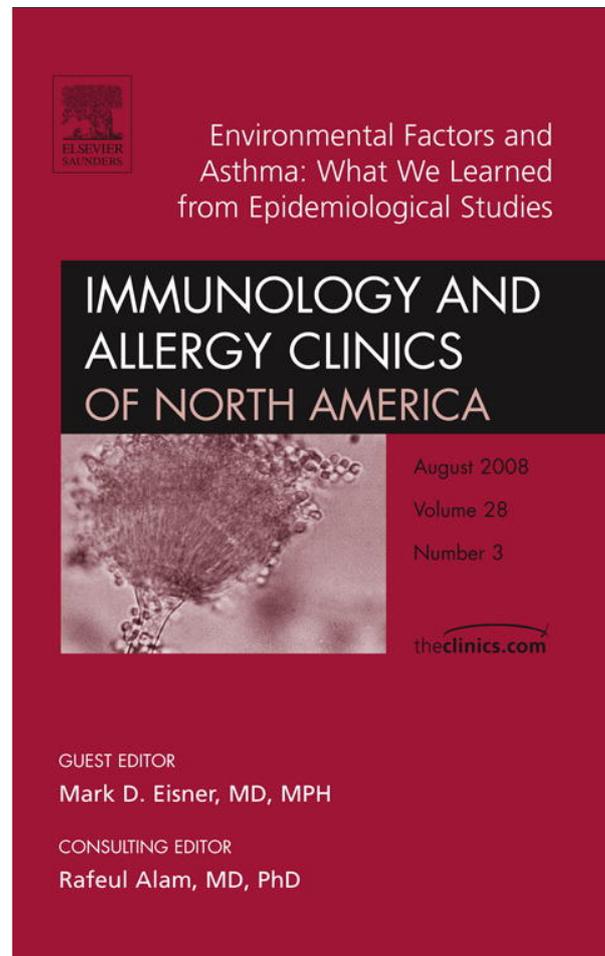


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Does the Social Environment Contribute to Asthma?

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The impact of the social environment on asthma has recently begun to receive increasing attention. This article reviews the current literature to investigate the impact of the social environment at three levels—the neighborhood level, the peer level, and the family level—and to explore pathways through which the social environment “gets under the skin” to impact asthma onset and morbidity. Research to date suggests that adverse social conditions at the neighborhood and family levels impact asthma morbidity through direct effects on physiologic systems as well as by altering health behaviors. The impact on asthma of social networks, such as friendships, is less clear and will need to be investigated further. Future research will need to take into account the impact of the social environment to develop more comprehensive models of asthma pathogenesis.

Does the social environment contribute to asthma?

Asthma is widely recognized as a complex disease with multiple contributing factors. These include environmental exposures, viral infections, and genetics, the roles of which have been well established [1–4]. However, in contrast to the above factors, the role of social factors in asthma has been less well-established. Clinical anecdotes have long existed describing patients whose asthma appeared to be exacerbated by social circumstances, such as a significant stressful life event. However, empirical evidence supporting this claim has only appeared more recently. This article provides an overview of studies that have addressed the role of the social environment in asthma,

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and discusses the biological mechanisms by which social factors might influence the course of asthma.

What is the social environment?

The social environment includes the connections a person has to a larger social community. This could include interpersonal or close relationships with others, the daily social contacts an individual has with others, as well as an individual's social position within a larger community. We argue that in considering the role of the social environment in asthma, it is important to acknowledge social factors at multiple levels. For example, the most proximal level of social environment is a person's family. For many, family members are those with whom interactions take place daily and those with whom individuals have had their longest relationships. Friendships or peer relationships represent the next level of social connection. These are often close others who provide an important source of social support and companionship. At the broadest level are neighborhood connections. Neighbors provide a source of social contact, but may not be as close as family and friends. In addition, neighborhoods often provide a sense of social identity and community for many individuals. In this article, we discuss the role of the social environment at each of these levels—neighborhood, peer, and family levels—in affecting asthma.

Neighborhood effects on asthma

We highlight two social factors at the neighborhood level that have been linked to asthma: socioeconomic status (SES) and exposure to violence.

Socioeconomic status

Although SES likely reflects a number of nonsocial factors [5,6], including material resources, access to health care, and housing quality, it also contains a social component in that it indicates a person's relative standing within a larger social community. This in turn affects how people perceive themselves within their social group [7], as well as how others interact toward them [8].

Across numerous studies, evidence shows that lower SES increases risk for morbidity among patients with asthma. For example, poor children and children from lower income families are significantly more likely to be hospitalized for asthma, to have greater asthma symptoms, and to have more severe asthma episodes compared with nonpoor and higher income children with asthma [9–11]. Fewer years of parent education also have been associated with greater risk of asthma hospitalizations and emergency department visits in children with asthma [12,13]. Longitudinal studies show similar patterns. For example, children whose fathers had less

prestigious occupations were more likely to be hospitalized because of asthma during the following 6 years compared with those whose fathers had more prestigious occupations [14]. In addition, neighborhoods with lower income levels and higher unemployment rates have been found to have higher rates of asthma hospitalizations [15–17]. Finally, population-based studies (which are distinct from the above studies that focus on children with pre-existing asthma) have also found that lower SES (as indicated by either parental education or neighborhood characteristics) is associated with greater risk of asthma-related emergency room visits, asthma hospitalizations, and more severe asthma in children [18,19].

