. Through latent growth curve modeling, Bellin and colleagues, (2014) found that higher caregiver reported exposure to violence predicted increased healthcare utilization right after the experience (baseline) and 2 months later.

Caregivers in this study reported high levels of violence exposure while also endorsing community protective factors. Additionally, both informal social control (e.g., intervening of neighbors for community goals) and social cohesion were found to moderate the association between violence and healthcare utilization. Bellin and colleagues (2014) highlighted that it may be helpful to focus on protective factors within the community when it comes to intervention work. The authors did not find any effect of community factors on child asthma symptoms or control, which they attributed to the data being caregiver self-report and a focus on the caregiver's experience with violence (and not the child's).

Koinis-Mitchell, Kopel, Salcedo, McCue, and McQuaid (2014) examined asthma outcomes related to both family and neighborhood stressors in a sample of 208 urban children 6 to 12 years of age (40% male) and their primary caregivers. The authors highlighted why neighborhoods and neighborhood stress need to be considered in pediatric asthma research. Reasons included previous findings that living in an urban area was linked to asthma regardless of race or household income and that higher rates of violence in the community have been associated with worse asthma outcomes (Aligne et al., 2000; Wright et al., 2004).

In Koinis-Mitchell and colleagues' (2014) study, 57% of families lived below the poverty line; approximately 53% of the sample reported being Latino, 22% Black, and about 23% non-Latino White. Families reported whether the child had an asthma controller medication and also completed the Asthma Control Test, the Asthma Assessment Form, and a neighborhood stress measure. Koinis-Mitchell and colleagues (2014) found that children endorsed a range of neighborhood stressors and highlighted the importance of using a child self-report of neighborhood stress versus a proxy parent report. They found that children who reported higher levels of neighborhood and family stress had poorer asthma control. Additionally, the authors

found that functional limitation due to asthma was related to neighborhood and family stress for children living below the poverty line. They stated that the cumulative impact of neighborhood stressors needs to be further researched to understand the underlying mechanisms between stressors and asthma morbidity.

Kopel and colleagues (2015) investigated the extent to which neighborhood safety, and specifically caregiver perceived neighborhood safety, was associated with asthma outcomes among inner-city school children. Participants were part of a larger study, the School Inner City Asthma Study (SICAS), which took place in a northeastern urban-city. This study included 219 children that were recruited from 25 inner-city schools. Children were ages 5 to 15 years old and had to have physician-diagnosed asthma. The primary predictor of the study was based off one dichotomous variable: "Is it safe to walk alone in the neighborhood at night?" Caregivers also completed questions about the child's asthma symptoms and asthma control. Other questions included how many missed days of school due to asthma and hospitalizations due to asthma the child had. In addition, at the baseline visit children completed a spirometry test to assess their lung function.

Children (52% female) had a mean age of 7.8 years old and were primarily Black (40.2%) or Hispanic (26.5%). Approximately 45% of families had a household income that was below $25,000. In 15% of homes there was not an adult that completed high school and there were no employed adults in 25% of homes. Forty-five percent of caregivers endorsed not feeling safe to walk alone at night in their neighborhood. Kopel and colleagues (2015) found a higher rate of uncontrolled asthma among children who had a caregiver that perceived their neighborhood to be unsafe, even after controlling for child age, race, gender, household income, tobacco smoke exposure, caregiver stress, and inhaled corticosteroid use. In addition, for

children who lived in neighborhoods that were perceived to be unsafe, the authors found higher rates of dyspnea (e.g., shortness of breath, labored breathing), night-time symptoms, need for quick-relief medication, and limitation in activity. There were no differences in spirometry ratings between the two groups. The authors attributed their findings to the possibility that children living in perceived unsafe neighborhoods were exposed to more violence, were of lower SES, had less access to healthcare and more stress, which may account for some of the worse asthma outcomes. Findings suggest that when clinicians are working with children with poorly controlled asthma, they should take into consideration the social context to help figure out reasons for increased symptoms.

In sum, the reviewed literature demonstrates that neighborhood factors have a significant impact on child asthma outcomes. The current study included an assessment of neighborhood stress as reported by the child or adolescent. In addition, whether families live in poverty was included in the cumulative risk models, as the material and social contexts of living in poverty can affect child/adolescent asthma outcomes.

Stressors Specific to Adolescents

In the current study, the previous literature on stressors associated with asthma outcomes was also applied to the adolescent cohort. The same cumulative risk model comprised of perceived discrimination, family functioning, caregiver stress, neighborhood stress, and poverty was tested in the adolescent cohort in addition to being tested in the younger cohort. Additionally, a second cumulative risk model was tested in the adolescent cohort. This second model (adolescent-specific) included poverty and neighborhood stress, as well as school related stress and relationship related stress (e.g., parent/caregiver, peer). These new stressors were

chosen based on their relevance to adolescents with asthma and are reviewed in the following section.

Adolescents with asthma face a unique set of stressors that may place them at higher risk for asthma morbidity (Bitsko, Everhart, & Rubin, 2013). Adolescence is a time of rapid change and sources of stress may differ from younger children. Specifically, adolescents may experience stress related to school and relationships (e.g., parent/caregiver, peer, romantic) more frequently (Rew et al., 2014; LaRue & Herrman, 2008). Urban adolescents have also reported neighborhood stress and poverty to be other prominent sources of stress (LaRue & Herrman, 2008). As children mature into adolescence, they begin to take over responsibility for their daily asthma care. Interventions involving parental assistance have proven effective, suggesting caregiver involvement in disease management is still central in adolescence to some degree (Duncan et al., 2013). A toxic stress framework may continue to serve as a useful model for understanding how stress influences asthma morbidity in urban adolescents. However, the degree to which asthma-related caregiver support buffers adverse asthma outcomes may differ between children and adolescents. Such information would be useful in designing developmentally appropriate treatment plans for adolescents with asthma that consider a toxic stress framework. Additionally, given that adolescents in the current study were from an urban area and exposed to an increased number of stressors, a cumulative risk model may more accurately explain the association between stress and asthma outcomes.

School and Relationship (Caregiver and Peer) Stressors. In their qualitative study that included seventeen focus groups with high-risk youth, LaRue and Herrman (2008) reported school as a main source of stress for adolescents regardless of age, gender, or grade level. This study included 120 teens ages 12 to 19 years. Seventy-two of the teens were female and 68%

were Black. The most discussed stressor during the focus groups was school, which included pressure to receive good grades, graduate, and get into college.

Another prominent stressor discussed by teens in LaRue and Herrman's (2008) focus groups was relationships, which included both peer and romantic relationships. Female participants discussed cheating and lying when it came to sources of stress in romantic relationships; male participants mentioned their romantic partners "got on their nerves" or were "in the way". The other common source of stress discussed by teens was their parents. Specifically, the teens discussed that their parents put pressure on them or had high demands. Teens in LaRue and Herrman's (2008) focus groups also discussed the interplay of stressors. For example, teens mentioned being tired from school and then going home where they had to complete tasks from their parents. In doing so, the teens had to put off school work, which added to their stress. Teens also reported they were distracted by peers and romantic partners. Findings from this study suggest that stressors adolescents face can build upon one another such that school stress, parental pressure, and peer pressure demands can accumulate into higher overall stress for adolescents.

In their mixed methods longitudinal study, Rew, Tyler, Fredland, and Hannah (2012) assessed adolescents' main concerns/sources of stress over their four years of high school. There were 216 students (142 female; 45% Hispanic) that completed the study every year during high school. As part of the study, adolescents were prompted with a question inquiring about their main worry/concern and were asked to respond in a free text format. The free text responses were coded and categorized; the frequency of each category of stress was determined. Rew and colleagues (2012) identified 21 categories based on the adolescents' responses. The most

frequently mentioned categories included education/school, relationships (with parents, peers, and romantic partners), expectations (specifically from parents), and the future.

Rew and colleagues (2012) found that each year anywhere from 41.0% to 51.9% of adolescents listed a school related issue as their main worry/concern. This source of stress included concerns about current schoolwork and also about college applications and being able to get accepted somewhere, which included worries about grades. Their findings on school as a main worry/concern are consistent with Stuart's (2006) findings that a main stressor for high school students is schoolwork. Stuart (2006) found that while students endorsed a number of stressors, school was mentioned most frequently and stress related to parents was the next most frequently mentioned.

Social relationships were also a common main source of worry/concern expressed by adolescents in Rew and colleagues' (2012) study. Social relationships constituted relationships with parents and peers. Adolescents reported feeling pressured by parents and strain on their relationships with their parents. In addition, adolescents had concerns with peers, which included "getting involved with the wrong crowd". These findings were also consistent with Stuart's (2006) study in that stress related to pressure from parents was the second highest endorsed stressor behind stress related to schoolwork.

In addition to adolescents reporting relationships with their parents as a major source of stress, this conflict can have an impact on their health, specifically asthma outcomes. Previous research has found an association between harsh parent-child interactions and asthma symptoms as well as the downregulation of anti-inflammatory responses (i.e., anti-inflammatory gene expression) in 10 to 20 year old children/adolescents (Ehrlich, Miller, & Chen, 2015). Ehrlich and colleagues (2015) had child and caregiver dyads complete a conflict task that was coded for

harsh and supportive interactions. Harsh interactions were positively associated with asthma symptom intensity. Also, research has found a link between maternal responsiveness and stimulation of cytokines that lead to inflammation in children/adolescents ages 10 to 17 with asthma (Tobin et al., 2015b). Greater rates of maternal responsiveness were associated with reduced stimulation of cytokines that lead to inflammation. Therefore, there may be biological underpinnings that can aid in explaining the association between caregiver-adolescent relationships and asthma outcomes.

Previous research involving objective assessments of caregiver-child conflict has also found an association between conflict and asthma symptoms among children/adolescents 10 to 17 years of age (Tobin et al., 2015a). Through use of novel technology (Electronically Activated Recorder [EAR]), Tobin and colleagues (2015a) were able to code audio recorded interactions over a 4 day period between 54 children and their primary caregivers to provide a more objective assessment of the child-caregiver relationship. This objective assessment had similar findings to self-report studies in which more conflict was associated with worse outcomes. In sum, the previous research suggests that not only is caregiver-adolescent relationships a source of stress for adolescents, but it may also impact their asthma outcomes. The current study assessed both stress related to school and stress related to relationships. For relationship stress, adolescents answered questions related to parent-child conflict and stress related to peer pressure.

Asthma Care among Adolescents. Although adolescents may be experiencing a broader range of stressors, they might also be given responsibility for their asthma care prematurely, which can complicate asthma management and potentially lead to worse outcomes. To understand this issue better, Bruzzese and colleagues (2012) conducted a study examining self-management behaviors of young adolescents with asthma. The authors included Hispanic and

Black children/young adolescents and considered racial/ethnic differences in asthma self-management behaviors. The sample consisted of 317 participants (187 Hispanic and 130 Black) recruited from public schools in New York that served low-income, children. All children were diagnosed with asthma and had uncontrolled asthma. They ranged in age from 11 to 14 years of age, with a mean of 12.71 years old. Ninety percent of caregivers were biological or adoptive mothers. Fifty-two percent of the sample was unemployed and 35% had less than a high school education. The children in this study were part of a larger randomized control study.

Overall, Bruzzese and colleagues (2012) found that the prevention and management behaviors of adolescents were suboptimal. Adolescents endorsed engaging in 6.19 of the nine prevention steps (e.g., taking daily medications, taking medication before exposure to triggers, asking for help, regular doctor visits). However, there was large variability in the implementation of each step. Adolescents also endorsed taking several of the management steps once symptoms began, including taking medication, staying clam, resting, and getting away from triggers. Older children were less likely to pay attention to how they felt, observe or track their symptoms, and ask for help when they were experiencing symptoms. Boys were more likely to take their daily preventive medication and to take medication before exposure to a trigger. In addition, the authors found that Black children were less likely to take their prescribed medication in cold weather.

Bruzzese and colleagues (2012) reported that although many adolescents (half the sample) reported taking prevention steps, they did not do this on a regular basis. While experiencing symptoms, many adolescents did not seek help or medical attention, which, according to the authors, may suggest inadequate supervision by caregivers. The authors stated that the adolescents' management reflected a reactive management style, which can lead to

worse outcomes. Bruzzese and colleagues (2012) also found that adolescents perceived their parents to have more responsibility over their asthma care and they did not feel responsible for caring for their asthma themselves. The authors suggested that some adolescents are given responsibility over their asthma prematurely and that adolescents may need assistance in obtaining the required skills to manage their asthma.

Findings from this study highlight the importance of assessing the support adolescents are receiving from their caregivers for asthma care. Thus, the current study assessed both the adolescent's perception of the care they receive from their primary caregiver related to their asthma, as well the caregiver's perception of their involvement in their adolescent's asthma care. Doing so allowed for better insight into the different perceptions of asthma-related care from both adolescents and caregivers.

Cumulative Risk

In considering how risk may impact asthma outcomes in urban children and adolescents, this study used a cumulative risk model to investigate the effects of stress on asthma outcomes. Cumulative risk conceptualizes the impact of multiple, overlapping risk factors on adverse outcomes rather than the individual impact of each factor alone (Evans & English, 2002; Koinis-Mitchell et al., 2007). A cumulative risk model is often a more accurate depiction of how families experience risk factors in that risk exposures do not occur in isolation (Everhart et al., 2008). Cumulative risk models take into account the number of stressors compared to a specific type of exposure. Further, cumulative risk models might explain more variance in the outcome than any one factor (Sameroff et al., 1993). Given that families in the current study were from an urban area and exposed to many overlapping stressors (e.g., poverty, neighborhood stress), a

cumulative risk model is likely a more accurate representation of a family's experience with daily stress.

Several landmark studies by Evans and colleagues have highlighted the importance of studying cumulative risk and its impact on children and their health. Evans and English (2002) examined the effect that poverty may have on socioemotional adjustment in children by means of exposure to many physical and psychosocial stressors. The authors point to the fact that there are still some large gaps in the literature when it comes to understanding the day to day conditions children in poverty face, including children from rural areas that live in poverty. Although their study looked at children in rural settings, the findings provide substance to the argument that children living in poverty experience multiple physical and psychosocial sources of stress in their everyday lives. Evans and English (2002) extended the literature by demonstrating this connection in another diverse sample of children living in poverty. The study sample consisted of 168 children that were in homes with an income below the federal poverty line and 119 children from middle class homes. The groups differed in single parent status and parental educational attainment. Children (51% male) had an average age of 9.2 years old and 97% were White. During home visits, caregivers completed questionnaires, while children completed cardiovascular tests, a urine sample, and questionnaires. While in the homes, research assistants conducted an observation of the physical environment. The authors developed a cumulative risk model comprised of three physical factors, which included crowding, noise level, and housing quality, and three psychosocial factors, which included family turmoil, child-family separation, and violence.

Compared with the middle class sample, children living in poverty experienced more stressors including crowding, more noise, and poorer quality housing. In addition, these children

also experienced more psychosocial stressors (family turmoil, child-family separation, and violence). Evans and English (2002) also found that the children in the poverty sample had greater difficulties with self-regulatory behaviors (i.e., delayed gratification) and elevated resting blood pressure, cortisol, and epinephrine levels. Additionally, the authors found that the cumulative risk model partially mediated the association between poverty and socioemotional adjustment. Due to the cross-sectional nature of the data, no causal statements could be made. However, this study does provide evidence that children living in poverty have greater exposure to stressors and more negative outcomes (e.g., psychological distress, worse self-regulatory behaviors and higher psychobiological stress) associated with the circumstances and experiences of living in poverty.

Poverty has been linked to a decreased life span and worse health outcomes in adulthood (Evans & Kim, 2007). These findings are seen regardless of the person's social status as an adult. Evans and Kim (2007) offered a possible explanation for the mechanisms underlying the association between poverty and health in their longitudinal study of 207 children (52% male) with a mean age of 13 years old. In their study, approximately 53% of the sample lived below the poverty line at recruitment and consisted of primarily White children who lived in rural areas of upstate New York. The authors calculated an income-to-needs ratio to determine if the family lived in poverty. They also conducted a number of physiological measures including, cortisol levels, creatinine levels, blood pressure, and cardiovascular reactivity. The authors included 6 risk domains in their cumulative risk models: crowding, noise, and substandard housing as physical risks, and family turmoil, child's separation from parents, and exposure to violence as social risks. Each risk was dichotomized at each wave of the study and cumulative risk scores ranged from 0 to 6.

Evans and Kim (2007) found that more time living in poverty was associated with higher levels of chronic HPA activity, which they stated was consistent with prior research (i.e., Evans & English, 2002; Lupien et al., 2000). Given the longitudinal study design, the authors reported a link between childhood poverty and a dysregulated stress response. During an acute-stressor protocol (i.e., impromptu math test), 13 year olds who had lived in poverty longer showed more muted cardiovascular reactivity (i.e., blood pressure reactivity). In addition, children who lived in poverty longer had greater cumulative risk exposure (e.g., physical and social stressors). The authors stated that their findings provide evidence for cumulative risk exposure having effects on childhood poverty and stress regulation. In essence, greater cumulative risk exposure during early childhood compromised children's ability to handle environmental demands.

