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Inner-city asthma

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The rate of asthma has increased dramatically in the past 20 years, both in the United States and worldwide [1]. Data collected by the Centers of Disease Control and Prevention over the last 4 decades show large and unexplained increases in asthma prevalence, hospitalization rates, and mortality in the United States [2]. Between 1980 and 1994, asthma prevalence rose from 3.1% to 5.4% [2]. This asthma epidemic caused heavy increases in health expenditure; direct and indirect monetary costs related to asthma were estimated at approximately \$12.7 billion in 1998 [3].

Childhood asthma is the most frequent health-related cause of school absenteeism, accounting for more than 10 million missed school days annually. Asthma is the most common admitting diagnosis for children [2,4,5], accounting for 159,000 annual hospitalizations with an average length of stay of 3.4 days. The largest increase in asthma hospitalizations has occurred in the youngest of children. From 1979 to 1980, the number of children aged 4 years and younger who were hospitalized for asthma was 34.3 per 10,000 population, but this number escalated to 58.3 per 10,000 in 1990 to 1992 and remained high at 49.7 per 10,000 in 1993 to 1994 [2]. In comparison, hospitalizations for children aged 5 to 14 years have remained relatively stable, with rates of 15.9, 20.6, and 18.0 per 10,000 population for the same respective time periods [2].

Asthma rates vary between regions, states, cities and neighborhoods. From 1988 to 1994, the Northeast region of the United States had more asthma hospitalizations than did the Western and Southern regions across all age groups. During that same time period, asthma hospitalizations increased in the Northeast region but decreased in the Midwestern and Western regions. By 1993 to 1994, age-adjusted hospitalizations were higher among persons residing in the Northeast

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than among persons residing in the West [2]. State-to-state differences also occur, with state-specific estimates for asthma prevalence ranging from 5.8% in Oklahoma to 7.2% in Nevada [6]. Though many state estimates are of self-reported prevalence (ie, not objective surveillance measures), they are consistent with other studies using outpatient data [7]. There is little state-to-state variation within each region, with most states having rates consistent with their region's average rate of asthma prevalence.

Urban areas seem to have a higher burden of asthma compared with less populous areas, with asthma prevalence often growing at a rapid rate. In the St. Louis metropolitan area, childhood asthma hospitalizations increased three-fold from 1983 to 1995, much higher than the increase of hospitalizations across the state of Missouri [8,9]. In Boston, the average rate of hospitalization in 2000 was over twice the rate in Massachusetts among children aged 18 years and younger [10].

Within cities, variation in rates of asthma occurs, with less affluent, inner-city areas being the most affected. Population-based surveys of children in inner-city New York [11] and Chicago [12] estimated the prevalence of recently symptomatic, physician-diagnosed asthma at between 8.6% and 14.3%, which is two to three times the US rate. Small-area analyses in New York, Chicago, Los Angeles, Boston [13], and Philadelphia [14] have shown excess asthma hospitalization rates in inner-city neighborhoods characterized by high rates of poverty, low educational attainment, and high percentages of racial and ethnic minority residents. Studies of public housing developments have found rates of adults reporting asthma diagnoses that are as high as 26% [15] to 40% [16].

Asthma differs from region to region, from neighborhood to neighborhood, and even from block to block within inner cities. Understanding the disproportionate burden of asthma in inner cities may provide insight into the roots of the asthma epidemic.

Aeroallergen exposure and inner-city asthma

Allergens seem to have a critical role in the severity and chronicity of asthma in inner cities. Epidemiologic studies have established an association between elevated serum total immunoglobulin E (IgE) and asthma [17]. Specific IgE antibody to indoor allergens, including those derived from dust mite, cockroach, cat, and dog, is also a common finding in asthma. Many studies have found multiple allergens in homes, and multiple sensitivities among inner-city residents with asthma. Case-control studies of children in Atlanta [18] and adults in Wilmington, Delaware [19], revealed that subjects presenting with acute asthma exacerbations were substantially more likely than nonasthmatic controls to have sensitivity to an indoor allergen (cockroach, dust mite, or cat) and significant exposure to that allergen in their homes. A study by Call and colleagues illustrates the complexity of asthma and the environment [18]. They studied 144 poor children, both with and without an asthma diagnosis, attending an inner-city