In contrast, relationships between low SES and asthma onset are less clear. Some studies have shown that low SES is associated with an increased prevalence of asthma. For example, children whose parents have fewer years of education are more likely to have a physician diagnosis of asthma [19]. Children from poorer families are also more likely to have a diagnosis of asthma [9,11]. Children who live in inner-city or low-income neighborhoods are more likely to have current asthma [16,18,20,21]. However, other studies have found no evidence for an association between neighborhood SES and childhood asthma prevalence rates [22,23]. Meanwhile, some studies have found individuals in higher SES groups to be more likely to have an asthma diagnosis [24,25]. In sum, evidence that living in a low-SES environment increases the risk for morbidity among those already diagnosed with asthma is fairly strong, but evidence that low-SES environments contribute to asthma onset is less clear-cut.

Neighborhood characteristics

Intriguing evidence has emerged recently to suggest that certain negative social characteristics of neighborhoods may be detrimental to asthma. For example, exposure to violence represents a community-level stressor that can take a toll on health [26]. Previous research has documented that greater exposure to violence is associated with a greater number of symptom days in children with asthma, as well as a greater number of nights of lost sleep for caretakers because of their child's asthma [27]. Similarly, greater problems in one's neighborhood, such as problems with crime and gangs, have been associated with greater asthma symptoms in children [28]. In addition, exposure to violence moderated the relationship between physical environment exposures and asthma. Children were at greater risk for being diagnosed with asthma if they had high levels of exposure to both traffic-related air pollution and violence [29].

Explaining neighborhood effects

Why would the neighborhoods that people live in affect asthma? One possibility is that life in a low-SES or high-violence neighborhood is more stressful on a day-to-day basis. Life in a low-SES household may involve

multiple competing demands that are often unpredictable. For example, low-SES families may experience multiple pulls on their limited resources, such as trying to pay bills so that services to their home do not get shut off while trying to put enough food on the table and while trying to deal with problems in the house, such as a plumbing leak. Demands may also spill over from one domain into another, creating conflicts between domains for families. For example, parents may need to work additional shifts to make extra money for the family, but this takes away from time with children and creates pressures to find additional childcare options. Demands in low-SES households may also create unpredictability in families' day-to-day lives. For example, low-SES children may not be able to count on a consistent schedule if parents must depend on unreliable public transportation for getting between work and home. Similarly, families that live in high-violence neighborhoods may also experience greater stress in their day-to-day lives. This may be because they have to constantly be vigilant for threats to their safety. This vigilance may be activated every time they leave their home, and may even extend to feeling unsafe in their home environment. As a result, families may have to change their pattern of activities. For example, if they do not feel safe, they might not leave the house to shop or for social activities. Hence life in low-SES or unsafe neighborhoods may be more stressful on a day-to-day basis [30]. In addition, families who live in low-SES or unsafe neighborhoods may have fewer material and social resources for coping with stressors when they arise, adding to the overall burden of stress for these individuals. In the section below on biological mechanisms, we discuss the notion that the experience of stress may directly influence biological systems in a way that has implications for asthma.

A second possibility is that neighborhood factors have effects on asthma by changing behaviors. For example, low-SES neighborhoods may have different social norms about the acceptability of certain health behaviors, such as smoking. If smoking is more prevalent and accepted in low-SES neighborhoods, individuals with asthma are more likely to both be exposed to secondhand smoke, as well as to be smokers themselves. Hence low-SES neighborhoods might increase the risk for health behaviors that are detrimental to asthma. In addition, neighborhoods with high levels of violence may create barriers to asthma management behaviors. For example, unsafe neighborhoods may make it difficult to fill asthma prescription medications in a timely fashion [31]. Finally, living in a neighborhood with frequent violence could affect asthma if individuals with asthma are more likely to spend time inside their homes, resulting in greater exposure to indoor allergens as well as more sedentary behaviors [31].

Social networks and asthma

A number of studies have asked whether the types of social networks or social support an individual has can impact asthma morbidity. Social

support can come from a number of sources, including friends, colleagues, teachers, physicians, and other asthma patients. Moreover, social support can be conceptualized both in terms of the perceived quality, or closeness, of social relationships, as well as the quantity—that is, the diversity or extensiveness—of one's social networks. Patients with asthma report that social networks are quite valuable and helpful when it comes to managing their asthma [32,33].

Although patients perceive their social networks to be beneficial, the studies that have examined whether they are related to asthma outcomes have mixed conclusions. On the positive end, a number of studies have found social support to be associated with asthma outcomes. For example, poor confidant support and negative support were related to a greater likelihood of hospital admissions in adult patients with asthma [34]. Conversely, having better social contacts predicted fewer asthma symptoms and, to a lesser degree, better peak expiratory flow rate over the course of 3 weeks [35]. Social support may also be most effective as a buffer against stress. For example, individuals who experienced both more negative life events and low social support reported the most episodes of cold-induced asthma exacerbations [36]. Finally, Levy and colleagues [37] reported that after an unrelated intervention in youth with asthma in one of three housing developments studied, those whose social networks improved were more likely to experience a decrease in asthma symptoms relative to baseline measures.