Further, Evans, Kim, Ting, Tesher, and Shannis (2007) examined the effects of cumulative risk in conjunction with maternal responsiveness in a young adolescent sample. Participants were the same cohort of children from the Evans and Kim (2007) study. The authors called attention to the fact that most work involving cumulative risk has had samples of preadolescent children and called for a need of this type of research with older children. Evans and colleagues (2007) included a potential protective factor (maternal responsiveness) and highlighted that much of the research involving cumulative risk has not focused on protective factors. There were nine domains of risk factors that were assessed, which included the following: residential density (crowding), noise levels, housing quality, exposure to family turmoil, child-family separation, violence, poverty, having a single parent, and maternal high school dropout. Risk scores on each factor were dichotomized and cumulative risk indices ranged from 0 to 9. Maternal responsiveness, allostatic load, and cardiovascular reactivity and recovery were also assessed.

Evans and colleagues (2007) found that higher cumulative risk levels were associated with elevated allostatic load as seen in previous studies with younger children. The authors also found this association held true longitudinally when they controlled for allostatic load from 3 to 4 year prior. However, the association between cumulative risk and allostatic load only held when maternal responsiveness was low. The authors stated that a possible explanation for why early risk exposure is related to poor health outcomes later in life might be through elevated allostatic load in childhood. Additionally, the authors found that young adolescents who had higher levels of cumulative risk had more muted cardiovascular reactivity and slower cardiovascular recovery patterns. The authors reported that the less efficient stress response comprised of more muted cardiovascular reactivity and slower recovery was important because: 1) the inefficient response and recovery may have been indicative of developing pathology and morbidity, and 2) these findings were in line with allostatic load theory. Findings suggest that allostatic load may be central to understanding early life risk exposure and poor health outcomes later in life.

Evans, Li, and Whipple (2013) conducted a review, which assessed the state of the literature on cumulative risk. The authors highlighted the importance of assessing for multiple risk factors in that exposure to multiple risk factors has been associated with worse developmental consequences. Further, children tend to experience multiple stressors simultaneously rather than each in isolation. By studying multiple risk factors, children that are the most at need for interventions can be identified and interventions can be designed to be more effective by targeting multiple risk factors. The authors defined multiple risk as, "an overarching term that encompasses any model with more than one risk factor as a variable. More specifically,

[cumulative risk] models operationalize multiple risk factor exposure in an additive manner, that is, no statistical interactions are examined" (p. 1345).

Evans and colleagues (2013) discussed the weaknesses and strengths of cumulative risk approaches. One of the main weaknesses of such models includes the lack of a theoretical background or explanation for why cumulative risk models may have more predictive power on child outcomes than a single risk factor. Further, there may be several different explanations for the greater predictive power. For instance, the biological effects of stress exposure may help explain why people are impacted more by cumulative risk (Evans et al., 2013). In addition, studying the underlying or mediational processes that occur may give a substantive foundation for the stronger effects of cumulative risk models. Another weakness includes the lack of theoretical reasoning for why certain factors are selected for cumulative risk models. Additionally, Evans and colleagues (2013) reported that little thought has been given to the different domains of risk that children may face. In many studies the designation of risk appears to be arbitrary. Also, cumulative risk models cannot take into account the interactions of risk factors. The authors reported that another weakness of cumulative risk models is that information is lost due to the risk factors being dichotomized. Typically risk factors are dichotomized (yes/no) and therefore with continuous variables the degree of risk is lost.

The current study attempted to address some of the shortcomings discussed by Evans and colleagues (2013), such as the lack of a theoretical foundation and information lost due to dichotomizing continuous variables. In the current study, multiple theoretical models were used to justify the inclusion of risk factors; these models included Bronfenbrenner's Social-Ecological Theory and the toxic stress framework proposed by the American Academy of Pediatrics and Johnson and colleagues (2013). These models/frameworks along with work by Wright (2011)

and Wood and colleagues, (2015) among others exemplifies the need to assess multiple stressors from multiple levels (e.g., individual, family, neighborhood) of child's daily life to examine the impact stress has on health outcomes and more specifically, asthma outcomes. In addition, in the current study, continuous variables were not dichotomized, and were standardized to allow for more variation.

In their review paper, Evans and colleagues (2013) also provided examples of the strengths of cumulative risk models. The authors stated that the greatest strength of cumulative risk models may be their ability to predict developmental outcomes. Additionally, the factors are unweighted in the model meaning that there is no assumption that one factor may have more of an impact than another. The authors also reported that cumulative risk models are parsimonious and do not need much statistical power; they provide an alternative to models and analyses that need large sample sizes. Lastly, Evans and colleagues (2013) reported that cumulative risk models are easily understood and can be easily communicated to other individuals, including policy makers.

Finally, Evan and colleagues (2013) discussed avenues of future directions for cumulative risk research. Two such avenues included: 1) examining mediating variables between cumulative risk and developmental outcomes, and 2) studying cumulative risk models across multiple domains. The current study sought to add to the literature on cumulative risk in children with asthma through these two ways. First, we examined the potential for caregiver asthma-related support to buffer the effects of cumulative risk on asthma outcomes. Second, the cumulative risk model in the current study included risk factors from multiple domains (e.g., individual level, family level, neighborhood level stressors).

In addition to the more general research on cumulative risk, several studies have examined cumulative risk within pediatric asthma populations. Specifically, Koinis-Mitchell and colleagues (2007) examined whether a cumulative risk index predicted asthma outcomes in urban children more so than one predictor alone. This study included 163 children and their primary caregivers (majority biological mothers). Children (58% male) ranged in age from 7 to 15 years old, with a mean age of 10.5 years old, and were Latino, Black, or non-Latino White. Forty-five percent of caregivers were married and 31% were never married. Fifty-five percent of caregivers were unemployed and had an average of 13 years of education (range of 5 to 17 years). The mean annual household income was $32,382 with a range from $500 to $100,000. Children had to be diagnosed with asthma and be receiving current treatment to be included in the study. Both caregivers and children completed a series of questionnaires.

Koinis-Mitchell and colleagues (2007) calculated a cumulative risk index score for each family comprised of: poverty, neighborhood disadvantage, perceived discrimination, acculturative stress, child asthma severity, and environmental tobacco smoke exposure. The authors found that the cumulative risk index accounted for asthma morbidity more than poverty or asthma severity alone. Higher levels of cumulative risk were associated with more functional impairment due to asthma. Additionally, children who had higher levels of cumulative risk also had more ED visits and hospitalizations in the last year. The authors found that the mean levels of asthma morbidity increased for up to 3 risk factors and then either leveled off or started to decline. The authors speculated this may be due to the fact that families with the highest number of risk factors have already been recognized by providers and provided with other means of support (e.g., educational programs, case workers). Another explanation is that families experiencing numerous risk factors may be more desensitized to changes in the child's asthma

symptoms and seek care at the ED less often. As far as group differences, the authors found higher rates of poverty and a greater number of cumulative risk factors experienced among Black families. The authors called for a need to examine cumulative risk models in future research, including with larger and diverse samples.

Other studies that examined cumulative risk in pediatric asthma samples includes another study by Koinis-Mitchell and colleagues (2010). Specifically, Koinis-Mitchell and colleagues (2010) examined racial/ethnic group differences on the cumulative risk dimensions. Across all groups, higher risk levels were associated with more functional limitation. Koinis-Mitchell and colleagues (2010) found that more Black and Latino families were categorized as high risk on several dimensions of the cumulative risk index as compared to non-Latino White families. Additionally, Koinis-Mitchell and colleagues (2012) found cumulative risk to be associated with more functional limitations in children with asthma. In this study, Koinis-Mitchell and colleagues (2012) also examined the role of protective factors in the association between cumulative risk and functional limitation in children with asthma. Positive attitudes toward school was a protective factor for Black families, whereas increased levels of asthma self-efficacy and family connectedness were protective factors for Latino families. In addition, the effect of cumulative risk on caregiver QOL for caregivers of a child with asthma has been examined (Everhart, Fiese, and Smyth, 2008). Everhart and colleagues (2008) found that as stressors accumulated, caregiver QOL worsened; cumulative risk predicted caregiver QOL as a quadratic function. Together, these studies demonstrate the utility of cumulative risk as a predictor of pediatric asthma outcomes.

The current study expanded upon previous cumulative risk research by including a wider age range of children and adolescent participants (7 to 17 years of age). In addition to the

younger cohort (7 to 12 year olds), this study included an adolescent cohort (13 to 17 year olds), which has not been as well researched in the cumulative risk literature. In addition, the current study aimed to understand the impact of developmentally appropriate stressors in a cumulative risk model for adolescents with asthma.

Ecological Momentary Assessment

The current study used ecological momentary assessment (EMA) to examine child asthma outcomes over a two week period. EMA surveys were completed by caregivers of the younger cohort (7 to 12 year olds). EMA is defined as a methodology that involves repeated assessments of an individual in their natural environment and may allow for results that more accurately reflect real life experiences (Smyth & Heron, 2014). There are three primary benefits of using EMA methodology over traditional self-report measures. First, EMA methodology is often advantageous over retrospective self-report measures because it limits the effect of social desirability and retrospective recall issues. Individuals are often completing assessments immediately or shortly after an event has happened, thus limiting the time between an event and assessment. Second, EMA data have greater ecological validity. Ecological validity is increased by collecting data from participants in their natural environment versus in a laboratory setting (Heron & Smyth, 2010). EMA also incorporates data collection at multiple time points over multiple days, allowing for a more accurate portrayal of the participant's experiences over time. Thirdly, EMA has the enhanced ability to capture within-person associations between variables and in doing so, identify temporal dynamics (Smyth & Heron, 2011). Patterns across days or weeks and changes within individuals can be assessed through EMA data collection.

EMA has had limited use with low-income, urban families. Specifically, few studies have assessed pediatric asthma through the use of EMA, and the majority of these have focused on

adolescent populations. For example, Dunton and colleagues (2015) examined the variability in stressors and the impact this variability had on asthma symptoms in ethnic minority adolescents. Participants were 20 Hispanic adolescents ages 12 to 17 years old that had chronic asthma. Adolescents completed surveys across a seven day time span. Participants were prompted to complete surveys randomly during seven different time intervals a day (signal-contingent) and also were prompted after they used their inhaler (event-contingent). Dunton and colleagues (2015) found that when adolescents experienced more stress, they were more likely to report asthma symptoms in the hours to follow. In this example, the variability in the amount of stress experienced (due to arguments, teasing, or parental disagreements) by adolescents was of interest. Through EMA, the authors were able to demonstrate that daily variability in stress was associated with asthma symptoms in adolescents.

Mulvaney and colleagues (2013) also used EMA via mobile technology with a group of adolescents with asthma (12 to 18 years old). Fifty-three caregiver-adolescent dyads completed the study, which assessed adolescent asthma symptoms and adherence. Adolescents completed one phone survey each day for 30 days via an interactive voice response system. Sixty-four percent of adolescents were Black (58% female). The authors found that baseline asthma control (measured by the Asthma Control Test) was correlated with increased symptoms and rescue inhaler use reported in the momentary assessments. Symptoms occurred across a variety of settings including school, home, and sporting events. Mulvaney and colleagues (2013) found that in the presence of peers, adolescents were less likely to use their rescue inhaler, suggesting the presence of peers as a potential barrier of adherence.

The current study used EMA surveys delivered via smartphone and were intended to capture the daily experiences of urban families of children with asthma (Aims 1; secondary data

analyses). Asthma outcomes including daily symptoms and ED visits were assessed over a two-week time period. This study also capitalized on the ecological validity and reduced memory bias aspects of EMA methodology. First, caregivers completed assessments in their home (i.e., natural setting) and in doing so assessments may more accurately reflect their real life experiences. Second, caregivers had a shorter recall length than most standard self-report measures. The short time frame (~24 hours) can help reduce retrospective recall biases. In addition, children provided daily reports of lung function via handheld spirometers; in the original data collection portion of this study, adolescents completed lung function assessments via handheld spirometers. This type of EMA or ambulatory assessment data is active, but non-self-report data (Smyth & Heron, 2014). Therefore, this study included objective EMA data to assess lung function from both children and adolescents.

Asthma Outcomes

The current study used a cumulative risk model to determine its impact on several well-established outcome measures of pediatric asthma including, quick-relief medication use, ED visits, asthma control, lung function, and quality of life (QOL). For instance, increased quick-relief medication use has been associated with neighborhood related stress (Kopel et al., 2015). Another adverse outcome for children that have asthma is number of ED visits (CDC, 2006). ED visit rates are higher among Black children than White children and this disparity continues to rise (CDC, 2006). ED visits have also been associated with family functioning (Kaugars et al., 2004) and neighborhood factors, including living in poverty (Chen & Schreier, 2008) and violence exposure (Bellin et al., 2014). In addition, cumulative risk models have been associated with more ED visits in children with asthma (Koinis-Mitchell et al., 2007).

Asthma control is defined by the NHLBI as, "the degree to which the manifestations of asthma are minimized by therapeutic interventions" (NHLBI, 2013). Poor asthma control has been associated with neighborhood stress (Koinis-Mitchell et al., 2014; Kopel et al., 2015). Additionally, lung function is another asthma outcome that is prevalent in research (Fiese, Winter, Wamboldt, Anbar, & Wamboldt, 2010; Kopel et al., 2015). Lung function can be obtained via spirometry tests, which assess how much air an individual can push out of their lungs.

In addition to assessing lung function and quick-relief medication use in children and adolescents with asthma, QOL is often used as an outcome measure in pediatric asthma to describe how a child's asthma is impacting his or her daily life (Juniper, 1997). QOL measures provide clinicians with a more complete picture of how asthma is impacting children across several domains of functioning. Research has shown that asthma QOL questionnaires often measure a different component of health status than clinical measures (e.g., spirometry readings; Juniper et al., 2004; van Gent et al., 2008). Children who experience lower levels of QOL may be distressed by limited activities, frustrated by symptoms during the school day, and feel different from their peers without asthma (Juniper, 1997). Thus, it is important to understand the factors that impact both lung function and QOL in children with asthma.

The current study also assessed child age as a potential moderator in associations between cumulative risk and child asthma outcomes (e.g., QOL, asthma control, quick-relief medication use, ED visits). Previous research suggests that asthma outcomes may differ by child age and that factors associated with QOL may differ by age (Kuehni & Frey, 2002; Miadich et al., 2015; Moreira et al., 2013). However, not all research has been consistent in terms of which age group may experience worse outcomes.

Moreira and colleagues (2013) assessed QOL in children with a number of chronic conditions and specifically included a cohort of children with asthma. Children were 8 to 18 years old and approximately 63% of the sample were male. Moreia and colleagues (2013) found that younger children with asthma reported better QOL than adolescents with asthma. Additionally, Miadich and colleagues (2015) assessed whether child age moderated the association between routine burden (related to asthma care) and asthma severity with child QOL. Participants included 192 children (63% male) ages 5 to 12 years of age and their caregivers. Child age significantly moderated the association between routine burden and child QOL as well as the association between asthma severity and QOL (Miadich, Everhart, Borschuk, Winter, & Fiese, 2015). Both findings were significant for older children, such that more routine burden and worse asthma severity were associated with poorer QOL. These findings suggest that asthma severity and routine burden associated with asthma care may be more proximal to QOL in older children with asthma. Together these studies suggest that QOL may differ by child/adolescent age or that factors related to QOL may impact children of different ages in different ways.

As previously discussed, adolescents are often given control of their asthma care prematurely by parents suggesting suboptimal asthma management among adolescents (Bruzzese et al., 2012). Adolescent asthma management also tends to be more reactive than proactive or preventative (Bruzzese et al., 2012), which can lead to more symptom exacerbations (i.e., poorer asthma control). This may suggest that asthma control would be poorer among adolescents with asthma. However, other research suggests that younger children may have poorer asthma control and increased frequency of symptoms (Kuehni & Frey, 2002). Kuehni and Frey (2002) examined asthma control among 572 children (64% male) ages 4 to 16 years old. Poorer asthma control including nighttime symptoms, morning symptoms, activity limitation, and frequency of severe

attacks that limit speech were all more common among younger children. Kuehni and Frey (2002) suggested that poorer asthma control among younger children may be due to greater asthma severity possibly related to increased frequency of viral infections or hesitation by doctors and parents to increase medication use. Although mixed in findings, previous research suggests that there may be differences in asthma outcomes (e.g., QOL, asthma control) based on child/adolescent age. Therefore, more research is needed to determine if there are age differences in child asthma outcomes and what factors may drive these differences.

Current Study

Failure to properly manage childhood asthma can result in increased emergency department (ED) visits, missed school days, and an estimated $50.1 billion spent on healthcare expenditures. Racial and ethnic minority children, especially those living in low-income, urban areas, are at particularly high risk for asthma morbidity and associated high healthcare use. Families living in urban settings are likely to experience additional stressors (e.g., neighborhood violence) that can contribute to increased asthma morbidity in children. Many stressors do not happen as isolated events. Rather it is the combined effect of multiple stressors that can have a lasting impact on a child's health trajectory.

Toxic stress, described as a continual activation of the stress response without adequate buffers against stress (e.g., caregiver support), experienced as a child has been linked to the development of asthma. In recent years, the toxic stress framework has been emphasized in research and policy statements. Specifically, the American Academy of Pediatrics' policy statement defined toxic stress and how it can affect a person's health across the entire lifespan (American Academy of Pediatrics, 2012). The Adverse Childhood Experiences (ACES) Study has also highlighted the impact of childhood exposures to dysfunction and abuse on adult health

problems (Felitti et al., 1998). Researchers and providers are acknowledging the connection between early life experiences and health problems later in life. However, research has yet to explicate processes related to toxic stress that increase asthma morbidity in urban children and adolescents with asthma.