emergency room at Grady Memorial Hospital in Atlanta. Controls and asthmatic patients were exposed heavily: 79% of home dust samples contained excessive mite allergen, and 87% contained excessive cockroach allergen. Asthmatic children were sensitized to the allergens. Sixty-nine percent of these children had IgE antibody to mite, cockroach, or cat, compared with only 27% of controls. Of 35 asthmatic children whose homes were sampled, 21 were sensitized and exposed to the same allergens, compared with 3 of 22 control children (odds ratio, 9.5; $P < 0.001$). The specific allergens that seem to have important roles in inner-city asthma are reviewed.

Cockroach

Cockroach allergen is a well-described allergic trigger for asthma that is important in low-income urban communities. Though many species of cockroach exist, the German cockroach (*Blattella germanica*), the most common cockroach species infesting homes in cities in the United States and many other parts of the world, is the best studied. Multiple allergens have been detected in different extracts of German cockroach, some that are specific to the German cockroach and some that are cross-reactive with allergens from other species, such as the American cockroach (*Periplaneta americana*) [20–23]. Two major German cockroach allergens, defined by the high prevalence of specific IgE among cockroach-allergic patients, have been designated *Bla g 1* and *Bla g 2* [23,24]. *Bla g 1* is a homologous protein found in German and American cockroaches, with 30% of patients in Virginia testing positive for the specific IgE [23]. *Bla g 2* has been purified only from German cockroach and is found in 60% of cockroach allergic patients in Virginia [24]. The protein was shown to be similar to aspartic proteases, a type of digestive enzyme [25]. Another German cockroach allergen, *Bla g 4*, is also common in cockroach allergic patients in Virginia; 40% to 60% of patients had the specific IgE [25], and its protein has shown similarities to a calycin [26].

Cockroach allergen is also a powerful trigger for asthma. Specific bronchial provocation studies show that most asthma patients with positive skin tests to cockroach extract developed an acute asthmatic response after inhalation of cockroach-extract aerosol in the laboratory [20,27]. Most of these patients also display a late asthmatic reaction (dual response) hours later [20]. Asthmatics with negative cockroach skin tests, in contrast, do not react after aerosol inhalation of cockroach extract [20,27].

Cockroach allergens are more common in urban than rural settings, though the prevalence and severity of allergen ranges considerably. In Washington, DC, 28% of 114 unselected allergy patients had positive allergy skin tests to German cockroach extract, compared with only 7.5% of 253 healthy controls [28]. In New York, 44% of 589 patients seen at hospital-based allergy clinics had positive skin test reactions to German cockroach extract [29]. Other studies have corroborated moderate-to-high rates of immediate cutaneous hypersensitivity to cockroach extract among asthma and allergy patients in other US cities [30–34] and in other parts of the world [35–37].

Differences in cockroach sensitivity may account for some of the variability in asthma rates within cities. In a study of asthmatic children in metropolitan Baltimore, urban children were more likely to have a positive skin test to cockroach extract than were suburban children [38]. Black race and low socioeconomic status were associated independently with skin test reactivity to cockroach extract [38]. In other studies, analysis by racial-ethnic subgroup revealed striking variation in the prevalence of skin test reactivity to cockroach extract, with a prevalence of 59% among Puerto Rican patients, an ethnic group that predominantly is found in cities [29].

A high proportion of low-income urban homes have detectable cockroach allergen in vacuumed house dust [18,19,38]. Twenty-seven (71%) of 38 of urban homes sampled in Atlanta had detectable *Bla g 1* in vacuumed dust (range, 4–1340 U/g of fine dust) [29]. In the National Cooperative Inner City Asthma Study (NCICAS), the homes of 406 (85%) of 476 urban asthmatic children had detectable *Bla g 1* allergen in bedroom dust, and 239 (50%) homes had levels of cockroach allergen, exceeding 8 U/g in sampled dust [33]. In the northeastern United States, high levels of *Bla g 1* or *Bla g 2* in vacuumed dust are associated with urban residence, black race, and low socioeconomic status [38]. Within a given home, the highest levels of cockroach allergens are usually in the kitchen, where food and moisture promote infestation. Substantial levels of allergen also are found in bedding, bedroom floors, and upholstered furniture [39,40].