Similarly, among children with asthma, the social networks of parents are related to clinical outcomes in their children. For example, parents who reported low social support were more likely to have children who were admitted to the emergency department [38] and who were at greater risk for continued atopic illness [39]. Conversely, families reporting good social support had children at reduced risk of both wheezing and asthma [40] and more likely to recover from atopic illness between the ages of 18 months and 3 years [39]. Weil and colleagues [41] also reported some evidence of an association between good social support in parents and marginally fewer days of wheeze in their children. That social support to the family can work in very practical ways was demonstrated in a Norwegian study that found that access to a specialized asthma health care facility was in part influenced by whether families of children with asthma had a physician in their social network [42].

Finally, in addition to morbidity outcomes, social support also may affect behaviors relevant to asthma. Two studies of youth with asthma found that receiving social support was related to better asthma self-management behaviors, such as greater medication adherence and more willingness to seek immediate help from other people in response to breathing problems [32,33]. Adults with asthma also reported their social network to be the main factor facilitating exercise and an active and healthy lifestyle [43].

In contrast to the above studies, a handful of studies have found no relationship between social support and asthma. Ngamvitroj and Kang [44] reported that social support was not a significant predictor of greater peak expiratory flow rate self-monitoring over the subsequent 3 months even though participants' satisfaction with their social support was very good. A study of youth with asthma found that, whereas family support was associated with better pulmonary functioning and fewer asthma symptoms, peer support was not related to either of those factors [28]. Finally, one study investigating the risk factors for asthma mortality matched adults who died of asthma with a comparison group of patients with asthma who were hospitalized but did not die. None of the factors relating to people's social networks, including bereavement, separation, or isolation, was found to be a significant risk factor for asthma mortality [45].

Finally, it is important to also consider the possibility that social networks may, in some circumstances, exert negative influences. For example, in certain situations, social networks may create peer pressure to engage in detrimental health behaviors. Precht and colleagues [46] found that high school students with asthma, in particular boys, were more likely than healthy students to smoke because of peer pressure, although the overall occurrence was rare. Chen and colleagues [28] also found that stronger peer support in youth with asthma was related to lower medication adherence. This too may reflect the desire of youth to fit in with their peers and not stand out because of their asthma, potentially resulting in their neglecting to take medications appropriately.

Explaining social network effects

Social support and social networks may affect asthma through a variety of pathways. For example, social networks are important sources of tangible support (ie, concrete assistance with tasks). This may take the form of providing rides to the doctor, watching siblings so that a parent can take a child with asthma to the doctor, or offering to pick up important medications from the pharmacy. The larger one's social network, the more options one has to draw on for this type of help. These types of assistance with asthma management behaviors may help explain one way in which social networks can be beneficial for coping with asthma. Living with a chronic illness such as asthma can require a number of changes to daily routines, and it can be difficult for many families to balance asthma management with the other daily responsibilities in life. Hence, social networks may provide additional sources of support for managing with all these tasks.

Second, social networks may also provide tangible support in the form of knowledge and advice. For example, patients with asthma may learn new information from others with asthma, such as information about new medications or helpful sources of care for asthma. Patients with asthma may also learn from others different strategies for better managing their asthma (eg,

for remembering to take their medications) and may find others to be helpful role models for both daily management and responses to symptom exacerbations.

Another form of support offered by social networks is emotional support. In contrast to the concrete assistance of tangible support, emotional support refers to providing empathy and understanding for another person's emotional reactions and feelings. This type of support could affect asthma outcomes in a number of ways. First, emotional support may help patients with asthma be more accepting of their illness. This may lead to more positive mental health states, with implications down the line for physical health. Second, support from others with respect to one's emotional responses may help patients with asthma remain calm during times of symptom exacerbations. This in turn may facilitate appropriate implementation of asthma action plans and optimal responses to asthma symptoms. Third, support from others may increase positive emotional states and decrease negative emotional states more generally in patients with asthma. In turn, these emotional experiences could alter physiologic states in ways that have implications for asthma.

As we described above, social networks can sometimes have negative influences on asthma, particularly among youth. It is possible that youth with asthma are more susceptible to negative peer pressure because their asthma already makes them socially vulnerable. For example, if youth have activity limitations due to asthma that make them feel different and less accepted, they may be more likely to engage in maladaptive behaviors, such as smoking, to fit in or gain approval from peers. Thus, even if they are aware of the negative impact of such behaviors, knowledge alone may not be sufficient to drive behavior, particularly if there are competing pulls from peers that conflict with this knowledge.

Family effects on asthma

Evidence suggests that both the quality of family relationships and the psychosocial traits that family members possess affect asthma outcomes. Much of this research has focused on children with asthma and has investigated how family factors affect them. We will review evidence for links between family relationship quality and asthma, as well as links between parent traits, such as parental depression and stress, and childhood asthma.