This study used previously collected EMA surveys to enhance understanding of processes that place children (7 to 12 years old) with asthma from low-income, urban settings at high risk for negative outcomes. Consistent with a toxic stress framework, this study considered the impact of a cumulative risk model of stressors including poverty, neighborhood stress, perceived discrimination, caregiver stress, and family functioning on outcomes in urban children (7-12 years) with persistent asthma. Cumulative risk conceptualizes the impact of multiple, overlapping risk factors on adverse outcomes rather than the individual impact of each factor alone. This study further aimed to examine a cumulative risk model as a predictor of asthma outcomes in a sample of 60 adolescents (13-17 years) from the same urban area.

Specific Aims

Aim 1: The first aim of the current study was to test a cumulative risk model (including poverty, neighborhood stress, family functioning, caregiver stress, perceived discrimination) based on a toxic stress framework as a predictor of asthma outcomes (e.g., lung function, quick-relief medication use, ED visits, asthma control, asthma-related quality of life [QOL]) in a sample of 60 urban children with persistent asthma (7-12 years). Figure 1 outlines the cumulative risk model. Specifically I hypothesized that:

1. Greater cumulative risk would be associated with worse child asthma outcomes (more ED visits and quick-relief medication use [measured via daily EMA surveys], worse lung function [measured via daily handheld spirometers], worse asthma-related QOL and

asthma control [measured via self-report questionnaires]).

2. Higher levels of caregiver support related to asthma care would minimize the impact of cumulative risk on negative child asthma outcomes.
3. Cumulative risk would be associated with momentary-level child asthma outcomes (daily EMA responses) over a two week period. Specifically, cumulative risk would be associated with momentary-level child ED visits and quick-relief medication use and daily lung function assessments.

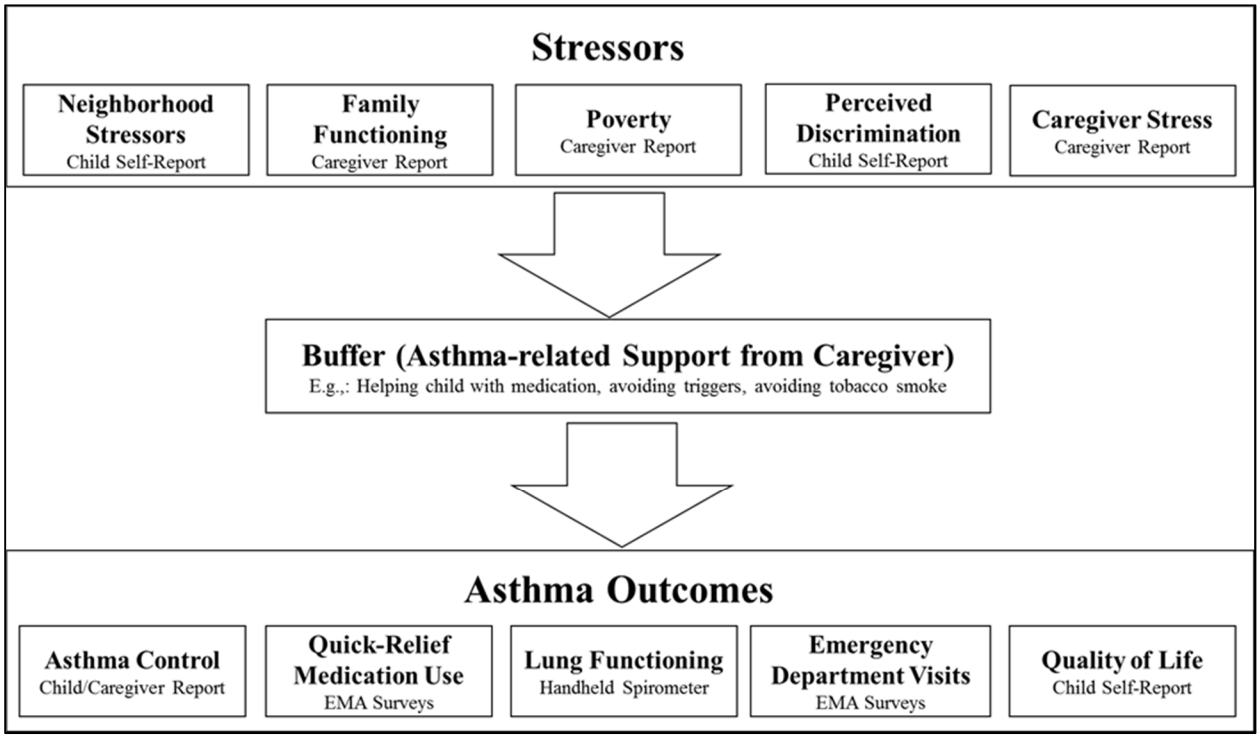

Figure 1. *Toxic stress theoretical framework of the current study. (American Academy of Pediatrics; Johnson et al., 2013).*

Aim 2: The second aim of the current study was to determine whether a new cumulative risk model of adolescent-specific stressors was a stronger predictor of asthma outcomes in a sample of 60 urban 13-17 year olds than the original risk model described in Aim 1. Adolescent-specific stressors included school stress and relationship stress (parent/caregiver conflict and

peer pressure). Specifically, I hypothesized that:

1. The new cumulative risk model for urban adolescents with asthma would include school and relationship (parent/caregiver conflict and peer pressure) stressors, as well as poverty and neighborhood stress. I expected this new model to account for more variance in adolescent asthma outcomes than the original model for 7-12 year olds.
2. Adolescents that experienced more stressors and less asthma-specific support from caregivers would have worse asthma outcomes (e.g., more ED visits and quick-relief medication use, worse lung function, worse asthma-related QOL and asthma control).

Aim 3: The third aim of the study involved combining both data sets to test child/adolescent age as a moderator of the association between cumulative risk and asthma outcomes. This data set included 121 urban families with a child with asthma (7 to 17 years old). This aim assessed whether age moderates the association between cumulative risk and asthma outcomes. I hypothesized that:

1. Age would significantly moderate the association between cumulative risk and asthma outcomes, such that younger children with higher cumulative risk outcomes would have worse asthma outcomes.

Methods

Overview

Aims 1: Analyses conducted for the first aim of the current study were done through secondary data analysis of the CARE Study (**C**hildhood **A**sthma in **R**ichmond Famili**e**s; Targeted Research Grant, Society of Pediatric Psychology, R. Everhart, PI). Sixty-one caregiver and child dyads (7-12 years old) completed an initial research session. Caregivers completed EMA surveys on a smartphone twice daily for 2 weeks. Children used an AM2 device (handheld spirometer) to

measure lung function twice daily for 2 weeks. **Aim 2:** Original data were collected from 60 urban families with an adolescent with asthma (13-17 years old) in Project AAIR (**A**dolescents with **A**sthma **I**n **R**ichmond; F31HL129681, S. Miadich, PI). Caregivers and adolescents completed an initial research session and adolescents used a handheld spirometer twice daily for 2 weeks. **Aim 3**: The two cohorts (CARE Study participants and Project AAIR participants) were combined for analyses.

CARE Study: Secondary Data Analysis

CARE Study Participants. Sixty-one child and caregiver dyads completed the CARE study (see Table 1 for demographic information).

Table 1.

CARE Study Caregiver and Child Demographics

Demographic Items	**Total Sample** N = 61
Caregiver	
Age, *M years (SD)*	37.90 (9.18)
Race/Ethnicity, *n* (%)	
African American/Black	55 (90.2)
Caucasian/White	4 (6.6)
Mixed/Multi-Racial	1 (1.6)
Other	1 (1.6)
Relation to child, *n* (%)	
Biological Mother	49 (80.3)
Step or Adoptive Mother	3 (4.9)
Biological Father	7 (11.5)
Grandmother	1 (1.6)
Other	1 (1.6)
Relationship Status, *n* (%)	
Single/Never Married	40 (65.6)
Married	9 (14.8)
Separated	6 (9.8)
Divorced	4 (6.6)
Widowed	2 (3.3)
Education, *n* (%)	
Less than a high school education	11 (18.0)
High school degree	20 (32.8)

Some college	21 (34.4)
College degree or higher	9 (14.8)
Income (Past Month), *n* (%)	
Less than $1,000	23 (37.7)
$1,000 - $1,999	15 (24.6)
$2,000 - $3,999	16 (26.2)
Greater than $4,000	5 (8.2)
Household size, *M (SD)*	4.31 (1.64)
Child	
Age, *M years (SD)*	9.59 (1.52)
Sex (Male), *n* (%)	42 (68.9)
Race/Ethnicity, *n* (%)	
African American/Black	55 (90.2)
Caucasian/White	4 (6.6)
Mixed/Multi-Racial	1 (1.6)
Other	1 (1.6)

CARE Study Procedures. Institutional Review Board approval was obtained prior to the start of this study. Two main recruitment methods were utilized. First, VCU's Center for Clinical and Translational Research's Biomedical Informatics Core (BIC) identified potential participants by searching electronic health records of pediatric patients in the VCU system. Participants were identified by age (7-12 years) and asthma diagnosis. Second, flyers were distributed at pulmonology clinics, pediatric offices, community centers, and neighborhood centers in Richmond, Virginia. Families identified through BIC were contacted via phone by trained research assistants (RAs) to assess eligibility and interest through a brief screening questionnaire.

Inclusion criteria for children included that they were 7 to 12 years of age with persistent asthma. Children met persistent asthma criteria in line with the NHLBI guidelines (2007) by having a current controller medication prescription or by endorsing at least one of the following: 1) daytime symptoms 2 or more times a week in the last four weeks, 2) nighttime symptoms 3 or 4 times a week in the last four weeks, 3) quick-relief medication use 2 or more times a week in the last four weeks, 4) activity limitation due to asthma symptoms in the last four weeks, or 5)

oral steroid prescription 2 more times in the past year. Primary caregivers needed to speak English and identify as Black, White, or Latino to be eligible. The inclusion of families from Latino, Black, and White backgrounds is consistent with the growing body of literature focused on pediatric asthma disparities (Canino et al., 2006; Everhart, Fedele, Miadich, & Koinis-Mitchell, 2014). Exclusion criteria included the child having an additional pulmonary disease, developmental delay, or severe psychiatric or other medical illness. For consenting purposes, children could not live in foster care or a group home. If the family was eligible and interested upon completion of screening, they were scheduled for an initial research session. Depending on family preference, this research session took place in either research space in the Center for Psychological Services and Development (CPSD) on VCU's campus or in the family's home. Families that wished to come to the CPSD but had no reliable transportation were provided taxis.

Consent and assent procedures were completed before the administration of questionnaires. Trained RAs administered questionnaires to caregivers and children using established procedures. For instance, an RA administered questionnaires to children in a developmentally appropriate manner, ensuring that the child understood the question and response options. Questionnaires were administered simultaneously and caregivers and children were separated to the extent that the setting would allow (e.g., opposite sides of the room in the research offices, opposite ends of the couch at homes). After questionnaires were completed, caregivers were shown how to complete EMA surveys on an Android smartphone provided to them for the two week EMA monitoring period. RAs completed practice surveys with caregivers in the session. Caregivers selected a beep schedule, which was programmed into the phone to prompt them to complete surveys twice a day. Both the child and caregiver were shown how to use the hand-held spirometer (AM2 device) that children were asked to use twice a day during

the two week monitoring period. Children were asked to blow into the device three times in the morning and evening (i.e., before they went to bed). At the end of the initial session, caregivers received a $25 gift card and children were given a pre-approved, age appropriate prize. RAs called to check-in with families a few days after the baseline session to ensure that they were not having any difficulty using the devices and to answer any questions the family had. At the end of the 14 day monitoring period, RAs retrieved devices from the families' homes. Caregivers received a $50 gift card and the child received another prize at this time.

EMA procedures. All caregivers were provided Samsung Galaxy Stellar phones for their monitoring period. Calling and texting were disabled as these phones were for research purposes only. The survey application used was developed by the DREAM (Dynamic Real-Time Ecological Ambulatory Methodologies) Initiative at the Survey Research Center at Penn State University. In the training session, caregivers were first asked about their prior knowledge of smartphones. If the caregiver indicated that he/she was unfamiliar with the technology and had not used a smartphone, training started with basic information about the phone (e.g., what is a home button). After covering smartphone basics, RAs completed a practice survey with caregivers. RAs and caregivers each had a phone and completed the survey alongside each other so that the RA could demonstrate as they went along. RAs began with the evening survey given that it was longer and covered more types of questions. The different types of questions and answer responses were explained to caregivers as they appeared in the survey: single response questions, multiple response questions (may include multiple screens), and slider-scale questions (Likert-scale items that had a slider bar underneath). At the end of each survey, caregivers were prompted to enter the PEF values from the handheld spirometer after each breath the child took (3 breaths in total at each time point).

After practicing a survey together, caregivers were asked if they had questions or wanted additional practice. A beep schedule was then selected from four pre-programmed options. The beeps served as a reminder for caregivers to complete the surveys. Caregivers were asked to leave the volume on and not turn off the phone during the monitoring period. Data from the smartphones were uploaded to the server managed by the DREAM Initiative in real-time if the caregiver was within signal. All data were sorted by participant identification numbers that were pre-programmed into the phone along with the selected beep schedule. Additionally, to ensure all data were maintained, data were also saved to the phone. Once the smartphone was retrieved, data were downloaded from the phone and then uploaded and merged onto the server. Smartphones were then cleared and reset to prepare them for use with the next family.

AM2 device procedures. Children and caregivers were both shown how to use the handheld spirometer (AM2 device); this ensured that caregivers could help their child if a problem arose. Children were instructed to sit upright with their feet flat on the floor while using the device. To use the device properly, children needed to take in a deep breath and breathe out as hard and as fast as they could until they heard the device beep. At this time, the device would display a PEF value. They were instructed to do this two more times (three in total). Caregivers were asked to record the three PEF values at the end of their surveys. Caregivers and children were encouraged to complete the surveys and use the spirometers at the same time to ensure that caregivers could record the PEF values in the smartphone. Data were saved to the devices and were downloaded to a laptop computer once the device was retrieved. Afterwards, devices were cleaned and reset for another participant.

Project AAIR: Original Data Collection

Project AAIR Participants. Sixty-one adolescent and caregiver dyads completed the

Project AAIR; however data from one dyad were not included due to the family having already completed the CARE Study (see Table 2 for demographic information on the 60 included dyads).

Table 2.

Project AAIR Caregiver and Adolescent Demographics

Demographic Items	**Total Sample** N = 60
Caregiver	
Age, *M years (SD)*	41.83 (8.42)
Race/Ethnicity, *n* (%)	
African American/Black	55 (91.7)
Latino	2 (3.4)
Caucasian/White	1 (1.7)
Mixed/Multi-Racial	1 (1.7)
Other	1 (1.7)
Relation to child, *n* (%)	
Biological Mother	54 (90.0)
Grandmother	5 (8.3)
Biological Father	1 (1.7)
Relationship Status, *n* (%)	
Single/Never Married	35 (58.3)
Married	13 (21.7)
Divorced	7 (11.7)
Separated	3 (5.0)
Widowed	2 (3.3)
Education, *n* (%)	
Less than a high school education	17 (28.3)
High school degree	18 (30.0)
Some college	21 (35.0)
College degree or higher	4 (6.7)
Income (Past Month), *n* (%)	
Less than $1,000	24 (40.0)
$1,000 - $1,999	16 (26.7)
$2,000 - $3,999	17 (28.3)
Greater than $4,000	3 (5.0)
Household size, *M (SD)*	4.47 (1.96)
Adolescent	
Age, *M years (SD)*	14.73 (1.38)
Sex (Female), *n* (%)	32 (53.3)
Race/Ethnicity, *n* (%)	
African American/Black	56 (93.3)

Latino	2 (3.4)
Caucasian/White	1 (1.7)
Mixed/Multi-Racial	1 (1.7)

Project AAIR Procedures. Institutional Review Board approval was obtained for this study. Adolescent and caregiver dyads were recruited using the same methods as the previous cohort: obtaining potential participants' contact information from BIC and flyer distribution in the same community. Also in line with the CARE Study, potential participants were screened over the phone to determine eligibility. Interested and eligible families were scheduled for a research session at the CPSD or in their home. Adolescents needed to be 13 to 17 years of age and meet persistent asthma criteria (outlined in CARE Study procedures). In addition, primary caregivers needed to speak English and identify as Black, White, or Latino. Exclusion criteria included the adolescent having an additional pulmonary disease, developmental delay, or severe psychiatric or other medical illness. For consenting purposes, adolescents could not live in foster care or a group home. Additionally, adolescents could not be a sibling of a child that was enrolled in the CARE Study to prevent duplicate family data. Procedures mirrored those of the CARE Study; however, EMA surveys were not administered as a part of Project AAIR.

Consent and assent forms were completed before questionnaires were administered. Trained RAs administered questionnaires to caregivers and adolescents, ensuring understanding of the items. Questionnaires were administered simultaneously with the caregiver and adolescent separated as much as the physical environment would allow (e.g., opposite sides of room in research offices, opposite sides of room or couch in family homes). Once questionnaires were completed, adolescents were trained to use the handheld spirometer (AM2 device) twice a day for 14 days (3 breaths at each time point). Adolescents and caregivers were each compensated $10 after the research session and adolescents were compensated $25 when spirometers were

retrieved.

AM2 device procedures. The handheld spirometers were preprogrammed before given to the adolescents. During the screening process, RAs obtained the height and weight of the adolescent and that information along with sex and age were used to set up the device for the adolescent. During the research session, the same instructions were given to adolescents as were given to the younger cohort. Adolescents were instructed to take three breaths every morning and evening across the two week monitoring period to assess their lung function. Data were downloaded from devices after they are retrieved and devices were cleaned and reset for future participants.