Cockroach allergens can be found even homes that do not have apparent infestation. In a study in Wilmington, Delaware, 20% of homes with no visible evidence of cockroach infestation had detectable levels of *Bla g 2* in at least one room [18]. Cockroach allergens seem to be associated with relatively large particles that remain airborne in detectable quantities for only a short time after disturbance [19].

Exposure to these allergens within the home may promote sensitization to cockroaches. Among 81 asthmatic children in Baltimore, the levels of cockroach allergen (*Bla g 1* or *Bla g 2*) in the bedroom were associated significantly with skin test reactivity to cockroach extract, even after adjustment for covariates such as age, race, and socioeconomic status [38]. Of the 22 asthmatic children with a bedroom level of *Bla g 1* or *Bla g 2* that exceeded 1 U/g of dust, over 80% had a positive skin test to cockroach extract.

Although many asthmatic children have multiple allergies, cockroach allergy is associated with particularly severe and chronic asthma [41]. The NCICAS examined skin test sensitivity, bedroom dust allergen levels, and asthma morbidity among 476 asthmatic children [42]. Children with the combination of a positive skin test reaction to cockroach extract and a bedroom level of *Bla g 1* exceeding 8 U/g of dust had significantly more hospitalizations for asthma, unscheduled medical visits, and parent-reported wheezing than did other children. Cockroach skin test reactivity without exposure and cockroach allergen exposure without skin test reactivity were not associated with increased morbidity. In this population, exposure to dust mite and cat allergens was much less common than exposure to cockroach allergen, and exposure to cat and dust mite allergens was not related significantly to asthma morbidity.

Cross-sectional studies of current hypersensitivity and exposure status are unable to elucidate the temporal and dose-response aspects of the relation of exposure to the development of allergic sensitization. A birth-cohort study in Boston has shown that exposure to cockroach allergen and sensitization is associated with an increased incidence of wheeze in the first year of life [43] and incident asthma among the sibling of the index children [44]. The same factors may relate to asthma severity in urban adolescents [45].

A combination of exposure and sensitization to cockroach allergens markedly increases the risk for morbidity from asthma. This combination seems to be more prevalent in inner cities and may explain some of the disparity in asthma rates and severity in this population.

Dust mites

Dust mites may be the best-studied allergen connected with asthma [46]. From the genus *Dermatophagoides*, these mites deposit feces onto household items such as bedding, drapes, upholstered furniture, and carpets. The two most common dust mite allergens, Der p 1 and Der p 2 [47], are very potent; direct inhalation causes an immediate fall in forced expiratory volume in 1 second (FEV₁) and late-phase response in sensitized asthmatics [48]. Studies have found varying rates of dust mite sensitization among asthmatics, from 31% to 24% of these allergens [42].

Compared with cockroach allergen, dust mite allergen may be found in some inner-city areas less frequently and in lower concentrations [49]. The prevalence and severity of dust mites differ from region to region, and regions with variable humidity during the course of the year may have lower dust mite levels. The NCICAS, a multicenter study limited to cities in the northeastern United States, found that house dust mite allergens were elevated (over 2 µg/g of house dust) in only 10% of homes, whereas 77% of homes had high levels of cockroach allergen (over 2 µg/g of house dust) [33]. In contrast, regions of the country with high humidity throughout the year may have high levels of dust mite allergens in most homes. Exposure to dust mite allergens may be high in some inner-city areas, where human crowding; high indoor humidity caused by leaking pipes and poor ventilation; and older carpeting, bedding, and upholstered furniture provide excellent conditions for mite proliferation [50].

Exposure to dust mite allergens may be a risk factor for the development of childhood asthma. Studies in England have shown that children who were exposed to dust mite levels exceeding 8 µg/g of bedroom dust developed asthma [51]. These results have been confirmed by a case-control study [52] and a birth-cohort study [53], which demonstrated that exposure to dust mite allergen in early life was associated with an increased risk for asthma in later childhood.