Family relationships

The quality of relationships with family members affects not only a child's psychologic well-being but also his or her physical well-being. For example, family relationships early in life predict the likelihood of children getting diagnosed with asthma years later. In a sample of children at risk for developing asthma, researchers measured parenting difficulties when infants were

3 weeks old. During home visits, clinicians assessed parenting difficulties by observing attitudes and behaviors parents exhibited toward their child, parents' ability to regulate their child's arousal and emotions, the supportiveness of parents' relationship with each other, and the balance in the home between childcare and other demands. Children were then followed for several years to track the development of asthma. Greater parenting difficulties when an infant was 3 weeks old prospectively predicted a greater likelihood of that child being diagnosed with asthma, both by the age of 3 and by the age of 8 [47,48].

In addition, among children already diagnosed with asthma, family relationship quality contributes to morbidity and mortality. One classic example of this is a case-control study that recruited two groups of children hospitalized for asthma. One group consisted of children who died of asthma following hospital discharge. The second group was made up of children who did not die, despite having similar severity of illness. A number of psychologic and social factors distinguished these two groups, including parent-patient conflict and family dysfunction. Children in the group who died from asthma were more likely to have persistent and severe arguments with parents, as well as to have families with greater dysfunction (eg, intense marital conflict) [49]. Similar types of relationships were found with respect to asthma morbidity outcomes. Among a sample of children with asthma, those who reported receiving less support from their parents had more asthma symptoms and poorer daily pulmonary function [28].

Parent characteristics

The psychosocial characteristics of a parent can also affect asthma outcomes in a child. For example, higher levels of maternal depression have consistently been linked to childhood asthma. Shalowitz and colleagues [50] documented that parents who reported greater symptoms of depression had children who were more likely to use health care services and who experienced more severe asthma symptoms. Similarly, among a sample of children hospitalized for asthma, depression in parents was associated with a greater number of unscheduled physician visits as well as a greater number of hospitalizations for asthma in their children [51]. Longitudinal studies show similar patterns. Weil and colleagues [41] found that children of parents having clinically significant levels of mental health problems (including depression) at baseline had twice the rate of hospitalization for asthma during a 9-month follow-up period compared with children of parents without mental health problems. Inner-city mothers with high levels of depressive symptoms at baseline were 30% more likely than nondepressed mothers to report taking their children to the emergency department for asthma during the following 6 months [52]. These longitudinal data suggest that depression precedes poor asthma outcomes, rather than the opposite explanation—that having a child with poorer asthma causes parents to become more depressed.

Evidence linking parental depression to childhood asthma appears to be stronger for morbidity outcomes among those already diagnosed with asthma than for predicting asthma onset [53].

Greater parental stress also has been linked to increased asthma morbidity in children. For example, Shalowitz and colleagues [50] documented that caregivers with more negative life stressors were more likely to have children who used health care services and who experienced more severe asthma symptoms. Longitudinal studies show similar effects. Among a sample of children diagnosed with asthma, reports indicating greater life stress for parents predicted a greater likelihood of asthma hospitalization as well as poorer daily functioning in children over the following 9 months [41]. High levels of caregiver stress when infants were 2 to 3 months old predicted a higher likelihood of repeated wheezing in children by 14 months of age [54]. As with maternal depression, evidence suggests that parental stress may not predict asthma diagnosis [48,53]. However, parental stress may interact with family relationship problems to predict asthma. Parents who experienced a combination of both parenting difficulties (when the child was 3 weeks of age) and high stress at the end of pregnancy were five times more likely to have a child who developed asthma by the age of 3 compared with those without parenting difficulties (with or without high stress), and 2.5 times as likely to develop asthma compared with those with parenting difficulties but with low stress [55].

Explaining family effects

How does the psychologic state of one individual (a parent) come to affect asthma outcomes in another individual (the child)? One possibility is that the difficulties that children experience early in life may program their biological systems in a way that increases their vulnerability to asthma later in life. For example, difficulties bonding with parents may lead to insecure attachments and difficulties regulating emotions, which in turn may enhance the biological responses to stresses in children [56]. Certain family characteristics may also influence the amount of stress a child perceives. For example, family conflict or a poor quality family relationship can be a source of stress in and of itself. In addition, parental depression or stress may create stress for the child by affecting how parents interact with their child or because children are sensitive in picking up on the psychologic states of their parents.

Another possibility is that the family environment influences how children cope with events in their own lives. Coping refers to cognitive and behavioral efforts aimed at dealing with external demands from the environment [57]. One important type of coping strategy is to draw on close others to help you with your problems. A parent struggling with depression or significant life stressors may be less available to help a child deal with his or her problems. Hence, having low levels of family support may heighten

the impact of a stressor, given that these children will have more limited resources for getting help with their problems. In addition, children from difficult family circumstances may have parents who model maladaptive coping strategies. For example, children may observe parents with depression or stress struggling to cope with their own problems, and may attempt similar ineffective coping strategies for their problems. Or, such parents may use less effective coping strategies when parenting their child, leading children to have greater difficulty regulating their own emotional responses in daily life.

Another possibility is that family factors affect the health behaviors a child engages in, which in turn affects asthma morbidity. For example, parents who have high levels of depression or stress may be less available to supervise their child's behaviors. This could lead to a greater influence of peers in shaping detrimental health behaviors, such as smoking. These parents may also have a more difficult time monitoring and ensuring their child's compliance with asthma medications. This could mean that, among children whose parents are depressed or stressed, asthma is more likely to be improperly managed on a daily basis, leading to greater asthma morbidity over time.