Measures

Table 3 illustrates the list of measures used in both studies, including the respondent/reporter and associated aims.

Table 3.

Measures Used in Both Studies

Measure	Respondent	Associated Aims
1. Demographic and SES Information	Caregiver	Aims 1, 2 & 3
2. Family Assessment Device	Caregiver	Aims 1, 2 & 3
3. Perceived Stress Scale	Caregiver and Adolescent	Aims 1, 2 & 3
4. Asthma Assessment Form	Caregiver	Aims 1, 2 & 3
5. Asthma Control Test	Caregiver and Child/Adolescent	Aims 1, 2 & 3
6. The Stress Index: Neighborhood Stress Scale	Child/Adolescent	Aims 1, 2 & 3
7. Child Racial/Ethnic Discrimination	Child/Adolescent	Aims 1, 2 & 3
8. Pediatric Asthma Quality of Life Questionnaire	Child/Adolescent	Aims 1, 2 & 3
9. Asthma-Related Caregiver Support Items	Caregiver/Adolescent	Aim 2
10. Adolescent Stress Questionnaire	Adolescent	Aim 2
11. Conflict Behavior Questionnaire	Adolescent and Caregiver	Aim 2

12. EMA Data	Caregiver	Aim 1
13. Spirometry Data	Child/Adolescent	Aims 1, 2 & 3

Measures Completed in Both Studies

Demographic Information. Caregivers self-identified their race/ethnicity, and reported on their child/adolescent's race/ethnicity, age, and grade. Caregivers disclosed their relationship to the child/adolescent and reported household size. Socioeconomic status (SES) information included caregiver marital status, male and/or female head of the household's education and occupation, and total household income (past year and month). Poverty was included as one of the potential stressors and was calculated for each family. An income-to-needs ratio was calculated based on household income and the poverty threshold for a family of that size. Poverty thresholds were retrieved for all years participants were seen and were matched based on visit date. If the income-to-needs ratio was greater than 1.0 the family was considered below the poverty line (U.S. Dept. of Health and Human Services, 2005).

Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983). The FAD is a 60 item questionnaire that assesses family functioning. Caregivers were asked to rate how much each statement describes their family on a 1 (strongly agree) to 4 (strongly disagree) scale. Six subscales are derived from the measure covering six dimensions of family functioning: problem solving, communication, roles, affective responsiveness, affective involvement, behavior control, along with an overall general functioning scale. Higher scores indicate more impaired functioning. For the CARE Study, the total FAD questionnaire had good internal consistency ($\alpha = 0.82$) and the general functioning subscale had adequate internal consistency ($\alpha = 0.74$). For Project AAIR, the total FAD questionnaire had good internal consistency ($\alpha = 0.83$) and the general functioning subscale also had good internal consistency ($\alpha = 0.80$). The general

functioning subscale was used in analyses.

Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). Perception of stress was assessed with the 14 item PSS. Participants were asked to reflect on the last month and rank their answers on a 0 (Never) to 4 (Very Often) scale. Positively worded items were reversed scored and then items were totaled. Higher scores on the PSS indicate more perceived stress. Items on the PSS completed by caregivers for the CARE Study had good internal consistency ($\alpha = 0.87$). For Project AAIR, the PSS completed by caregivers had adequate internal consistency ($\alpha = 0.79$).

Asthma Assessment Form (Rosier et al., 1994). The Asthma Assessment Form evaluates child asthma severity over both the past 4 weeks and 12 months. Frequency of both asthma symptoms and limitations due to asthma are assessed. Specifically, frequency of asthma attacks, symptoms (e.g., coughing, wheezing, shortness of breath) at morning, during the night, and during activities, and medication use are assessed. In addition, the Asthma Assessment Form covers how many missed days of school, doctor visits, ED visits, and hospital stays the child has had in the last 4 weeks and 12 months. ED visit information from this form was used in both CARE and AAIR analyses.

Asthma Control Test (ACT; Nathan et al., 2004; Liu et al., 2007). Asthma control was assessed with the two versions of ACT depending on child age. The first is a 5 question assessment designed to be completed by children 12 years or older (Nathan et al., 2004). The 5 items are all ranked on a 5 point scale assessing their asthma over the past 4 weeks. The brief 5 item survey was established with the internal consistency reliability of 0.84 and ratings from the ACT were in 71% to 78% agreement with specialist's ratings during development. Liu and colleagues (2007) developed the 7 item childhood asthma control test for children 4 to 11 years

of age. Caregivers responded to 3 questions assessing the child's asthma symptoms and children responded to 4 questions. In this version, the child's answer responses have written content, but are also depicted by faces. Cronbach's alpha of the 7 items at development was 0.79. Lower scores on the ACT indicate worse asthma control. For the CARE Study, the 5 item version (children 12 years and older) had excellent internal consistency ($\alpha = 0.91$) and the 7 item version (children 4 to 11 years old and caregiver) had adequate internal consistency ($\alpha = 0.75$). For Project AAIR, the 5 item version (children 12 years and older) had good internal consistency ($\alpha = 0.80$).

The Stress Index: Neighborhood Stress (Attar et al., 1994). In the 23 item stress index, children/adolescents were instructed to think about the past year when determining whether an event has occurred. If the child/adolescent responds yes an event has happened, they were then asked to rate how stressful the event was on a scale from 1 "not at all stressful/upsetting" to 3 very "stressful/upsetting". This stress index assesses three domains of stress including life transitions, circumscribed events, and exposure to violence. Higher scores indicate more perceived neighborhood related stress. Items on the neighborhood stress index completed by children from the CARE Study had good internal consistency ($\alpha = 0.80$). Items on the neighborhood stress index completed by adolescents from Project AAIR had good internal consistency ($\alpha = 0.81$).

Child Racial/Ethnic Discrimination (Landrine et al., 2006). Children/adolescents were asked two questions about their perceived racial/ethnic discrimination in Yes/No format. These questions asked if the child/adolescent had been treated badly because of their race or ethnicity or because of the color of their skin.

Pediatric Asthma Quality of Life Questionnaire (PAQLQ; Juniper et al., 1996). The

23 item, widely-used PAQLQ assesses physical, emotional, and social impairment due to asthma. Children/adolescents rated responses on a 7-point scale from 1 “extremely bothered/all of the time” to 7 “not at all bothered/none of the time”. An overall score of QOL was determined from the mean of all responses, with lower scores indicating poorer QOL. Clinical asthma control outcomes have been used to validate the PAQLQ, including β-agonist use and morning peak flow rates (Juniper et al., 1996). The PAQLQ total scale had excellent internal consistency ($\alpha = 0.93$) in the CARE Study. The PAQLQ total scale also had excellent internal consistency ($\alpha = 0.95$) in Project AAIR.

Lung Function Data. Children/adolescents used handheld spirometers (AM2 device), which measure how much air a person can push out of his or her lungs. Children/adolescents were instructed to take a deep breath and blow into the spirometer and repeat three times. Children/adolescents measured their lung function via handheld spirometer twice a day (morning and evening) for two weeks. AM2 devices yield both forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) ratings for each time. The highest values from each time point were retained and an FEV_1/FVC ratio was calculated for each time point for analyses.

EMA Data Completed in the CARE Study

EMA Surveys. The morning survey assessed stressful events that might occur that day and how confident caregivers believed they would be in managing that stressful event. The evening survey assessed: stress experienced, family functioning, child’s asthma symptoms and medication use, if the child missed school or went to the ED that day, and assistance children received from caregivers in relation to their asthma. EMA survey items related to family functioning were adapted from the FAD (Epstein et al., 1983), items related to stress were adapted from the PSS (Cohen et al., 1983), and items related to neighborhood stress were

adapted from the Stress Index: Neighborhood Stress Scale (Attar, Guerra, & Tolan, 1994). Asthma survey items were adapted from the Asthma Assessment Form (Rosier et al., 1994). All adapted EMA questions are from measures included in the initial research session battery of questionnaires.

Measures Completed in Project AAIR

Asthma-Related Caregiver Support. In the original cohort (7 to 12 year olds) asthma-related caregiver support was assessed through EMA questions derived from the Asthma Management Efficacy Scale (Bursch, Schwankovsky, Gilbert, & Zeiger, 1999). These items include: "how much did you help your child avoid being around tobacco smoke" and "how much did you help your child avoid things that he/she is allergic to?" (both rated "all of the time", "some of the time", "none of the time"); "how sure are you that you gave your child his or her medications correctly today" and "how sure were you that you could control your child's asthma at home today?" (both rated "not at all sure", "a little bit sure", "sure", "very sure"); and "how difficult or challenging did you find taking care of your child's asthma today?" (0 = "not at all" to 6 = "extremely"). Given that the adolescent/caregiver dyads did not complete EMA surveys, a brief questionnaire about caregiver support from the EMA survey items was administered to both the caregiver and adolescent to obtain multi-informant data regarding asthma-related caregiver support.

Adolescent Stress Questionnaire (ASQ; Byrne et al., 2007). The ASQ is a 58 item measure that assesses multiple domains of stress that adolescents may experience. Adolescents ranked how stressful they find each item on a 1 "not at all stressful (or is irrelevant to me)" to 5 "very stressful" scale. The measure generates 10 stress subscales: home life, school performance, school attendance, romantic relationships, peer pressure, teacher interaction, future uncertainty,

school/leisure conflict, financial pressure, and emerging adulthood. Higher scores indicate more perceived stress. The school performance and peer pressure subscales were included in the adolescent-specific cumulative risk model as assessments of stress related to school and stress related to peers. Questions on the school performance subscale assessed stress related to studying, keeping up with schoolwork, concentration, understanding of material, and teacher expectations. Questions on the peer pressure subscale assessed stress related to fitting in, physical appearance, feeling judged, and disagreements with peers. The school performance subscale had excellent internal consistency ($\alpha = 0.90$) and the peer pressure subscale had adequate internal consistency ($\alpha = 0.76$).

Conflict Behavior Questionnaire (CBQ; Robin & Foster, 1989). The CBQ is comprised of two separate forms: one for the parent and one for the adolescent that assess the parent-child relationship. Each 20 item questionnaire was replied to in a true/false format reflecting the parent-child relationship over the past two weeks. Caregivers were given one form to fill out about their relationship with the child enrolled in the study. Adolescents were presented with two forms, one for the caregiver completing the study with them and one for if they have a second caregiver to report on. The caregiver reported CBQ had excellent internal consistency ($\alpha = 0.92$). The adolescent reported CBQ of the primary caregiver had good internal consistency ($\alpha = 0.89$).

Data Analysis

All analyses except the multilevel modeling (MLM) analyses were conducted using IBM SPSS Statistics, Version 23.0 software (IBM Corp., Armonk, NY, USA). MLM analyses were conducted using SAS 9.4 software (SAS Institute, Cary, North Carolina).

Preliminary Analyses. Potential covariates (e.g., race/ethnicity, gender, child age) were assessed prior to any analyses. Correlation analyses were used to assess for continuous covariates

(e.g., child age) and analysis of variance (ANOVA) tests were conducted to assess for categorical covariates (e.g., race/ethnicity, gender). Because the cohorts were combined for the third aim, the two groups were compared on demographics to ensure there were no significant differences between the two groups. Any variable that was significantly different between the two groups was assessed as a potential covariate in combined data analyses. Skewness and kurtosis were assessed in the variables included in analyses and data transformations were conducted as needed.

Multiple Imputation. In order to include the full cohorts in analyses, multiple imputation was conducted. This was done for each data set. Pattern analyses revealed that less than 5% of data were missing in each data set; however, due to small sample size multiple imputation was still conducted. Therefore, observed results could be compared to imputed data. Pattern analysis graphs indicated that data were missing at random. The imputation method was chosen by SPSS based on the best fit for the data. Linear regression models were used for continuous variables and logistic regression models were used for dichotomous variables. Five imputations were conducted with variables that were missing data. These 5 data sets are then averaged together to calculate pooled data results. Pooled results from the 5 imputations were reported along with the original data results. Previous research suggests reporting on both complete observed data as well as pooled data for comparison and discussion of any differences between results (Klebanoff & Cole, 2008; Sterne et al., 2009; Hayati Rezvan, Lee, & Simpson, 2015). Both observed and pooled results are presented in tables in the results section.

Power Analyses. G*Power 3.1 software was used to conduct power analyses. Given that the sample size in this study was predetermined (i.e., secondary data analysis from CARE), post hoc power analyses were conducted. The hierarchical multiple regressions for each cohort (N =

60) were powered at .75 with two predictors (i.e., covariates, cumulative risk index) and .68 with three predictors (i.e., multiple covariates, cumulative risk index) for a medium effect ($f^2 = .15$). The moderation analyses for each cohort were powered at .68 with three predictors of interest (i.e., covariates, cumulative risk index, interaction term). Additionally, power was calculated for the combined group analysis (N = 120). The moderation analyses for the combined cohorts were powered at .95 with three predictors of interest (i.e., covariates, cumulative risk index, interaction term).

Cumulative Risk Models. The first hypothesis in both aims one and two involved cumulative risk models. Specifically, cumulative risk models were generated to test the following hypotheses:

1) Greater cumulative risk will be associated with worse child asthma outcomes (more ED visits and quick-relief medication use [measured via daily EMA surveys], worse lung function [measured via daily handheld spirometers], worse asthma-related QOL and asthma control [measured via self-report questionnaires]; Aim 1);

2) The new cumulative risk model for urban adolescents with asthma will include school and relationship (parent, peer, romantic) stressors, as well as poverty and neighborhood stress. I expect this new model will account for more variance in adolescent asthma outcomes than the original model for 7-12 year olds (Aim 2).

A cumulative risk score was calculated for each family. Poverty and perceived discrimination were dichotomized before being included in the risk score. For example, 1 was assigned to families that were under the poverty threshold (i.e., if the income-to-needs ratio was > 1) and 0 for families above the poverty threshold. Continuous variables, such as neighborhood stress, were standardized before being included in the model. Instead of dichotomizing using a

sample-specific quartile cut-off value (Sameroff et al., 1993), we standardized continuous variables to maintain the continuous nature of the variable (Everhart et al., 2008; Whisman & McClelland, 2005). Two cumulative risk scores were calculated for the adolescent sample, one that matches the cumulative risk model calculated for the younger cohort and one that contains adolescent specific stressors (e.g., stress related to school and relationships [parents and peers]). A new cumulative risk score was calculated for all families in aim three. The data were combined and the continuous variables were standardized for the combined cohort.

In accord with other cumulative risk research, the scores of the risk factors were summed to create the cumulative risk index. A series of hierarchical multiple regression analyses were conducted to assess the ability of the cumulative risk model to predict each asthma outcome (e.g., QOL, asthma control, ED visits, medication use). Covariates were entered in the first step of the model and the cumulative risk score was entered in the second step. In the second aim, effect sizes were examined to compare the original model to the adolescent-specific model in determining which model better predicted adolescent asthma outcomes. In addition, hierarchical regressions were conducted for each risk factor to assess if the cumulative risk models were more effective in predicting each asthma outcome than one single risk factor. For the adolescent cohort, the cumulative risk model with the greater effect size (i.e., the original cumulative risk model or the cumulative risk model containing adolescent-specific stressors*)* was assessed in relation to single predictors. These analyses were only conducted if the cumulative risk models were a significant predictor of the asthma outcome.

False Discovery Rate (FDR) Correction. To control for Type I error, the Benjamini-Hochberg (1995) FDR correction was conducted in each group of analyses that had significant results. This type of correction is not as conservative as other approaches (e.g., Bonferroni

correction) and allows for greater power. FDR corrections help correct for Type 1 error, while also maximizing the reduction of Type II errors. In this approach, the unadjusted p-values are ordered in ascending order (i.e., $p_1 \leq p_2 \leq \ldots \leq p_m$). The largest p-value remains the same. Starting with the second highest p-value, each value is multiplied by the total number of tests over its rank in the list. For example, for the second highest p-value the calculation would be p-value*(n/n-1). If the adjusted p-value is less than 0.05, the test will be significant.

Moderation Analyses. The second hypothesis in both aims one and two, along with aim three were tested with moderation analyses. Specifically, moderation analyses tested the following hypotheses:

1) Higher levels of caregiver support related to asthma care will minimize the impact of cumulative risk on negative child asthma outcomes in the younger cohort (7 to 12 year olds; Aim 1);

2) Adolescents that experience more stressors and less asthma-specific support from caregivers will have worse asthma outcomes (Aim 2);

3) Age will significantly moderate the association between cumulative risk and asthma outcomes, such that younger children with higher cumulative risk scores will have worse asthma outcomes (Aim 3).

An aggregate score of caregiver support was calculated from specific EMA questions in the 7 to 12 year old sample (CARE) and from the caregiver support items in the 13 to 17 year old sample (AAIR). Both adolescents and caregivers reported on the support items in the older sample, and adolescent responses were used in analyses. Moderation analyses were conducted to determine whether caregiver support related to the child/adolescent's asthma care buffers the association between cumulative risk and asthma outcomes. Moderation analyses were run for

both age groups. The cumulative risk model with the largest effect size in the adolescent group was used in moderation analyses for this cohort.

The independent variable (i.e., cumulative risk) and moderating term were centered (i.e., caregiver asthma-related support) and an interaction term was created before moderation analyses were conducted. Covariates were entered into block 1, cumulative risk scores into block 2, caregiver asthma-related support scores into block 3, and the interaction term into block 4. For significant tests, post-hoc probing of the moderation effects were conducted (Holmbeck, 2002). The simple slopes of each group were calculated and the moderations were graphed. Figure 2 gives an example of the moderation analyses to determine whether asthma-related caregiver support buffers the association between cumulative risk and asthma outcomes. This model was tested in the younger sample.