Pets and other animals

Many pet allergens are ubiquitous. Exposure can continue even after removal of the pets. Because many inner-city homes have pets, or a history of pets, many children with asthma are exposed to pet allergens continuously. Given the small

size of the protein, it has been difficult to eradicate pet allergens, and vacuuming can disperse allergens to the air [54].

Specific IgE testing shows that as many as 22% of children in inner cities tested positive for cat allergen [33]. Data from the Childhood Asthma Management Program (CAMP) have demonstrated a strong direct correlation between increased sensitivity to inhaled methacholine and skin test sensitivity to tree, weed, *Alternaria*, cat, dog, and indoor molds [55]. When the relationship was examined by step-wise regression, the skin test sensitivities to dog, *Alternaria*, and cat showed the strongest association with bronchial reactivity to methacholine. Sensitization to certain pet allergens has an important role in the ongoing bronchial reactivity in children with mild-to-moderate asthma.

As the CAMP cohort demonstrates, attempts to remove pets and pet allergens is difficult. Despite environmental controls to reduce or remove pet allergens, 39% of patients who reported frequent asthma symptoms after exposure to animals continued to live with a furry pet in their homes [56]. Many of these patients were atopic and suffered from nocturnal asthma, a feature of severe asthma that is associated with increased asthma mortality. Compared with patients without awakenings, patients with nocturnal asthma symptoms had greater eosinophil counts, higher IgE levels, and greater frequency of positive skin tests, especially to indoor allergens [57]. The combination of a positive skin test to dog and a high level of dog allergen in settled dust was associated significantly with a greater risk for awakening from asthma. The same significant association was found between the combination of a positive skin test to cat and a high level of cat allergen in settled dust [57].

Unwanted infestations of mice and rats characterize many inner-city homes. Like pet allergens, rodent hair, urine, and fecal allergens are difficult to remove after eradication of the pests. Though no good commercial testing for home dust for rats is available, many labs run assays for mouse urinary proteins, which may mimic rat exposure.

Studies have shown that sensitization and exposure to rodents can make asthma worse. Epidemiologic studies among workers in laboratories with rodents showed increases in asthma symptoms and decreased FEV₁ among workers who handled rats [58]. Other studies showed positive associations between rodent allergy and asthmatic symptoms [59].

Many inner-city residents are exposed and are allergic to rodents. In the NCICAS, 19% of asthmatic children were sensitized to rats, and 15% were sensitized to mice [33]. Little is known, however, about residential exposure to rodent allergens and its impact on asthma morbidity in urban populations.

Mold and dampness

Certain conditions, such as dampness, have multiple effects on the home indoor environment, resulting in poor health [60]. Though the definition of normal indoor humidity changes considerably by region or by season, most experts agree a range from 20% to 60% is acceptable. Excessive humidity or dampness causes growth of

mold and fungi, encourages growth of dust mites, and invites pests into the home. These conditions are easy to screen for in an office setting, because many families can judge excessive humid conditions or report mold odor or mold growth in the home to a physician [61]. Inner-city housing is notorious for its old and dilapidated infrastructure, which invites leaks and mold growth. Although many sanitary codes call for the prevention of mold growth, families continue to have problems with mold allergy and asthma as a result of these conditions.

Beyond changing the structure of housing, few options remain to change the humidity of indoor structures. Effects of portable dehumidification systems on humidity and levels of house dust mite have been small [62]. New interventions have focused on housing construction and renovation to prevent water intrusion. Though these studies have not focused on connecting health outcomes with construction changes, new studies are underway that focus on these possible connections.

Infections

Viral respiratory infections are known to worsen existing asthma [63], but their role in the pathogenesis of asthma and allergy remains unclear. Respiratory syncytial virus (RSV) infection in infancy has been associated with subsequent episodes of wheezing, reduced lung function, and airway hyperresponsiveness, but has not been associated with allergic sensitization [64–66]. By age 13, children with a history of RSV bronchiolitis have no increased risk for wheezing [67]. These findings suggest that symptomatic RSV bronchiolitis in infancy may be a marker of hyperreactive airways rather than a causal risk factor for the subsequent development of asthma. The role of RSV infection in the pathogenesis of asthma, however, has yet to be established fully.