Biological mechanisms

The above studies have linked social environment factors to clinical asthma outcomes. However, the question still remains of how the larger social environment gets "under the skin" to influence disease. Is there evidence that social environment factors are linked to biological processes implicated in asthma? In this section, we first briefly overview the types of inflammatory mechanisms relevant to asthma, and then address whether social factors are associated with these pathways. Other articles have provided broader overviews of biological systems implicated in biopsychosocial models of asthma [58–60].

Inflammatory pathways in asthma

One pathway to airway inflammation in asthma involves the activation of certain T helper cells, which in turn release cytokines that coordinate inflammatory responses. These cells, known as Th-2 cells, promote B cell proliferation and differentiation, leading to a humoral response involving antibody synthesis. For example, the Th-2 cytokines IL-4 and IL-13 bind to B cells, inducing them to synthesize and release IgE antibodies. IgE then binds to mast cells residing in the airways, causing them to degranulate and release allergic mediators, such as histamines and leukotrienes. Histamines and leukotrienes cause edema, smooth muscle constriction, and mucus production. A second type of inflammatory response is generated when Th-2 cells release IL-5. This cytokine recruits eosinophils into the airways, where they bring about inflammation and obstruction. Eosinophils also release mediators,

such as eosinophil cationic protein and major basic protein, which can bring about damage to airway cells, and leukotrienes, which cause edema and further bronchial constriction.

Social factors and inflammatory responses

At the neighborhood level, low SES has been linked to asthma-related inflammatory markers in children with asthma. In one study, adolescents diagnosed with persistent asthma were recruited from either low-SES or high-SES neighborhoods (based on the percentage of people living below poverty in each neighborhood). Peripheral blood was drawn from adolescents, and their cells were stimulated with a combination of phorbol myristate acetate and ionomycin to induce the production of cytokines. Adolescents with asthma from low-SES neighborhoods displayed significantly greater production of IL-5 compared with adolescents with asthma from high-SES neighborhoods, despite their cells having been exposed to an equivalent dose of mitogens [61]. In a subsequent study, children with asthma were recruited from a range of SES backgrounds. A linear relationship was found, such that as family SES decreased, production of IL-5, IL-13, and eosinophil counts all increased in children with asthma, but not among healthy children [62]. These findings suggest that low-SES children with asthma exhibit heightened inflammatory responses, and that the direction of these responses may help explain why low SES is linked to more severe exacerbations of asthma.

At the level of social networks and social support, one previously mentioned study [28] found that unlike family and neighborhood factors, peer support was not related to any of the biological variables investigated, including the production of cytokines implicated in asthma or eosinophil counts, in youth with asthma. These findings suggest that the link between peer support and biological processes in children with asthma is weak, perhaps consistent with the fact that the link between social support and clinical asthma outcomes is mixed. Another study examining the effect of social support on stress before and after examinations in high school students found that perceived social support decreased during examination time and that this decrease was accompanied by a decrease in natural killer cells [63]. However, this pattern for natural killer cells was found in both adolescents with asthma and in healthy adolescents, suggesting that social support may not activate immune pathways specific to asthma.

At the family level, recent evidence suggests that parent psychologic states are associated with child biological profiles. Higher levels of parental depression have been associated with eosinophil cationic protein (ECP), a substance released when eosinophils are activated. Specifically, greater parental depression at baseline predicted increases in ECP over the following 6 months in a sample of children with asthma and a sample of healthy children [64]. With respect to parental stress, higher levels of parental

perceived stress at baseline predicted greater increases over the following 6 months both in ECP as well as in stimulated production of IL-4 in these same samples of children [64]. Among young children, high levels of parental stress during the first 6 months of the child's life predicted higher IgE levels at 2 years of age, as well as an increased proliferation of children's lymphocytes after *in vitro* exposure to selected allergens [65]. These findings suggest that poorer parent mental health is associated with inflammatory profiles in children in a direction detrimental to asthma.

In earlier sections, we hypothesized that one reason why larger social environmental factors influence health outcomes is because environmental factors, such as low SES and poor family relationships, affect the stress that an individual experiences. In turn, clinical evidence indicates individuals who experience greater stress also are at risk for poorer asthma outcomes [35,66,67]. Recent evidence corroborates this clinical data, suggesting that stress affects inflammatory processes of individuals with asthma. For example, in one study, college students with asthma were tested during periods of high stress (final examination period) and during periods of low stress (no major examination) [68]. Each time, participants inhaled increasing dosages of allergens to which they were sensitized (ragweed, cat, or dust mite) until their pulmonary functioning declined by 20% or more. There was evidence of a greater immune response to this challenge during times of stress. During final examinations, the allergen challenge elicited greater numbers of eosinophils in both sputum and blood compared with periods outside of examination time. A parallel finding emerged for *in vitro* production of IL-5 in sputum treated with phytohemagglutinin.