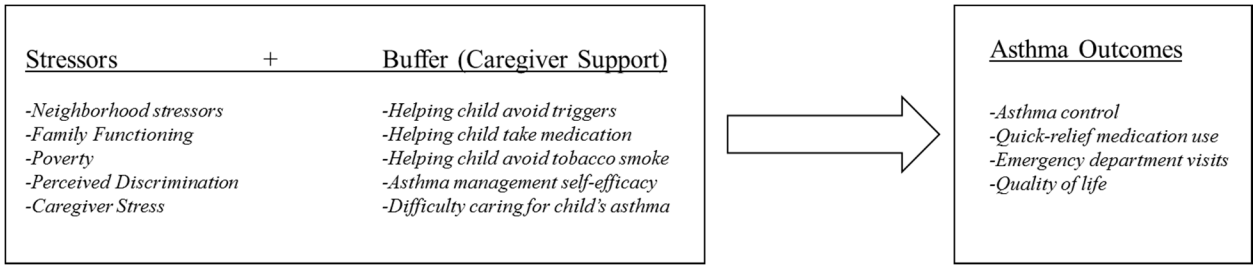

Figure 2. *Moderation of asthma-related caregiver support on the association between cumulative risk and asthma outcomes.*

In addition, for the third aim, the data sets were combined to assess child age as a moderator in associations between the cumulative risk index and asthma outcomes. This also allowed for higher powered analyses with all 121 families included. The original cumulative risk model generated for the younger cohort was assessed in the combined group (e.g., poverty, neighborhood stress, perceived discrimination, family functioning, caregiver stress). Because several of the variables were continuous, a new cumulative risk score was calculated for each

family with the standardized variables for the combined group.

Multilevel Modeling Analyses. The last hypothesis in aim one was tested using multilevel modeling. Specifically, multilevel modeling tested the following hypothesis:

1) Cumulative risk will be associated with momentary-level child asthma outcomes (daily EMA responses) over a two week period. Specifically, cumulative risk will be associated with momentary-level child ED visits and quick-relief medication use, and daily lung function assessments.

Due to the longitudinal nature of the spirometry data, MLM analyses were used to test whether the cumulative risk model predicted child lung function across the monitoring period. Spirometers yield both FEV_1 and FVC ratings and the highest values at each time point were retained. An FEV_1/FVC ratio was calculated for each time point for analyses. Analyses on both child morning lung function and evening lung function were conducted. The association between cumulative risk and adolescent lung function was also assessed with MLM analyses.

A random coefficients multilevel model was tested to account for the nested structure of the EMA data. A multilevel model was run for each EMA asthma outcome (e.g., daily symptoms, daily quick-relief medication use, ED visits, lung function). The PROC MIXED procedure in SAS was used to estimate the models. Covariates were entered into all models along with day of study (time variable). Predictors were centered before being entered into the models.

Results

CARE Study Analyses

Descriptive Information

Descriptive information of the CARE cohort (7 to 12 year olds) is presented in Table 4. Approximately 48.3% of the participants had ACT scores less than 19, which indicated that their asthma may be poorly controlled. Also, 68.85% of children had been to the ED at least once in the last 12 months, with 59.05% reporting more than one visit to the ED due to asthma. Based on the needs-to-income ratio, 75.0% of families lived below the poverty line.

Table 4.

Measure Scores of the CARE Cohort (7-12 year olds)

Measure	**Mean (SD)**	**Score Range**
Child QOL	5.24 (1.29)	2.48 – 7.00
Asthma Control (ACT)	18.60 (5.16)	4 – 26
Quick-Relief Medication Use	45% - 0 to 2 days a week 26.7% - 3 to 6 days a week 26.7% - Every day of the week use 1.7% - Did not use their medication	
ED Visits	2.07 (2.11)	0 – 10
Below Poverty Line	75%	
Child discrimination – Yes	22%	
Neighborhood Stress	11.08 (8.37)	0 – 40
Caregiver Perceived Stress (PSS)	24.95 (6.00)	10 – 41
Family Functioning (FAD General Functioning Subscale)	1.89 (0.36)	1.17 – 2.58
Asthma-related Caregiver Support	6.39 (2.15)	3.00 – 11.05

CARE Covariate Testing

Child age was significantly correlated with child QOL ($r = .30$, $p = .02$). Child age was not correlated with any of the other outcomes. QOL, asthma control, medication use, and ED visits did not differ by child sex or by child race/ethnicity. Income was not examined as a

potential covariate due to poverty being included in the cumulative risk model. Skewness and kurtosis were examined for variables included in analyses. Skewness and kurtosis of all variables were less than |2|, with only neighborhood stress being greater than |1.5| in kurtosis.

CARE Cumulative Risk Regression Analyses

Observed data results found the cumulative risk model to significantly predict QOL after controlling for child age ($\Delta R^2 = .07$, ΔF (1, 52) = 4.23, $p = .045$). Higher cumulative risk scores were related to lower QOL scores; however, pooled analyses revealed only a trend in significance (results of all pooled analyses can be found in Table 5). The cumulative risk model did not predict asthma control based on observed data ($R^2 = .04$, F (1, 53) = 2.33, $p = .13$) or pooled analyses. In addition, the cumulative risk model did not predict quick-relief medication use based on observed data ($R^2 = .04$, F (1, 53) = 2.04, $p = .16$) or pooled analyses. Logistic regression analysis revealed the cumulative risk model did not predict whether or not the child was seen in the ED in the last year based on observed data (χ^2 (1) = .00, p = .998). According to the Wald criterion, the cumulative risk model was not a significant predictor of whether the child had been to the ED due to asthma (b = .00, SE = .33, $\chi^2(1) = .00$, $p = .998$). Pooled analyses had the same non-significant findings.

Table 5.

Cumulative Risk as a Predictor of Child (7-12) Asthma Outcomes

Outcome	**Model**	***B***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	-.15	.08	-0.30, -0.004	-2.06	54	.045*
	Pooled	-.13	.07	-0.27, 0.01	-1.78	60	.07
Asthma Control	Observed	-.47	.31	-1.09, 0.15	-1.53	54	.13
	Pooled	-.51	.31	-1.11, 0.09	-1.67	60	.095

Quick-Relief Medication Use	Observed	.07	.05	-0.03, 0.18	1.43	54	.16
	Pooled	.07	.05	-0.03, 0.17	1.37	60	.17
ED Visits	Observed	.00	.33				.998
	Pooled	.02	.12				.87

Note. *$p < .05$

Cumulative Risk and QOL in the CARE Cohort

The cumulative risk model predicted child QOL after controlling for child age based on observed data. Therefore, using only observed data, the cumulative risk model was evaluated to determine whether it was a stronger predictor of QOL than any one individual stressor related to child QOL. Using the observed data, the correlations between each stressor and QOL were examined. Neighborhood stress ($r = -.30$, $p = .02$) and child perceived discrimination ($r = -.34$, $p = .01$) were correlated with child QOL; therefore, cumulative risk was examined against these two stressors. Child age was controlled for in all analyses. The cumulative risk model did not predict child QOL after controlling for child perceived discrimination and child age according to the observed data ($\Delta R^2 = .04$, $\Delta F\ (1, 51) = 2.48$, $p = .12$; Table 6). Additionally, the cumulative risk model did not predict child QOL after controlling for neighborhood stress and child age according to the observed data ($\Delta R^2 = .03$, $\Delta F\ (1, 51) = 1.55$, $p = .22$).

Table 6.

Cumulative Risk and Individual Stressors Predicting Child QOL

Stressor	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
Neighborhood Stress	Observed	-.12	.10	-0.31, -0.07	-1.24	54	.22
Child Discrimination	Observed	-.13	.08	-0.29, 0.04	-1.57	54	.12

Note. *$p < .05$

Cumulative Risk and Asthma Control, Quick-Relief Medication Use, and ED Visits

The cumulative risk model was not a significant predictor of asthma control, quick-relief medication use, or ED visits in the first set of analyses. Therefore, follow-up analyses assessing whether the cumulative risk model was a stronger predictor than any one stressor were not conducted involving these three outcomes.

Asthma-Related Caregiver Support Moderation Analyses in the CARE Cohort

Asthma-related caregiver support scores were calculated from 5 questions that caregivers responded to in the EMA surveys (See Table 7). EMA data were collapsed across days to assess between-group differences. Averages were calculated from caregiver EMA responses for each of the five questions listed in Table 7. The five averages were then added together to compute a total score of asthma-related caregiver support. This score was used as the moderator variable in analyses. Scores could range from 2 to 20, with lower scores indicating more support. Asthma-related caregiver support was correlated with both child QOL (r = -.37, p = .005) and asthma control (r = -.34, p = .01).

Table 7.

Asthma-Related Support Items

Question	Answer Options and Scoring
How sure are you that you gave your child his or her medications correctly today?	1 – Very sure 2 – Sure 3 – A little bit sure 4 – Not at all sure
How difficult or challenging did you find taking care of your child's asthma today?	0 – Not at all 1 2 3 4 5 6 – Extremely

How much did you help your child avoid things that he/she is allergic to?	1 – All of the time 2 – Some of the time 3 – None of the time 0 – My child does not have allergies
How much did you help your child avoid being around tobacco smoke?	1 – All of the time 2 – Some of the time 3 – None of the time 0 – My child was not at risk for being around tobacco smoke
How sure were you that you could control your child's asthma at home today?	1 – Very sure 2 – Sure 3 – A little bit sure 4 – Not at all sure

The cumulative risk model did not significantly predict child asthma control. However, the asthma-related caregiver support score was significantly correlated with both QOL and asthma control; therefore, it was examined as a moderator between cumulative risk and these two outcomes. Asthma-related caregiver support was approaching significance in moderating the association between cumulative risk and asthma control based on observed data ($\Delta R^2 = .07$, ΔF (1, 47) = 3.96, $p = .052$). However, this moderation was not significant in pooled analyses (Table 8). Asthma-related caregiver support did not moderate the association between cumulative risk and child QOL according to both the observed data ($\Delta R^2 = .01$, ΔF (1, 47) = 0.36, $p = .55$) and pooled analyses.

Table 8.

Moderating Role of Asthma-Related Caregiver Support in the Younger Cohort (7-12)

Outcome	**Model**	***B***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	-.02	.04	-0.10, 0.05	-0.60	50	.55
	Pooled	-.03	.03	-0.09, 0.04	-0.87	60	.39
Asthma Control	Observed	-.30	.15	-0.60, 0.003	-1.99	50	.052
	Pooled	-.17	.15	-0.48, 0.14	-1.14	60	.26

Note. *$p < .05$

Child Lung Function and EMA Analyses

Covariance testing revealed that the toeplitz covariance structure was the best fitting structure for both lung function models. Cumulative risk scores calculated from baseline assessments were used to predict longitudinal lung function. Cumulative risk scores were not a significant predictor of morning lung function assessments (b = -.01, SE = 0.01, t(37) = -1.09, p = .28). However, cumulative risk scores were a significant predictor of evening lung function assessments (b = -.02, SE = 0.01, t(37) = -3.06, p = .004), with higher cumulative risk associated with worse lung function. One advantage of MLM is its ability to account for missing data points; thus, observed data were used for MLM analyses.

Variance components was the best fitting covariance structure for the quick-relief medication use model. Cumulative risk scores were not a significant predictor of daily child quick-relief medication use as reported by caregivers (b = -.04, SE = 0.08, t(397) = -0.46, p = .65). Covariance testing revealed that compound symmetry was the best fitting covariance structure for the ED visits model. Additionally, the cumulative risk model did not significantly predict ED visits due to asthma over the monitoring period as reported by caregivers (b = .003, SE = 0.001, t(397) = 1.06, p = .29).

Project AAIR Analyses

AAIR Descriptive Information

Descriptive information of the AAIR cohort (13-17 year olds) are presented in Table 9. Approximately 63.3% of the adolescents had ACT scores that indicated their asthma may be poorly controlled (i.e., scores of 19 or below). Also, 71.7% of adolescents had been to the ED at least once in the last 12 months, with 45.0% reporting more than one visit to the ED due to asthma. Based on the needs-to-income ratio, 76.7% of families lived below the poverty line.

CBQ scores for the adolescent-reported scale on their relationship with their primary caregiver ranged from 0 to 18 (M = 4.28, SD = 4.46), with higher scores indicating more conflict. Adolescent reports of conflict behavior correlated with caregiver reports (r = 0.54, p < .001).

Table 9.

Measure Scores of the AAIR Cohort (13-17 year olds)

Measure	Mean (SD)	Score Range
Child QOL	5.32 (1.28)	2.17 – 7.00
Asthma Control (ACT)	17.85 (4.98)	7 – 25
Quick-Relief Medication Use	46.6% - 0-2 days a week 31.0% - 3-6 days a week 22.4% - Every day of the week use	
ED Visits	1.55 (1.44)	0 – 6
Below Poverty Line	76.7%	
Child discrimination – Yes	11.7%	
Neighborhood Stress	12.30 (8.76)	0 – 33
Caregiver Perceived Stress (PSS)	25.53 (8.23)	9 – 54
Family Functioning (FAD General Functioning Subscale)	1.84 (0.46)	1.00 – 2.92
Caregiver-Adolescent Conflict (CBQ- Adolescent Report)	4.28 (4.46)	0 – 18
School Performance (ASQ)	17.82 (8.32)	7 – 34
Peer Pressure (ASQ)	10.83 (4.79)	7 – 32
Adolescent reported asthma-related caregiver support	6.87 (2.80)	2 – 12

AAIR Covariate Testing

Adolescent age was significantly correlated with quick-relief medication use (r = .33, p = .01) and ED visits (r = .39, p = .002). Adolescent age was not correlated with any of the other outcomes. Both adolescent QOL (F(1, 58) = 8.11, p = .01) and asthma control (F(1, 58) = 8.25, p

= .01) differed by adolescent sex. Male adolescents had better QOL (M = 5.80, SD = 1.28) than female adolescents (M = 4.90, SD = 1.15). Additionally, male adolescents had higher levels of asthma control (M = 19.71, SD = 4.72) than female adolescents (M = 16.22, SD = 4.69). None of the outcome variables differed by adolescent race/ethnicity. Income was not examined as a potential covariate due to poverty being included in the cumulative risk model. Skewness and kurtosis were examined for variables included in analyses. Skewness and kurtosis of all variables were less than |2|, except for the peer pressure ASQ subscale scores where the skewness was 2.12 and the kurtosis 6.06. A log transformation was conducted on this variable.

Original Cumulative Risk Model Analyses in the AAIR Cohort

The original cumulative risk model (i.e., poverty, adolescent perceived discrimination, neighborhood stress, caregiver perceived stress, family functioning) was first examined as a predictor of asthma outcomes in the adolescent cohort. Observed data results found the cumulative risk model to significantly predict QOL after controlling for adolescent sex (ΔR^2 = .22, ΔF (1, 56) = 17.95, $p < .001$). Pooled analyses had the same finding (see Table 10 for results of all pooled analyses). Higher cumulative risk scores were related to worse QOL scores. Additionally, the cumulative risk model was a significant predictor of asthma control after controlling for adolescent sex based on both observed data (ΔR^2 = .14, ΔF (1, 56) = 10.46, p = .002) and pooled analyses. Higher cumulative risk scores were associated with lower ACT scores (i.e., poorer asthma control). The cumulative risk model was trending toward significance as a predictor of quick-relief medication use after controlling for adolescent age based on both observed data (ΔR^2 = .05, ΔF (1, 54) = 3.32, p = .07) and pooled analyses. Higher cumulative risk scores were associated with more quick-relief medication use. Logistic regression analysis revealed that the cumulative risk model did not predict whether or not the adolescent was seen in

the ED in the last year after controlling for adolescent age. The overall model was significant based on observed data (χ^2 (2) = 10.25, p = .006), but according to the Wald criterion, the cumulative risk model was not a significant predictor (b = .10, SE = .13, χ^2(1) = .63, p = .43). Pooled analyses had the same non-significant finding.

Table 10.

Original Cumulative Risk Model as a Predictor of Adolescent Asthma Outcomes

Outcome	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	-.24	.06	-0.35, -0.13	-4.24	58	.00*
	Pooled	-.24	.06	-0.35, -0.13	-4.23	59	.00*
Asthma Control	Observed	-.74	.23	-1.19, -0.28	-3.23	58	.002*
	Pooled	-.76	.23	-1.21, -0.32	-3.34	59	.001*
Quick-Relief Medication Use	Observed	.07	.04	-0.01, 0.15	1.82	56	.07
	Pooled	.07	.04	-0.004, 0.15	1.87	59	.06
ED Visits	Observed	.10	.13				.43
	Pooled	.10	.13				.45

Note. *p < .05

Adolescent-Specific Cumulative Risk Model Analyses

Next, the adolescent-specific cumulative risk model of stress (i.e., poverty, neighborhood stress, stress related to school, caregiver-adolescent conflict [relationship with parent], peer pressure) was examined as a predictor of asthma outcomes in the adolescent cohort. The adolescent-specific cumulative risk model significantly predicted QOL after controlling for adolescent sex based on the observed data (ΔR^2 = .39, ΔF (1, 54) = 42.19, p < .001). Pooled analyses had the same finding (Table 11). Higher cumulative risk scores were related to lower QOL scores. Additionally, the adolescent-specific cumulative risk model was a significant predictor of asthma control after controlling for adolescent sex based on both observed data (ΔR^2 = .27, ΔF (1, 54) = 24.18, p < .001) and pooled analyses. Higher cumulative risk scores were

associated with lower ACT scores (i.e., poorer asthma control). The adolescent-specific cumulative risk model was not a significant predictor of quick-relief medication use after controlling for adolescent age based on both observed data ($\Delta R^2 = .01$, $\Delta F\ (1, 52) = 0.73$, $p = .40$) and pooled analyses. Logistic regression analysis revealed that the cumulative risk model did not predict whether or not the adolescent was seen in the ED in the last year after controlling for adolescent age. The overall model was significant based on observed data ($\chi^2\ (2) = 11.29$, $p = .004$), but according to the Wald criterion, the cumulative risk model was not a significant predictor (b = .17, SE = .12, $\chi^2(1) = 2.16$, $p = .14$). Pooled analyses had the same non-significant finding.