Some epidemiologic data have suggested that early-life infections may protect against the development of asthma. Ecologic analyses have revealed that populations with relatively high rates of early-childhood respiratory infections have a relatively low prevalence of asthma [68–70]. A prospective cohort of children in Tucson, Arizona, showed that tracheobronchitis and pneumonia in the first 3 years of life were associated with decreased allergy skin test reactivity and lower serum total IgE concentrations at age 6 years, compared with children without these diagnoses [71]. Data from Europe indicate that children exposed to farm animals in their home environments are less likely to develop hay fever, allergic sensitization, and asthma than are nonexposed children [72–74]. Some investigators have hypothesized that early-childhood vaccination and antibiotic administration may influence the maturation of the immune system and increase the risk for allergies, but this theory has not been demonstrated convincingly [75]. The “hygiene hypothesis” proposes that early-life infections and exposure to bacterial products such as endotoxin may activate the T helper cell 1 (T_H1) immune response pathway and inhibit the development of the T_H2 responses involved in allergy [76,77]. This hypothesis may explain the asthma prevalence patterns observed in many parts of the world, with lower prevalence among poor, rural

populations and higher prevalence among wealthier, urban populations. In the United States, however, higher asthma prevalence and severity are seen among inner-city children, who do not necessarily experience fewer infections than their counterparts from other socioeconomic groups. In contrast, these children are less likely to have access to health care, which results in less antibiotic use and lower vaccination rates [78]. The hygiene hypothesis does not seem to explain the excess asthma morbidity of inner-city populations in the United States.

Indoor and outdoor air pollution and inner-city asthma

Environmental tobacco smoke

Passive exposure to environmental tobacco smoke (ETS) is more common in low-income, urban communities than in other demographic groups. Although the prevalence of cigarette smoking has declined in the United States during the past decade, its prevalence remains high in inner-city areas [79]. Knowledge about the adverse health effects of tobacco smoke is relatively low among poor and less well-educated people [80,81]. Higher levels of smoking in the inner city likely contribute to asthma morbidity in poor neighborhoods.

Environmental tobacco smoke seems to be a risk factor for the development of asthma in early childhood and an aggravating factor that increases morbidity among children who already have asthma. Prospective cohort studies have demonstrated a dose-response relationship between the amount of ETS exposure and the risk for asthma and wheezing in early childhood [82,83]. Maternal smoking during pregnancy (prenatal exposure) seems to be an even stronger risk factor for wheezing and early-childhood asthma than postnatal ETS exposure [84,85].

Despite the evidence that ETS may be a risk factor for early-childhood asthma, it may have a more important role as a trigger to future asthma attacks. An extensive scientific review published in 1992 by the US Environmental Protection Agency concluded that there was strong data to show that parental smoking aggravates childhood asthma [86]. A more recent comprehensive meta-analysis [87] confirmed that ETS exposure from parental smoking is associated with increased severe disease among children with established asthma. This meta-analysis also concluded that parental smoking is a risk factor for the development of nonallergic, wheezy bronchitis in early childhood, but it did not support ETS exposure as a risk factor for allergic asthma that persists into later childhood [87]. Other studies also showed that childhood ETS exposure did not seem to be a risk factor for allergic sensitization [88]. This finding suggests that ETS may have a greater interaction with the airways rather than with the immune system.

Outdoor air pollution

Although outdoor air pollution has been associated with reduced pulmonary function [81,89,90] and with an increase in emergency room visits [91] and hospitalizations [92,93] for asthma, there is little published evidence that ambient

levels of air pollution are higher in inner-city areas than in nearby, more affluent communities. Outdoor exposures that may aggravate asthma include *Alternaria* spores, ozone [94], and fine particles [95].

Diesel fuel particles are an outdoor pollution that seems to be specific to inner cities. Many inner-city neighborhoods are dependent on buses for transportation to work and school, and most of these buses run on diesel fuel. Many inner-city and poor neighborhoods disproportionately house and use buses. In New York, six of the seven bus terminals in Manhattan are housed above 125th Street, and over 3000 buses are housed in Harlem [96].