In a separate series of studies, high school students were studied before an examination (baseline period of low stress) and after examinations (high-stress period), and their peripheral blood cells were stimulated *in vitro* with various mitogens. In one study, students with asthma had greater production of IL-5 postexamination compared with students who were healthy. In contrast, there were no group differences in IL-5 production at baseline. This suggests that under conditions of low stress, individuals with asthma do not differ from healthy individuals in their responsiveness to mitogens, but that periods of stress heighten the responsiveness of Th-2 immune cells to mitogens in individuals with asthma [69]. A second study from this group documented that examination stress was associated with reduced production of the Th-1 cytokines IFN- γ and IL-2, but increased production of the proinflammatory cytokine IL-6 (argued by this group to represent Th-2) across both a sample of students with asthma and a sample of healthy students [70]. Collectively, these studies suggest that, among patients with asthma, stress can amplify the Th-2 cytokine response to asthma triggers and mitogens. Over time, such an inflammatory pattern could lead to more frequent or severe symptoms of asthma.

Taken together, the studies reviewed in this section provide intriguing evidence that the larger social environment—in particular at the family and

neighborhood levels—can alter biological systems in individuals. This link between the social environment and biology is important to provide plausible explanations for how it is that social environments come to influence the course of diseases. With additional empiric evidence from future studies, researchers can begin to develop more comprehensive models of how larger social factors affect individuals psychologically and behaviorally, and in turn what the biological and long-term health implications of these effects are.

Summary

In this article, we addressed the question of whether the social environment affects the onset or course of asthma. We reviewed evidence showing that neighborhood-level factors, such as low SES and community violence, are associated with greater asthma morbidity. In addition, family-level factors, such as poor quality family relationships, parental depression, and parental stress, are associated with poorer asthma outcomes. In contrast, peer-level factors, such as social support, were more mixed in their association with asthma, with some studies finding that support is beneficial for asthma, and others finding no relationship or even detrimental effects of peers on asthma. Longitudinal studies indicate that neighborhood and family social factors precede asthma, rather than being a consequence of asthma. The strongest evidence exists for social factors predicting morbidity among those already diagnosed with asthma, with evidence being more mixed for social factors predicting the development of asthma. Social factors likely operate via direct effects on physiologic systems, as well as by changing health behaviors in individuals with asthma. In sum, while exposure to factors in the physical environment has long been recognized as contributing to asthma, the studies reviewed in this article highlight the importance of also considering social exposures in asthma. To push the field forward in developing more comprehensive models of asthma, the concept of environmental exposures in asthma needs to be more broadly defined. Exposures, both physical and social, affect asthma in significant ways.

References

- [1] Sigurs N, Gustafsson PM, Bjarnason R, et al. Severe respiratory syncytial virus bronchiolitis in infancy and asthma and allergy at age 13. *Am J Respir Crit Care Med* 2005;171(2):137–41.
- [2] Nelson HS, Szefer SJ, Jacobs J, et al. The relationships among environmental allergen sensitization, allergen exposure, pulmonary function, and bronchial hyperresponsiveness in the childhood asthma management program. *J Allergy Clin Immunol* 1999;104:775–85.
- [3] Ober C, Hoffjan S. Asthma genetics 2006: the long and winding road to gene discovery. *Genes Immun* 2006;7(2):95–100.
- [4] Weiss ST. Gene by environment interaction and asthma. *Clin Exp Allergy* 1999;29(2):96–8.
- [5] Yen IH, Yelin EH, Katz P, et al. Perceived neighborhood problems and quality of life, physical functioning, and depressive symptoms among adults with asthma. *Am J Public Health* 2006;96(5):873–9.