Table 11.

Adolescent-Specific Cumulative Risk Model as a Predictor of Adolescent Asthma Outcomes

Outcome	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	-.28	.04	-0.36, -0.19	-6.50	56	.00*
	Pooled	-.27	.04	-0.35, -0.18	-6.33	59	.00*
Asthma Control	Observed	-.92	.19	-1.30, -0.55	-4.92	56	.00*
	Pooled	-.92	.18	-1.27, -0.57	-5.21	59	.00*
Quick-Relief Medication Use	Observed	.03	.04	-0.04, 0.10	0.85	53	.40
	Pooled	.03	.03	-0.04, 0.09	0.81	59	.42
ED Visits	Observed	.17	.12				.14
	Pooled	.18	.12				.12

Note. *$p < .05$

Comparison of the Two Cumulative Risk Models in Adolescents

Based on effect sizes, the adolescent-specific cumulative risk model was a more robust predictor of adolescent QOL and asthma control. The adolescent-specific model (including adolescent sex) accounted for 50.2% of the variance in adolescent QOL, with a 38.9% change in variance due to cumulative risk; on the other hand, the original cumulative risk model accounted

for 32.7% of the variance, with a 21.6% change in variance due to cumulative risk. The adolescent-specific model (including adolescent sex) accounted for 40.0% of the variance in asthma control, with a 26.9% change in variance due to cumulative risk; the original cumulative risk model accounted for 25.2% of the variance, with a 14.0% change in variance due to cumulative risk.

The original cumulative risk model that was tested in the younger cohort was a more robust predictor of quick-relief medication use than the adolescent-specific model. The original model (including adolescent age) accounted for 15.9% of the variance in quick-relief medication use, with a 5.2% change in variance due to cumulative risk; the adolescent-specific cumulative risk model accounted for 11.2% of the variance, with a 1.2% change in variance due to cumulative risk.

Adolescent-Specific Cumulative Risk and QOL

Due to the adolescent-specific cumulative risk model being a more robust predictor of adolescent QOL, this model was assessed to see if it was a stronger predictor than any one individual stressor. Adolescent QOL was correlated with all 5 stressors included in the model: poverty (r = -.34, p = .01), neighborhood stress (r = -.48, p < .001), school performance (r = -.65, p < .001), peer pressure (r = -.41, p = .001), and caregiver-adolescent conflict (r = -.40, p = .002). Therefore, the adolescent-specific cumulative risk model was examined in comparison to all individual stressors. The adolescent-specific cumulative risk model was a stronger predictor of QOL than poverty, controlling for adolescent sex according to both the observed data (ΔR^2 = .32, ΔF (1, 53) = 33.63, p < .001) and pooled analyses (Table 12). The adolescent-specific cumulative risk model was a significant predictor of QOL after controlling for the effects of neighborhood stress and child sex according to both the observed data (ΔR^2 = .20, ΔF (1, 53) =

21.54, $p < .001$) and pooled analyses. Additionally, the adolescent-specific cumulative risk model was a more robust predictor of QOL than stress related to school performance, controlling for child sex according to both the observed data ($\Delta R^2 = .09$, ΔF (1, 53) = 10.21, $p = .002$) and pooled analyses. The adolescent-specific cumulative risk model was a significant predictor of QOL after controlling for the effects of peer pressure and child sex according to both the observed data ($\Delta R^2 = .29$, ΔF (1, 53) = 31.47, $p < .001$) and pooled analyses. Lastly, the adolescent-specific cumulative risk model was a more robust predictor of QOL than stress related to caregiver-adolescent conflict, controlling for child sex, according to both the observed data ($\Delta R^2 = .23$, ΔF (1, 53) = 24.38, $p < .001$) and pooled analyses. All findings were still significant with the FDR correction.

Table 12.

Adolescent-Specific Cumulative Risk and Individual Stressors Predicting Adolescent QOL

Stressor	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
Poverty	Observed	-.28	.05	-0.37, -0.18	-5.80	56	.00*
	Pooled	-.26	.05	-0.36, -0.17	-5.48	59	.00*
Neighborhood Stress	Observed	-.26	.06	-0.38, -0.15	-4.64	56	.00*
	Pooled	-.26	.06	-0.38, -0.15	-4.69	59	.00*
School Performance	Observed	-.20	.06	-0.33, -0.08	-3.20	56	.002*
	Pooled	-.18	.06	-0.30, -0.05	-2.83	59	.005*
Peer Pressure	Observed	-.32	.06	-0.43, -0.20	-5.61	56	.00*
	Pooled	-.30	.06	-0.41, -0.19	-5.36	59	.00*
Caregiver-Adolescent Conflict	Observed	-.32	.07	-0.45, -0.19	-4.94	56	.00*
	Pooled	-.31	.06	-0.43, -0.19	-4.99	59	.00*

Note. *$p < .05$

Adolescent-Specific Cumulative Risk and Asthma Control

The adolescent-specific cumulative risk model was also a more robust predictor of asthma control; thus, this model was assessed to determine whether it was a stronger predictor than any one individual stressor. Adolescent asthma control was correlated with all 5 stressors included in the model: poverty (r = -.36, p = .005), neighborhood stress (r = -.40, p = .001), school performance (r = -.59, p < .001), peer pressure (r = -.35, p = .006), and caregiver-adolescent conflict (r = -.33, p = .01). Therefore, the adolescent-specific cumulative risk model was examined in comparison to all individual stressors. The adolescent-specific cumulative risk model was a stronger predictor of asthma control than poverty, controlling for adolescent sex according to both the observed data (ΔR^2 = .19, ΔF (1, 53) = 17.31, p < .001) and pooled analyses (Table 13). The adolescent-specific cumulative risk model was a significant predictor of asthma control after controlling for effect of neighborhood stress and child sex according to both the observed data (ΔR^2 = .16, ΔF (1, 53) = 14.37, p < .001) and pooled analyses. The adolescent-specific cumulative risk model was a more robust predictor of asthma control than stress related to school performance, controlling for child sex according to the observed data (ΔR^2 = .04, ΔF (1, 53) = 4.12, p = .05) and pooled analyses. The adolescent-specific cumulative risk model was a significant predictor of asthma control after controlling for the effect of peer pressure and child sex according to both the observed data (ΔR^2 = .22, ΔF (1, 53) = 19.88, p < .001) and pooled analyses. Finally, the adolescent-specific cumulative risk model was a more robust predictor of asthma control than stress related to caregiver-adolescent conflict, controlling for child sex according to both the observed data (ΔR^2 = .15, ΔF (1, 53) = 14.10, p < .001) and pooled analyses. All findings in this group of analyses were still significant with the FDR correction.

Table 13.

Adolescent-Specific Cumulative Risk and Individual Stressors Predicting Adolescent Asthma Control

Stressor	Model	*b*	SE	95% CI	*t*	*df*	*p*-value*
Poverty	Observed	-.86	.21	-1.27, -0.44	-4.16	56	.00*
	Pooled	-.84	.20	-1.22, -0.46	-4.30	59	.00*
Neighborhood Stress	Observed	-.94	.25	-1.44, -0.45	-3.80	56	.00*
	Pooled	-.96	.24	-1.41, -0.51	-4.08	59	.00*
School Performance	Observed	-.56	.28	-1.11, -0.01	-2.03	56	.05*
	Pooled	-.63	.27	-1.15, -0.11	-2.39	59	.02*
Peer Pressure	Observed	-1.10	.25	-1.60, -0.61	-4.46	56	.00*
	Pooled	-1.06	.24	-1.53, -0.60	-4.52	59	.00*
Caregiver-Adolescent Conflict	Observed	-1.07	.29	-1.64, -0.50	-3.76	56	.00*
	Pooled	-1.02	.26	-1.52, -0.52	-3.97	59	.00*

Note. *$p < .05$

Cumulative Risk, Quick-Relief Medication Use, and ED Visits

The original cumulative risk model was only trending toward significance in predicting adolescent quick-relief medication use and the adolescent-specific model did not predict quick-relief medication use; therefore, follow-up analyses assessing whether a cumulative risk model was a stronger predictor than any one stressor were not conducted. Additionally, neither cumulative risk model was a significant predictor of adolescent ED visits (or trending towards significance); therefore, follow-up analyses were not conducted.

Asthma-Related Caregiver Support Moderation Analyses in the AAIR Cohort

Asthma-related caregiver support was assessed by the same 5 questions as in the younger cohort (see Table 7), except questions were administered through paper questionnaires at the research session to both adolescents and caregiver versus via EMA on smartphones. Scores could range from 2 to 20, with lower scores indicating more support from the caregiver.

Adolescent and caregiver reports of asthma-related caregiver support were not correlated (r = .13, p = .35). In an item by item analysis, the only question in which caregiver and adolescent responses were correlated was, "How difficult or challenging did you find taking care of your child's/your asthma in the last week?" (r = .29, p = .02). Caregiver reported asthma-related caregiver support was correlated with quick-relief medication use (r = .29, p = .03). Adolescent reported asthma-related caregiver support was correlated with QOL (r = -.46, p = .001), asthma control (r = -.36, p = .007), and was trending toward significance for ED visits (r = .26, p = .056). Given the central focus on adolescent asthma outcomes in the present study, the adolescent report of asthma-related caregiver support was used in moderation analyses.

Asthma-related caregiver support was assessed as a moderator in the association between the adolescent-specific cumulative risk model and adolescent outcomes (e.g., QOL, asthma control, quick-relief medication use, ED visits). One moderation with asthma control as the outcome variable was significant with the pooled data. Adolescent reported asthma-related caregiver support did not moderate the association between cumulative risk and asthma control based on the observed data (ΔR^2 = .02, ΔF (1, 46) = 1.77, p = .19). However, pooled analyses revealed a significant moderator effect (Table 14). Asthma-related caregiver support did not moderate the association between cumulative risk and QOL, controlling for adolescent sex, according to both the observed data (ΔR^2 = .00, ΔF (1, 46) = 0.01, p = .92) and pooled analyses. Additionally, asthma-related caregiver support did not moderate the association between cumulative risk and quick-relief medication use, controlling for adolescent age, according to both the observed data (ΔR^2 = .00, ΔF (1, 45) = 0.01, p = .93) and pooled analyses. Based on the Wald criterion, results of the logistic regression model predicting whether the adolescent was seen in

the ED in the last year were not significant (b = .02, SE = .05, $\chi^2(1) = 0.17$, $p = .68$). Pooled analyses had the same non-significant findings of the interaction term.

Post-hoc probing analyses were conducted with pooled data for the significant moderator effect of asthma-related caregiver support on the association between adolescent-specific cumulative risk and adolescent asthma control. The simple slope for the lower support group was significant (b = -1.21, 95% CI [-1.75, -0.68], $p < .001$). The simple slope for the higher support group was also significant (b = -0.56, 95% CI [-1.04, -0.08], $p = .02$). While the association was significant for both groups, the adolescents that received lower levels of asthma-related caregiver support had a more dramatic decrease in asthma control as their cumulative risk levels rose (see Figure 3).

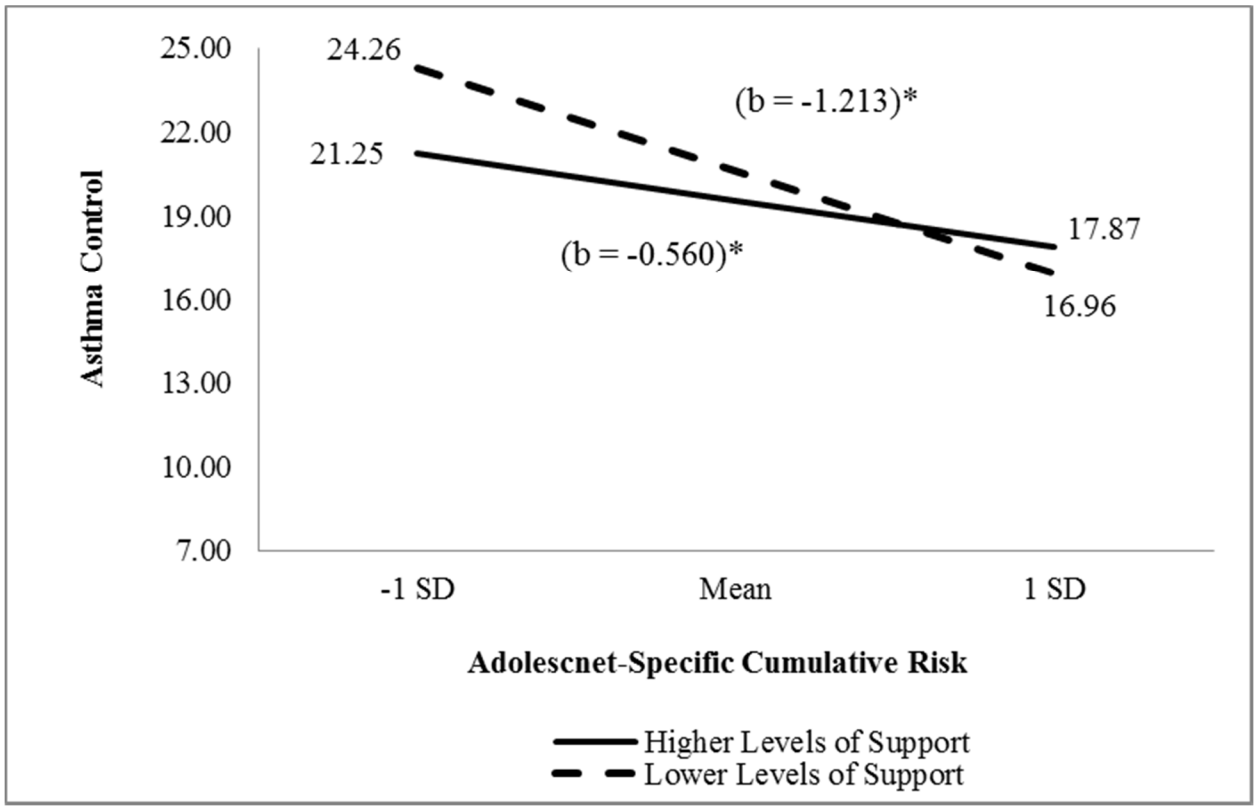

Figure 3. *Regression lines for association between adolescent-specific cumulative risk and adolescent asthma control moderated by asthma-related caregiver support.*

Note. b = unstandardized regression coefficient (i.e., simple slope)

*$p < .05$

Table 14.

Moderating Role of Asthma-Related Caregiver Support between Adolescent-Specific Cumulative Risk and Adolescent Asthma Outcomes

Outcome	Model	*b*	SE	95% CI	*t*	*df*	*p*-value*
QOL	Observed	-.002	.02	-0.04, 0.03	-0.10	50	.92
	Pooled	.001	.02	-0.03, 0.03	-0.08	59	.93
Asthma Control	Observed	.09	.07	-0.05, 0.23	1.33	50	.19
	Pooled	.12	.06	-0.003, 0.24	1.92	59	.05*
Quick-Relief Medication Use	Observed	.001	.01	-0.03, 0.03	0.08	49	.93
	Pooled	.001	.01	-0.02, 0.03	0.09	59	.93
ED Visits	Observed	.02	.05				.68
	Pooled	.02	.05				.68

Note. *$p < .05$

Adolescent Lung Function Analyses

Covariance testing revealed that the toeplitz covariance structure was the best fitting structure for all models. Cumulative risk scores calculated from baseline assessments were used to predict longitudinal lung function. Both cumulative risk models were tested as a predictor of lung function. Observed data were used for MLM analyses. The original cumulative risk model was not a significant predictor of adolescent lung function assessments ($b = .005$, SE = 0.003, $t(54) = 1.80$, $p = .078$). The adolescent-specific cumulative risk scores were not a significant predictor of adolescent lung function assessments ($b = .001$, SE = 0.002, $t(52) = 0.30$, $p = .77$).

Combined Cohort Analyses

Demographic Differences Among the Cohorts

The two cohorts were compared on demographic variables and findings are presented in Table 15. Any significant differences between the two groups were tested as potential covariates in combined group analyses. Additionally, the season in which the initial research session took place was examined for differences between the two cohorts and included in Table 15.

Table 15.