- [6] Yen IH, Yelin E, Katz P, et al. Impact of perceived neighborhood problems on change in asthma-related health outcomes between baseline and follow-up. *Health Place* 2008;14: 468–77.
- [7] Adler NE, Epel ES, Castellazzo G, et al. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in health white women. *Health Psychol* 2000;19:586–92.
- [8] Blascovich J, Mendes WB, Hunter SB, et al. Perceiver threat in social interactions with stigmatized others. *J Pers Soc Psychol* 2001;80:253–67.
- [9] Miller JE. The effects of race/ethnicity and income on early childhood asthma prevalence and health care use. *Am J Public Health* 2000;90:428–30.
- [10] Wood PR, Smith LA, Romero D, et al. Relationships between welfare status, health insurance status, and health and medical care among children with asthma. *Am J Public Health* 2002;92(9):1446–52.
- [11] Simon PA, Zeng ZW, Wold CM, et al. Prevalence of childhood asthma and associated morbidity in Los Angeles County: impacts of race/ethnicity and income. *J Asthma* 2003;40(5): 535–43.
- [12] Dales RE, Choi B, Chen Y, et al. Influence of family income on hospital visits for asthma among Canadian school children. *Thorax* 2002;57(6):513–7.
- [13] Maziak W, Von Mutius E, Keil U, et al. Predictors of health care utilization of children with asthma in the community. *Pediatr Allergy Immunol* 2004;15(2):166–71.
- [14] Amre DK, Infante-Rivard C, Gautrin D, et al. Socioeconomic status and utilization of health care services among asthmatic children. *J Asthma* 2002;39:625–31.
- [15] Goodman DC, Stukel TA, Chang C. Trends in pediatric asthma hospitalization rates: regional and socioeconomic differences. *Pediatrics* 1998;101:208–13.
- [16] Claudio L, Stingone JA, Godbold J. Prevalence of childhood asthma in urban communities: the impact of ethnicity and income. *Ann Epidemiol* 2006;16(5):332–40.
- [17] Castro M, Schechtman KB, Halstead J, et al. Risk factors for asthma morbidity and mortality in a large metropolitan city. *J Asthma* 2001;38(8):625–35.
- [18] Persky VW, Slezak J, Contreras A, et al. Relationships of race and socioeconomic status with prevalence, severity, and symptoms of asthma in Chicago school children. *Ann Allergy Asthma Immunol* 1998;81(3):266–71.
- [19] Cesaroni G, Farchi S, Davoli M, et al. Individual and area-based indicators of socioeconomic status and childhood asthma. *Eur Respir J* 2003;22(4):619–24.
- [20] Grant EN, Wagner R, Weiss KB. Observations on emerging patterns of asthma in our society. *J Allergy Clin Immunol* 1999;104(2):S1–9.
- [21] Webber MP, Carpiniello KE, Oruwariye T, et al. Prevalence of asthma and asthma-like symptoms in inner-city elementary schoolchildren. *Pediatr Pulmonol* 2002;34(2):105–11.
- [22] Saha C, Riner ME, Liu G. Individual and neighborhood-level factors in predicting asthma. *Arch Pediatr Adolesc Med* 2005;159(8):759–63.
- [23] Juhn YJ, St Sauver J, Katusic S, et al. The influence of neighborhood environment on the incidence of childhood asthma: a multilevel approach. *Soc Sci Med* 2005;60(11): 2453–64.
- [24] Yawn BP, Wollan P, Kurland M, et al. A longitudinal study of the prevalence of asthma in a community population of school-age children. *J Pediatr* 2002;140(5):576–81.
- [25] Graham PJ, Rutter ML, Yule W, et al. Childhood asthma: a psychosomatic disorder? *Br J Prev Soc Med* 1967;21:78–85.
- [26] Wright RJ. Health effects of socially toxic neighborhoods: the violence and urban asthma paradigm. *Clin Chest Med* 2006;27(3):413–21.
- [27] Wright RJ, Mitchell H, Visness CM, et al. Community violence and asthma morbidity: the inner-city asthma study. *Am J Public Health* 2004;94(4):625–32.
- [28] Chen E, Chim LS, Strunk RC, et al. The role of the social environment in children and adolescents with asthma. *Am J Respir Crit Care Med* 2007;176:644–9.

- [29] Clougherty JE, Levy JI, Kubzansky LD, et al. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. *Environ Health Perspect* 2007;115:1140–6.
- [30] Brady SS, Matthews KA. The effect of socioeconomic status and ethnicity on adolescents' exposure to stressful life events. *J Pediatr Psychol* 2002;27:575–83.
- [31] Wright RJ, Steinbach SF. Violence: an unrecognized environmental exposure that may contribute to greater asthma morbidity in high risk inner-city populations. *Environ Health Perspect* 2001;109:1085–9.
- [32] Sin MK, Kang DH, Weaver M. Relationships of asthma knowledge, self-management, and social support in African American adolescents with asthma. *Int J Nurs Stud* 2005;42(3):307–13.
- [33] Knight D. Beliefs and self-care practices of adolescents with asthma. *Issues Compr Pediatr Nurs* 2005;28:71–81.
- [34] Wainwright NW, Surtees PG, Wareham NJ, et al. Psychosocial factors and incident asthma hospital admissions in the EPIC-Norfolk cohort study. *Allergy* 2007;62(5):554–60.
- [35] Smyth JM, Soefer MH, Hurewitz A, et al. Daily psychosocial factors predict levels and diurnal cycles of asthma symptomatology and peak flow. *J Behav Med* 1999;22(2):179–93.
- [36] Smith A, Nicholson K. Psychological factors, respiratory viruses and exacerbation of asthma. *Psychoneuroendocrinology* 2001;26:411–20.
- [37] Levy JI, Brugge D, Peters JL, et al. A community-based participatory research study of multifaceted in-home environmental interventions for pediatric asthmatics in public housing. *Soc Sci Med* 2006;63(8):2191–203.
- [38] Rand CS, Butz AM, Kolodner K, et al. Emergency department visits by urban African American children with asthma. *J Allergy Clin Immunol* 2000;105:83–90.
- [39] Gustafsson PA, Kjellman NI, Bjorksten B. Family interaction and a supportive social network as salutogenic factors in childhood atopic illness. *Pediatr Allergy Immunol* 2002;13(1):51–7.
- [40] Berz JB, Carter AS, Wagmiller RL, et al. Prevalence and correlates of early onset asthma and wheezing in a healthy birth cohort of 2- to 3-year olds. *J Pediatr Psychol* 2007;32(2):154–66.
- [41] Weil CM, Wade SL, Bauman LJ, et al. The relationship between psychosocial factors and asthma morbidity in inner-city children with asthma. *Pediatrics* 1999;104:1274–80.
- [42] Finnvold JE. Access to specialized, health care for asthmatic children in Norway: the significance of parents' educational background and social network. *Soc Sci Med* 2006;63(5):1316–27.
- [43] Mancuso CA, Sayles W, Robbins L, et al. Barriers and facilitators to healthy physical activity in asthma patients. *J Asthma* 2006;43(2):137–43.
- [44] Ngamvitroj A, Kang DH. Effects of self-efficacy, social support and knowledge on adherence to PEFr self-monitoring among adults with asthma: a prospective repeated measures study. *Int J Nurs Stud* 2007;44(6):882–92.
- [45] Sturdy PM, Victor CR, Anderson HR, et al. Psychological, social and health behaviour risk factors for deaths certified as asthma: a national case-control study. *Thorax* 2002;57(12):1034–9.
- [46] Precht DH, Keiding L, Nielsen GA, et al. Smoking among upper secondary pupils with asthma: reasons for their smoking behavior: a population-based study. *J Adolesc Health* 2006;39(1):141–3.
- [47] Mrazek DA, Klinnert M, Mrazek PJ, et al. Prediction of early-onset asthma in genetically at-risk children. *Pediatr Pulmonol* 1999;27:85–94.
- [48] Klinnert MD, Nelson HS, Price MR, et al. Onset and persistence of childhood asthma: predictors from infancy. *Pediatrics* 2001;108:e69.
- [49] Strunk RC, Mrazek DA, Fuhrmann GS, et al. Physiologic and psychological characteristics associated with deaths due to asthma in childhood. *J Am Med Assoc* 1985;254:1193–8.

- [50] Shalowitz MU, Berry CA, Quinn KA, et al. The relationship of life stressors and maternal depression to pediatric asthma morbidity in a subspecialty practice. *Ambul Pediatr* 2001; 1(4):185–93.
- [51] Brown ES, Gan V, Jeffress J, et al. Psychiatric symptomatology and disorders in caregivers of children with asthma. *Pediatrics* 2006;118(6):E1715–20.
- [52] Bartlett SJ, Kolodner K, Butz AM, et al. Maternal depressive symptoms and emergency department use among inner-city children with asthma. *Arch Pediatr Adolesc Med* 2001; 155(3):347–53.
- [53] Horwood LJ, Fergusson DM, Shannon FT. Social and familial factors in the development of early childhood asthma. *Pediatrics* 1995;75:859–68.
- [54] Wright RJ, Cohen S, Carey V, et al. Parental stress as a predictor of wheezing in infancy: a prospective birth-cohort study. *Am J Respir Crit Care Med* 2002;165:358–65.
- [55] Klinnert MD, Mrazek PJ, Mrazek DA. Early asthma onset: the interaction between family stressors and adaptive parenting. *Psychiatry* 1994;57:51–61.
- [56] Taylor SE, Lerner JS, Sage RM, et al. Early environment, emotions, responses to stress, and health. *J Pers* 2004;72(6):1365–93.
- [57] Lazarus RS, Folkman S. *Stress, appraisal, and coping*. New York: Springer Publishing Company; 1984.
- [58] Wright RJ, Rodriguez M, Cohen S. Review of psychosocial stress and asthma: an integrated biopsychosocial approach. *Thorax* 1998;53:1066–74.
- [59] Wright RJ. Stress and atopic disorders. *J Allergy Clin Immunol* 2005;116:1301–6.
- [60] Chen E, Miller GE. Stress and inflammation in exacerbations of asthma. *Brain Behav Immun* 2007;21:993–9.
- [61] Chen E, Fisher EB Jr, Bacharier LB, et al. Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosom Med* 2003;65:984–92.
- [62] Chen E, Hanson MD, Paterson LQ, et al. Socioeconomic status and inflammatory processes in childhood asthma: the role of psychological stress. *J Allergy Clin Immunol* 2006;117(5): 1014–20.
- [63] Kang DH, Coe CL, Karaszewski J, et al. Relationship of social support to stress responses and immune function in healthy and asthmatic adolescents. *Res Nurs Health* 1998;21(2): 117–28.
- [64] Wolf JM, Miller GE, Chen E. Parent psychological states predict changes in inflammatory markers in children with asthma and healthy children. *Brain Behav Immun* 2008;22:433–41.
- [65] Wright RJ, Finn P, Contreras JP, et al. Chronic caregiver stress and IgE expression, allergen-induced proliferation, and cytokine profiles in a birth cohort predisposed to atopy. *J Allergy Clin Immunol* 2004;113(6):1051–7.
- [66] Sandberg S, Paton JY, Ahola S, et al. The role of acute and chronic stress in asthma attacks in children. *Lancet* 2000;356:982–7.
- [67] Kilpelainen M, Koskenvuo M, Helenius H, et al. Stressful life events promote the manifestation of asthma and atopic diseases. *Clin Exp Allergy* 2002;32(2):256–63.
- [68] Liu LY, Coe CL, Swenson CA, et al. School examinations enhance airway inflammation to antigen challenge. *Am J Respir Crit Care Med* 2002;165:1062–7.
- [69] Kang D, Coe C, McCarthy DO, et al. Cytokine profiles of stimulated blood lymphocytes in asthmatic and healthy adolescents across the school year. *J Interferon Cytokine Res* 1997;17: 481–7.
- [70] Kang DH, Fox C. Th1 and Th2 cytokine responses to academic stress. *Res Nurs Health* 2001;24(4):245–57.