Combined Cohorts Caregiver and Child/Adolescent Demographics

Demographic Items	**CARE Study** N = 61	**Project AAIR** N = 60	**Total Sample** N = 121	**P-value***
Caregiver				
Age, *M years (SD)*	37.90 (9.18)	41.83 (8.42)	39.83 (9.00)	.02*
Race/Ethnicity, *n* (%)				.58
African American/Black	55 (90.2)	55 (91.7)	110 (90.9)	
Latino	0 (0.0)	2 (3.4)	2 (1.7)	
Caucasian/White	4 (6.6)	1 (1.7)	5 (4.1)	
Mixed/Multi-Racial	1 (1.6)	1 (1.7)	2 (1.7)	
Other	1 (1.6)	1 (1.7)	2 (1.7)	
Relation to child, *n* (%)				.02*
Biological Mother	49 (80.3)	54 (90.0)	103 (85.1)	
Step or Adoptive Mother	3 (4.9)	0 (0.0)	3 (2.5)	
Biological Father	7 (11.5)	1 (1.7)	8 (6.6)	
Grandmother	1 (1.6)	5 (8.3)	6 (5.0)	
Other	1 (1.6)	0 (0.0)	1 (0.8)	
Relationship Status, *n* (%)				.58
Single/Never Married	40 (65.6)	35 (58.3)	75 (62.0)	
Married	9 (14.8)	13 (21.7)	22 (18.2)	
Separated	6 (9.8)	3 (5.0)	9 (7.4)	
Divorced	4 (6.6)	7 (11.7)	11 (9.1)	
Widowed	2 (3.3)	2 (3.3)	4 (3.3)	
Education, *n* (%)				.35
Less than a high school	11 (18.0)	17 (28.3)	28 (23.1)	
High school degree	20 (32.8)	18 (30.0)	38 (31.4)	
Some college	21 (34.4)	21 (35.0)	42 (34.7)	
College degree or higher	9 (14.8)	4 (6.7)	13 (10.7)	
Income (Past Month), *n* (%)				.85
Less than $1,000	23 (37.7)	24 (40.0)	47 (38.8)	
$1,000 - $1,999	15 (24.6)	16 (26.7)	31 (25.6)	
$2,000 - $3,999	16 (26.2)	17 (28.3)	33 (27.3)	
Greater than $4,000	5 (8.2)	3 (5.0)	8 (6.6)	

Household size, *M (SD)*	4.31 (1.64)	4.47 (1.96)	4.39 (1.80)	.64
Child/Adolescent				
Age, *M years (SD)*	9.59 (1.52)	14.73 (1.38)	12.14 (2.96)	.00*
Sex (Male), *n* (%)	42 (68.9)	28 (46.7)	70 (57.9)	.01*
Race/Ethnicity, *n* (%)				.44
African American/Black	55 (90.2)	56 (93.3)	111 (91.7)	
Latino	0 (0.0)	2 (3.4)	2 (1.7)	
Caucasian/White	4 (6.6)	1 (1.7)	5 (4.1)	
Mixed/Multi-Racial	1 (1.6)	1 (1.7)	2 (1.7)	
Other	1 (1.6)	0 (0.0)	1 (0.8)	
Season of Visit				.16
Fall	24 (39.3)	29 (48.3)	53 (43.8)	
Winter	8 (13.1)	12 (20.0)	20 (16.5)	
Spring	14 (23.0)	13 (21.7)	27 (22.3)	
Summer	15 (24.6)	6 (10.0)	21 (17.4)	

Note. Household size is mean number of individuals including children in home; *p-value results are from analyses assessing differences in variables across CARE and AAIR cohorts.

Covariate Testing

Child QOL differed by child sex ($F(1, 119) = 5.42$, $p = .02$). Male children had higher QOL ($M = 5.51$, $SD = 1.29$) than female children ($M = 4.97$, $SD = 1.22$). No other asthma outcomes differed by child sex. Asthma control differed by the caregiver's relationship to the child ($F(4, 115) = 2.94$, $p = .02$). Children whose primary caregiver was their biological father reported better asthma control ($M = 23.00$, $SD = 1.51$), followed by biological mothers ($M = 17.95$, $SD = 5.15$), and then grandmothers ($M = 16.67$, $SD = 3.14$); children whose primary caregiver was their step or adoptive mother had the poorest asthma control ($M = 15.67$, $SD = 4.16$). Whether the child was seen in the ED due to asthma in the last year differed by caregiver age ($F(1, 118) = 5.62$, $p = .02$). The average caregiver age was younger for children seen in the ED due to asthma ($M = 38.58$ years, $SD = 8.67$) compared to children who were not seen in the ED due to asthma in the last year ($M = 42.75$ years, $SD = 9.18$). Child age was not examined as a covariate due to it being the moderator variable.

Cumulative Risk Analyses for Combined Cohort

The original cumulative risk model (i.e., poverty, child perceived discrimination, neighborhood stress, caregiver perceived stress, family functioning) was first examined as a predictor of asthma outcomes in the combined cohort. Observed data results found the cumulative risk model to significantly predict QOL after controlling for child sex ($\Delta R^2 = .20$, ΔF (1, 109) = 29.21, $p < .001$). Pooled analyses had the same finding (Table 16). Higher cumulative risk scores were related to worse QOL scores. Additionally, the cumulative risk model was a significant predictor of asthma control after controlling for caregivers' relationship to the child based on both observed data ($\Delta R^2 = .10$, ΔF (1, 109) = 12.38, $p = .001$) and pooled analyses. Higher cumulative risk scores were associated with lower ACT scores (i.e., poorer asthma control). The cumulative risk model was also a significant predictor of quick-relief medication use based on both observed data ($\Delta R^2 = .07$, ΔF (1, 108) = 7.50, $p = .007$) and pooled analyses. Higher cumulative risk scores were associated with more quick-relief medication use. Lastly, logistic regression analysis revealed that the cumulative risk model did not predict whether or not the child was seen in the ED in the last year (b = .08, SE = .09, $\chi^2(1) = .78$, $p = .38$). Pooled analyses had the same non-significant finding of the cumulative risk model.

Table 16.

Original Cumulative Risk Model as a Predictor of the Combined Cohort Asthma Outcomes

Outcome	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	-.24	.04	-0.32, -0.15	-5.41	111	.00*
	Pooled	-.22	.04	-0.31, -0.14	-5.17	129	.00*
Asthma Control	Observed	-.66	.19	-1.03, -0.29	-3.52	111	.001*
	Pooled	-.67	.19	-1.04, -0.31	-3.63	120	.00*

Quick-Relief Medication Use	Observed	.09	.03	0.02, 0.15	2.74	111	.007*
	Pooled	.08	.03	0.02, 0.14	2.53	120	.01*
ED Visits	Observed	.08	.09				.38
	Pooled	.08	.09				.36

Note. *$p < .05$

Child Age Moderation Analyses in the Combined Cohort

Child age was not found to moderate the association between the cumulative risk model and any of the asthma outcomes. Child age was not correlated with any of the asthma outcomes. Child age was not tested as a moderator in the association between cumulative risk and whether or not the child was seen in the ED due to asthma because both the cumulative risk model and child age were not related to ED visits. Child age did not moderate the association between cumulative risk and child QOL, when controlling for child sex according to both the observed data ($\Delta R^2 = .002$, ΔF (1, 107) = 0.25, $p = .62$) and pooled analyses (Table 17). Child age was not a significant moderator of the association between cumulative risk and asthma control when controlling for caregiver relation to the child based on observed data ($\Delta R^2 = .01$, ΔF (1, 107) = 0.55, $p = .46$). Additionally, this moderation was not significant in pooled analyses (Table 17). There was no difference in significance when caregiver's relationship to the child was removed from the model. Lastly, child age did not moderate the association between cumulative risk and quick-relief medication use according to both the observed data ($\Delta R^2 = .00$, ΔF (1, 106) = 0.01, $p = .94$) and pooled analyses.

Table 17.

Child Age as a Moderator Between Cumulative Risk and Combined Cohort Asthma Outcomes

Outcome	**Model**	***b***	**SE**	**95% CI**	***t***	***df***	***p*-value***
QOL	Observed	.01	.02	-0.02, 0.04	0.50	111	.62
	Pooled	.001	.02	-0.03, 0.03	0.09	120	.93

Asthma Control	Observed	.05	.07	-0.08, 0.18	0.74	111	.46
	Pooled	.04	.07	-0.09, 0.17	0.66	120	.51
Quick-Relief Medication Use	Observed	.001	.01	-0.02, 0.02	0.08	111	.94
	Pooled	.01	.01	-0.02, 0.03	0.44	120	.66

Note. *$p < .05$

Discussion

Overall Summary of Main Findings

In the present study, the original cumulative risk model (i.e., poverty, neighborhood stress, family functioning, caregiver stress, perceived discrimination) developed for younger children (7-12 years) was a predictor of child QOL in the younger cohort, and QOL and asthma control in the adolescent cohort (13-17 years). However, this finding in the younger cohort (7-12 years) was not supported in pooled data analyses. Additionally, the original cumulative risk model predicted QOL, asthma control, and quick-relief medication use in the combined cohort analyses (children 7-17 years). The adolescent-specific cumulative risk model (i.e., poverty, neighborhood stress, school performance, caregiver-adolescent conflict, peer pressure) was a significant predictor of adolescent QOL and asthma control. Although asthma-related caregiver support was correlated with QOL and asthma control in both cohorts, it was only a significant moderator of the association between cumulative risk and asthma control among adolescents. Additionally, child age did not moderate associations between cumulative risk and asthma outcomes in the combined cohort of children. Findings are discussed in more detail in the following sections.

CARE Study Findings

In the first aim of the study, we examined a cumulative risk model (including poverty, neighborhood stress, family functioning, caregiver stress, perceived discrimination) based on a

toxic stress framework as a predictor of asthma outcomes (e.g., lung function, quick-relief medication use, ED visits, asthma control, asthma-related QOL) in a sample of 60 urban children with persistent asthma (7-12 years). Cumulative risk predicted child QOL and evening lung function assessments, but did not predict any of the other asthma outcomes; thus, findings only partially supported our hypothesis that the cumulative risk model would predict multiple child asthma outcomes. The cumulative risk model was a significant predictor of child QOL, such that higher cumulative risk scores were associated with lower QOL. However, this finding was only trending toward significance when pooled data were examined. This could be due to multiple imputation using approximation to estimate data points (Sterne et al., 2009). The underlying algorithms used to estimate missing data may not replicate the setting in which the observed data were collected. As there was a low percentage of missing data in the sample, it is possible that the association was true for families in this specific study.

Much of the general cumulative risk research has connected cumulative risk to physiological health outcomes such as HPA dysregulation or increased cardiovascular activity (Evans, 2003; Evans et al., 2007). The limited pediatric asthma research that has focused on cumulative risk has linked greater cumulative risk to more functional impairment in children with asthma (Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2012). In essence, the accumulation of stress has been associated with more frequent and intense episodes, more frequent symptoms between asthma attacks, and greater impairment due to symptoms for children with asthma. Previous research suggests that individual stressors, such as family functioning and caregiver stress/mental health are negatively associated with QOL in children with asthma (Chen & Schreier, 2008; Crespo et al., 2011; Kaugars et al., 2004). In the presence of high levels of stress, children (and families) may find it more challenging to manage their

asthma, which could affect adherence and lead to poorer asthma outcomes including worse QOL (Crespo et al., 2011). Moreover, stress is considered a trigger for asthma exacerbations in that stressful experiences may cause a child's airway to be sensitized (Chen & Miller, 2007). The current study findings extend this body of literature by suggesting that the accumulation of stress may be negatively associated with QOL in children with asthma. However, given the inconsistencies between observed and imputed data in the current study, the association between cumulative risk and child QOL needs to be explored further and replicated.

In the younger cohort, the cumulative risk model was not found to be a stronger predictor of child QOL than either neighborhood stress or child perceived discrimination. In meta-analytic studies, racial discrimination among children has been shown to have stronger negative effects on psychological well-being as compared to adults (Lee & Ahn, 2013; Schmitt et al., 2014). More specifically, DuBois and colleagues (2002) found that children's experiences with discrimination among Black youth were associated with adaptive difficulties (e.g., emotional, behavioral problems) in emerging adolescence. Additionally, perceived discrimination among Black youth has been related to increased odds of asthma prevalence and poorer asthma control (Thakur et al., 2017). For Black youth, personal experiences with discrimination may be related to more structural experiences of racism and discrimination including substandard housing, living in close proximity to higher levels of air pollution, or neighborhood stress. These factors have been associated with poor asthma outcomes among individuals from minority backgrounds (Thakur et al., 2017). For instance, Black youth who experienced perceived discrimination were more likely to experience more frequent symptoms and nighttime awakenings due to symptoms (Thakur et al., 2017). Stress related to personal experiences of discrimination may serve as a trigger for more asthma symptoms among youth.

Previous research has also documented an association between neighborhood stress and asthma morbidity (Chen & Schreier, 2008; Kopel et al., 2015; Wright et al., 2004). Neighborhood stress is often related to access to care and other resources, such that distance from healthcare facilities and access to public transportation could lead to inconsistent asthma care. Unlike the limited literature on perceived discrimination and pediatric asthma, there are several studies linking neighborhood factors to pediatric asthma morbidity. Negative neighborhood factors, including violence, higher rates of crime, housing deterioration, living in lower SES areas, and perceived safety, have all been associated with worse asthma outcomes including poorer asthma control, increases in symptoms and ED visits, and higher prevalence of asthma (Chen & Schreier, 2008; Koinis-Mitchell et al., 2014; Kopel et al., 2015; Suglia et al., 2010). When faced with higher levels of neighborhood stress and increased exposure to violence, child asthma management may decline due to caregivers' primary concerns of their children's safety (Koinis-Mitchell et al., 2014). Additionally, previous research has shown that living in an urban area may be linked to asthma regardless of race or household income and that higher rates of violence in the community have been associated with worse asthma outcomes (Aligne et al., 2000; Wright et al., 2004). Therefore, consistent with existing literature, findings from the current study suggest that neighborhood stress may negatively influence asthma morbidity, specifically asthma-related QOL, in urban, school-aged children with asthma. In fact, findings from the current study suggest that perceived discrimination and neighborhood stress may be each individually be stronger predictors of QOL than a cumulative risk model in children with asthma.

The cumulative risk model was not a significant predictor of asthma control, quick-relief medication use, or whether the child had visited the ED due to asthma in the last year.

Additionally, the cumulative risk model did not predict caregiver EMA reports of child asthma symptoms or asthma-related ED visits across the monitoring period. The cumulative risk model also did not predict child morning lung function assessments from the spirometers (FEV_1/FVC). These findings are inconsistent with previous research that has linked cumulative risk to poorer asthma outcomes among urban children (Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2010; Koinis-Mitchell et al., 2012). Inconsistencies between previous research and the current study may be partially attributed to differences in samples. The current study included predominately Black children and 75% of families lived below the poverty line; previous research has included families from Black, Latino, and White backgrounds and 50% or less of families lived below the poverty line (Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2010). Perhaps findings related to cumulative risk and certain asthma outcomes do not hold true in more homogenous samples.

Additionally, stressors included in the current cumulative risk model may not have truly reflected sources of risk experienced by families living in the urban Richmond area. There may be a need to include stress from triggers such as secondhand smoke exposure, which have been included in other models (Koinis-Mitchell et al., 2007). The current model did not account for stress due to poor air quality or the presence of triggers, such as pet dander or pollen. Inclusion of additional sources of stress may present a more well-rounded portrayal of daily life for urban children with asthma. As suggested in previous studies, an alternative explanation for the non-significant findings could be that urban families faced with many potential sources of stress have already been identified by healthcare providers (Koinis-Mitchell et al., 2007). These families may already be receiving more consistent healthcare or other forms of support, such as

counseling, asthma education programs, or social work services. Thus, perhaps their child's asthma is well-managed despite an accumulation of risk.

Interestingly, however, the cumulative risk model did predict child evening lung function assessments from the spirometers. There is limited research on morning versus evening lung function in pediatric asthma; however, this finding could be due to circadian variation in pulmonary function. In people with asthma, pulmonary function tends to fall in the morning (Medarov, Pavlov, & Rossoff, 2008), and there may be more variability in evening assessments of lung function. Pediatric studies of pulmonary function have also found FEV_1 values to be lowest in the morning (Delfino et al., 2008). Most research involving morning and evening spirometry assessments has focused on particulate matter concentrations and the negative effects of greater concentrations on lung function at both time points (Yamazaki et al., 2011). However, it is possible that different air quality exposures throughout the day (e.g., air pollution during rush hour, home air quality, school air quality, environmental tobacco smoke exposure) can help explain differences in lung function. Additionally, physical activity and quick-relief medication use throughout the day have also been found to impact evening lung function assessments (Delfino et al., 2008; Yamazaki et al., 2011). In the current study, all of these factors may help to explain the association between cumulative risk and evening lung function, but not morning lung function assessments.

We also found that asthma-related caregiver support was associated with higher QOL and better asthma control. This finding is consistent with previous literature connecting less support from parents to more asthma symptoms and worse daily lung function among children with asthma (Chen et al., 2007). Caregivers/parents play an important role in child asthma management and less support from caregivers may mean more inconsistencies in care, including

worse medication management, irregular medication refills, and fewer doctor appointments. However, asthma-related caregiver support was not a significant moderator in any analyses between cumulative risk and child asthma outcomes (e.g., QOL, asthma control). These findings are inconsistent with previous research suggesting a buffering effect of a supportive caregiver between early life events or stress and health outcomes (Johnson et al., 2013). It is important to note that the cumulative risk model was not associated with asthma control and this may partially explain the null moderation results. Therefore, non-significant moderation findings may be better explained by the lack of association between the cumulative risk model and child asthma outcomes rather than the association between asthma-related caregiver support and child asthma outcomes.

Project AAIR Findings

In the second aim of the study, two cumulative risk models were tested in the adolescent cohort (13-17 year olds). Most of the literature on cumulative risk and pediatric asthma to date has focused on younger children. Although teens as old as 13 years, or even 15 years, have been included in previous research studies, the age range of child samples has typically started at 6 or 7 years of age (Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2012). Even developmental cumulative risk research has focused on elementary and middle school age children; therefore, older adolescents have largely been excluded from such studies (Evans, 2003; Evans et al., 2007). The present study extended the cumulative risk and pediatric asthma research by focusing specifically on a cohort of adolescents who ranged in age from 13 to 17 years old.

First, the original cumulative risk model (i.e., poverty, neighborhood stress, family functioning, caregiver stress, perceived discrimination) examined in the younger cohort was also tested in the adolescent cohort. This cumulative risk model was a significant predictor of

adolescent QOL, asthma control, and was trending toward significance in predicting quick-relief medication use. Poorer asthma control and worse child QOL have been documented in the presence of greater neighborhood stress and worse family functioning among children with asthma (Chen & Schreier, 2008; Kaugars et al., 2004; Koinis-Mitchell et al., 2014; Kopel et al., 2015). Additionally, perceived discrimination among Black youth has been associated with poorly controlled asthma (Thakur et al., 2017). The findings of the current study extend the current body of literature by suggesting that the accumulation of risk is associated with poorer asthma outcomes in adolescents. Notably, findings suggest that a model of risk factors typically experienced by younger samples may be relevant in predicting asthma outcomes in adolescence.

Next, the adolescent-specific cumulative risk model of stress (i.e., poverty, neighborhood stress, stress related to school, caregiver-adolescent conflict [relationship with parent], peer pressure) was examined as a predictor of adolescent asthma outcomes. Similar to the original risk model, the adolescent-specific model was a significant predictor of adolescent QOL and asthma control. Thus, cumulative risk may be a salient predictor of QOL and asthma control regardless of child age. Moreover, findings are consistent with previous research linking individual sources of stress including poverty, harsh parent-child interactions, and greater levels of neighborhood stress with poorer health outcomes for children with asthma (Koinis-Mitchell et al., 2014; Kopel et al., 2015; Tobin et al., 2015a). Additionally, previous research has found an association between stressful life events among inner-city adolescents, including events related to school and family relationships, and asthma prevalence and morbidity (Turyk et al., 2008). Stress may be associated with worse asthma morbidity through several pathways: 1) stress is associated with the dysregulation of the immune system and HPA axis, which may impact inflammatory responses related to asthma; 2) stress can negatively impact health behaviors

which can lead to poorer asthma management; and 3) stress can directly affect the mental health of adolescents and caregivers, which can hinder asthma care (Kaugars et al., 2004; Shonkoff et al., 2012; Turyk et al., 2008; Wood et al., 2015). Current findings add to the growing body of literature linking stress and asthma morbidity in urban adolescents with asthma.

In concordance with our hypotheses, the adolescent-specific model was a better predictor of adolescent QOL and asthma control when considering effect sizes. However, the original cumulative risk model was a more robust predictor of quick-relief medication use. Neither model predicted ED visits. Adolescents with asthma may be at higher risk for asthma morbidity due to the greater variety of stressors they experience (e.g., school, caregivers, peer pressure, neighborhood, work, family) (Bitsko et al., 2013). As children emerge into adolescence their priorities and values may shift, with peers and school becoming a more central focus of their self-identity (Bitsko et al., 2013); stress related to these areas may impact their overall health more so than for younger children. The current study findings partially support this notion, in that adolescent-specific cumulative risk was a more robust predictor of adolescent QOL and asthma control than the non-adolescent-specific risk model.

Given the aforementioned findings, the adolescent-specific cumulative risk model was examined in comparison to each individual stressor (i.e., poverty, neighborhood stress, school performance, peer pressure, caregiver-adolescent conflict) for QOL and asthma control. The adolescent-specific cumulative risk model was a more robust predictor of both QOL and asthma control than every individual stressor. This finding is consistent with previous research that has found cumulative risk to be a stronger predictor of asthma outcomes than any single individual factor (Koinis-Mitchell et al. 2007). Koinis-Mitchell and colleagues (2007) found cumulative risk to be a stronger predictor of functional impairment than poverty or severity of asthma alone.

Therefore, the combination of stressors may be a stronger predictor of adolescent QOL and asthma control than any one stressor on its own. The QOL findings oppose what was found in the younger cohort, where several individual factors were stronger predictors of QOL than the cumulative risk model. Cognitive functioning developments during adolescence may allow teens to better understand the implications of stress on their health and potential additive effects of stress (Bitsko et al., 2013). For example, in focus group research, teens have discussed the interplay of stressors, specifically being tired from school and then going home to complete tasks from their parents. In doing so, the teens had to put off school work, which added to their stress (LaRue & Herrman, 2008). Therefore, the combination of stressors may be more important in adolescent QOL and asthma control because teens are better able to process and understand the ramifications of multiple sources of stress on their health.

Asthma-related caregiver support was also examined as a moderator between cumulative risk and asthma outcomes in the adolescent cohort. Inconsistent with our hypotheses, adolescent reported asthma-related caregiver support did not moderate the association between adolescent-specific cumulative risk and adolescent QOL, quick-relief medication use or whether the adolescent was seen in the ED due to asthma in the last year. As discussed in the younger cohort, these findings are inconsistent with the toxic stress theoretical framework suggesting the buffering effect of a supportive caregiver between early life events or stress and health outcomes (Johnson et al., 2013). Previous research focused more broadly on pediatric chronic conditions found that supportive family relationships have been related to better psychological adjustment in children (Drotar, 1997). However, previous research did not specifically address urban families with an adolescent with asthma. Chen and colleagues (2007) also found an association between less parental support and more asthma symptoms and worse daily lung function among

youth with asthma. The youth sample in their study consisted of primarily White and Asian children and adolescents with asthma, whereas the current study included urban, predominately Black teenagers. Demographic differences between previous research and the current study may also give insight into these discrepancies. Family interactions (e.g., caregiver-adolescent interactions) may differ based on cultural backgrounds, which could be one explanation for the inconsistencies between previous research and current findings.

Contrary to findings with other asthma outcomes, asthma-related caregiver support moderated the association between adolescent-specific cumulative risk and asthma control in pooled data analyses; these findings were inconsistent in observed data analyses. One explanation is that there may not have been enough power to detect the moderation in the observed data; the pooled analyses incorporated 9 more participants. Post-hoc probing analyses revealed the association was significant for both groups (i.e., low support, high support). However, the adolescents who received lower levels of asthma-related caregiver support had a more dramatic decrease in asthma control as their cumulative risk levels rose. This significant finding supports the buffering hypothesis of asthma-related caregiver support and is consistent with findings related to worse asthma outcomes with low parental support (Chen et al., 2007). For instance, Chen and colleagues (2007) found that less parental support was associated with more asthma symptoms and worse lung function. For adolescents perceiving low asthma-related support from their caregivers, the accumulation of stress may have a greater negative impact on asthma control than for adolescents who feel that they are receiving high levels of support from their caregivers. However, because only the pooled analysis was significant, this moderation should be interpreted with caution.

Combined Cohort Findings

For the third aim, the two cohorts were combined and the original cumulative risk model (i.e., poverty, neighborhood stress, family functioning, caregiver stress, perceived discrimination) was examined as a predictor of child asthma outcomes (e.g., QOL, asthma control, quick-relief medication use, ED visits). In the combined group, higher cumulative risk scores were associated with worse QOL, poorer asthma control, and more quick-relief medication use. However, the cumulative risk model was not a predictor of whether the child was seen in the ED due to asthma in the last year. These findings differed from those of the younger cohort (7-12 years) in that there were multiple significant findings in the combined cohort analyses. One rationale for this difference is that the combined cohort analyses were higher powered due to a larger sample size, and thus were able to detect significant findings. Additionally, the wider age range of participants could be driving the findings of the combined analyses.

As previously discussed, these significant findings support previous cumulative risk research linking higher levels of stress with worse asthma morbidity (Koinis-Mitchell et al., 2007). However, consistently through all three groups of analyses, cumulative risk has been unrelated to whether the child or adolescent was seen in the ED due to asthma in the last year. One possible explanation may be the high rate of ED use among families in this sample (i.e., limited variability in ED use). Approximately 70% of the combined cohorts reported that the child/adolescent was seen in the ED due to asthma in the last year. According to the CDC (2006), ED use for pediatric asthma care remains high and Black children are more frequently treated in the ED than White children (CDC, 2012). Current research confirms that the racial disparity in ED use for asthma care is still present and may not be attributable solely to SES (Franklin, Grunwell, Bruce, Smith, & Fitzpatrick, 2017). Although system-level factors

including access to care (e.g., transportation, insurance) may be one factor driving the higher rate of ED use among Black children with asthma, research is still needed on the underlying causes of this disparity.

Child age was examined as a moderating variable of the association between cumulative risk and the child asthma outcomes. Contrary to our hypotheses, child age was unrelated to child asthma outcomes and did not moderate associations between cumulative risk and QOL, asthma control, quick-relief medication use, or ED visits. These findings are somewhat inconsistent with previous literature suggesting differences in QOL among children based on age (Moreira et al., 2013). Moreira and colleagues (2013) found that younger children with asthma reported better QOL than adolescents with asthma. Moreover, adolescents are often given control of their asthma care prematurely by parents suggesting suboptimal asthma management among adolescents (Bruzzese et al., 2012). Adolescent asthma management also tends to be more reactive than proactive or preventative (Bruzzese et al., 2012), which can lead to more symptom exacerbations (i.e., poorer asthma control). However, other research suggests that younger children may have poorer asthma control and increased frequency of symptoms (Kuehni & Frey, 2002). Kuehni and Frey (2002) suggested that poorer asthma control among younger children may be due to greater asthma severity possibly related to increased frequency of viral infections or hesitation by doctors and parents to increase medication use. Although findings are mixed, previous research suggests that there may be differences in asthma outcomes (e.g., QOL, asthma control) based on child/adolescent age. We did not find this to be true of child age in the current study. Our findings may instead suggest that regardless of child age, increased cumulative risk has a detrimental impact on asthma outcomes in both children and adolescents.

Limitations

There are several limitations of the present study. First, the sample sizes of the two individual cohorts were small and limited in statistical power. Significant effects may not have been detected due to the small sample sizes; therefore, multiple imputation was conducted to investigate if there were any differences between observed data and an imputed full data set. The small samples of the two groups also limited the generalizability of the findings. All participants were recruited from an urban area and findings may not generalize to children and adolescents living in other areas (e.g. rural areas). Children and adolescents living in rural areas may also face different stressors than children from urban areas (Kopel, Phipatanakul, & Gaffin, 2014; Parsons, Beach, & Senthilselvan, 2017). Different sources of stress may be more appropriate to include in other cumulative risk models, such as stress related to air pollution, household condition, or environmental tobacco smoke exposure. Additionally, children and adolescents had to meet persistent asthma criteria and could not have any other pulmonary disease, severe psychiatric condition, or development delay. Findings are limited to children who meet these criteria.

The central focus of the CARE Study was to examine caregiver QOL; therefore, measurement selection was based around this outcome. For instance, family functioning was assessed by caregivers and not children. Additionally, the CARE study focused on asthma-related caregiver support, instead of a more global assessment of caregiver support. Although support related to medical care for asthma may be a proxy for more general support, global assessments of caregiver support should be included in future work. Global assessments would include support in more areas of the child's life than just medical care and may provide information on how supported the child feels across other aspects of their daily life. Caregiver support has been associated with immune function, such that inadequate support was related to

dysregulated immune responses (Johnson et al., 2013), which can have long-term impacts on health. In the CARE cohort, caregivers responded to support items through EMA surveys and a child assessment of caregiver support was not collected. Future work should include global assessments of caregiver support in which both the child and caregiver provide reports (similar to the multiple informant reports in the adolescent cohort).

Additionally, a majority of the data collected in both studies was self-reported. Lung function was the single objective assessment. Self-report data, specifically from the initial research session, are subject to social desirability and time recall biases. More objective assessments should be included in future research. For example, counters on inhalers can be used to track medication use or objective assessments of family functioning can be recorded and coded. Caregivers and children/adolescents completed questionnaire packets simultaneously and separately, however the two groups were only separated as much as the physical environment would allow (e.g., opposite sides of the room). Dyads were sometimes in close proximity when the research sessions took place in family homes. Therefore, some reports may be an underestimation, such as caregiver-adolescent conflict behavior.

Lastly, the current study used EMA methodology only with the younger cohort to capture the daily experiences of children with asthma and their caregivers. Future research should incorporate EMA methodology to specifically assess the daily stressors children and adolescents with asthma may face. Children and adolescents could complete assessments themselves in addition to caregiver assessments to allow for multi-informant data collection.

Future Directions and Clinical Implications

Findings suggest several directions for future research. First, stressors among younger children need to be explored further to delineate which sources of stress are most central to

asthma outcomes among urban children. Previous research suggests that the accumulation of stress is associated with the development of asthma and greater asthma morbidity (Chen & Schreier, 2008; Suglia et al., 2010). However, the current study found that for the younger group of children (7-12 years), the cumulative risk model of stress was only associated with child QOL, and even that finding was disputed with pooled analyses. Further exploration of cumulative risk is needed with younger children with asthma. The current study may have been hindered by the choice of specific stressors and assessment tools. Focus group research could help determine the prominent sources of stress that school-aged children with asthma experience. Cumulative risk models could then be developed from these focus group findings.

Secondly, the findings in the adolescent cohort need to be replicated. The current study included participants from an urban area and findings should be replicated with larger and more diverse groups of adolescents from similar settings. For example, the sample was predominately Black. Future research should include adolescents from other backgrounds to determine if findings extend to adolescents of other races/ethnicities. Future research could include additional stressors in cumulative risk models, such as ones related to romantic partners or financial pressures, to try to gather a more comprehensive assessment of stress for adolescents. From there, intervention work could target the accumulation of stress for adolescents with asthma. Specifically, interventions could include strategies for handling overlapping sources of stress and ways to minimize the cumulative effects.

Lastly, the buffering role of caregiver support needs further exploration. Although research suggests that support from a caregiver may mitigate the effects of negative early life experiences on health outcomes (Johnson et al., 2013), asthma-related caregiver support was only found to be a buffer in one analysis. As discussed in the limitations, a more global measure

of caregiver support is needed in future work examining the buffering effect of caregiver support in the association between cumulative risk and child asthma outcomes. Additionally, a more specific assessment of asthma-related caregiver support could be developed. Questions used in the present study were adapted from a self-efficacy scale related to asthma management (Bursch et al., 1999). Future work could include more pointed questions related to caregivers' involvement in their child's daily asthma management routine. For example, assessments of reminding the child to take their medication, observing the child taking their medication, asking the child daily about their symptoms, scheduling regular check-up appointments, and how often they refill their children's medications could be included. Previous research has found that caregiver involvement in adolescent asthma management improves medication adherence (Duncan et al., 2013), which highlights the importance of assessing caregiver support in asthma management among both children and adolescents.

The present study also has implications for healthcare providers and clinicians. Previous research (Chen & Schreier, 2008; Crespo et al., 2011; Koinis-Mitchell et al., 2007; Koinis-Mitchell et al., 2012; Tobin et al., 2015a) along with the current findings call for a focus on stress that children and adolescents with asthma may experience and how the accumulation of stress can complicate disease outcomes. First, education on the effect that stress may have on a child's asthma may be important for families that face a variety of stressors in their daily lives. Pediatric healthcare providers could include this topic in their discussions of asthma care with families. Second, stress assessments for children and adolescents with asthma in medical settings might be warranted. Stress assessments could include multi-informant reports from child/adolescent and primary caregiver dyads, as well as cortisol testing (objective assessment). These assessments could offer healthcare providers a more comprehensive picture of environmental influences

affecting the child's asthma. Therefore, if needed, clinicians could have more detailed discussions about the role of stress in asthma care and strategies that may decrease the effects of stress and improve asthma outcomes. Also, healthcare providers could provide families with referrals or contact information for resources (e.g., counselors, social workers, community centers) in more severe cases, that otherwise may not have been provided. Previous focus group research has found that caregivers of inner-city children with asthma want and need educational programs on asthma medications and accessible community resources that could help ameliorate stress associated with caring for a child with asthma (Bellin et al., 2017). Lastly, stress management programs could be built in as an option in asthma care, especially for adolescents with asthma. Previous research has found that community-based stress management interventions for children with asthma have been effective at reducing stress and improving physical health, including pulmonary function (Long et al., 2011). Stress management programs could include education, relaxation training, and coping techniques.

Overall, findings suggest that the accumulation of stress can have negative impacts on asthma outcomes (e.g., poorer QOL, worse asthma control, more quick-relief medication use), especially for urban adolescents with asthma. Further research is needed to determine the most central sources of stress urban school-aged children with asthma experience and to replicate findings for adolescents with asthma. Additionally, asthma-related caregiver support needs to be examined further as a potential buffer in the association between cumulative stress and asthma outcomes in children and adolescents.

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Vita

Samantha Ann Miadich was born on September 25, 1986 in Garfield Heights, Ohio. She received a Bachelor of Arts in Psychology from the University of Dayton, Dayton, Ohio in 2008. She received a Master of Arts in Clinical Psychology from Ball State University, Muncie, Indiana in 2010. She worked at Case Western Reserve University as a Research Assistant II for several years before entering her doctoral program at Virginia Commonwealth University in 2013.