Laboratory research has shown alarming connections between diesel fuel particles and immune-system modification [97–100]. Studies have shown that diesel exhaust particles increase IgE production, the hallmark of allergic disease that is linked with asthma severity [97]. Other studies have shown that direct inhalation of diesel exhaust particles in mice caused an increase in inflammation and hyperresponsiveness of the airways with exposure to the allergen ovalbumin [98]. In vitro studies suggest that bronchial epithelial cells from atopic, asthmatic patients react to diesel exhaust particles differently than do cells from non-asthmatic subjects, with more exaggerated release of inflammatory mediators; however, no difference was seen in the amount of ciliary activity between asthmatic and nonasthmatic bronchial cells [99]. In vivo studies in humans who were sensitized to ragweed showed excess allergic response to ragweed allergens when exposure was accompanied by diesel exhaust particles [100]. These data suggest that exposure to diesel exhaust particles can increase immune-system response when sensitized individuals are exposed to allergens. Diesel exposure may cause sensitized individuals to respond to allergens at lower levels. Given the disproportionate amount of diesel exhaust exposure in inner cities, this finding provides a possible explanation for the increased prevalence and severity of asthma in this population.

Other contributing factors

In the United States, asthma is one of the most common chronic diseases of childhood across all socioeconomic classes; however, socioeconomic class and race affect the rates of asthma prevalence, asthma severity, and asthma mortality in low-income, urban communities. Blacks are three times more likely to be hospitalized and four times more likely to die from asthma than are whites [54,101–107]. Data from the National Health and Nutrition Examination Survey (NHANES) and Hispanic Health and Nutrition Examination Survey (HHANES) studies reveal that Hispanic children of Puerto Rican ancestry reported a prevalence of asthma that was nearly four times that of white children, whereas Mexican-American children had a prevalence that is similar to whites. The prevalence in black children was about 3.4 times that of whites [108].

Asthma severity varies among different racial groups. In New York, the average annual rate of asthma hospitalization was higher among Hispanics than

among all other racial categories: 62.9 per 10,000 among Hispanics; 59.9 among blacks; 12.2 among whites [109]. Hispanic residents of New York also had an asthma mortality rate (1.3 per 100,000) that was more than three times higher than that of non-Hispanic whites (0.4 per 100,000) [109].

In general, morbidity from asthma is higher in urban populations. In urban settings, this increase in asthma morbidity among black children parallels poor outcomes from other health problems [11,102,110–115]. Increased asthma severity and mortality in inner cities also is caused by the absence of regular primary care [11,116–118]. Urban children experience more difficulty in managing their asthma for a variety of reasons, including lack of health insurance, limited access to high-quality primary health care, the stresses of living in an urban area, lack of family and community support, and lack of knowledge of asthma and its treatment [111,112,119]. Inner-city communities with high rates of asthma morbidity have been shown to have relatively low use of anti-inflammatory controller medications [13]. In examining patterns of health care, health care use, and other asthma-related health behaviors among inner-city asthmatic children and their families, the NCICAS found that caregivers often had limited problem-solving skills in relation to asthma and had a high degree of psychologic distress [120]. Caretaker mental health problems and behavior problems of the child were associated with increased asthma morbidity [121].

Even with access to health care, differences in use of health care resources are likely to cause high rates of asthma hospitalization in inner cities. Asthma hospitalization is potentially preventable with appropriate outpatient management; however, there are multiple barriers to the effective use of outpatient services among poor, inner-city residents [102,121], including financial barriers that may prevent asthmatics from seeking care except during severe crises, from purchasing medications, and from implementing environmental control measures. Language and educational barriers may impair the ability to understand medical advice and learn appropriate self-management skills. These factors may act together to reduce the use of asthma medications, especially the use of chronic anti-inflammatory medication. Because inhaled anti-inflammatory medication provides no immediate symptomatic benefit, the rationale for its use is more difficult for patients to understand. Without good preventive medications and environmental controls, unnecessary asthma hospitalizations often occur.

Stress

Among inner-city children with asthma, parental psychosocial problems are associated with increased asthma morbidity [121]. Although psychosocial dysfunction impacts the care of the child and use of health care, there is growing evidence that psychologic stress may have neuroendocrine and immunologic effects. These effects may increase a child's risk for developing asthma and allergy and may aggravate asthma once it has developed. Birth-cohort data have revealed that family stress in early childhood is associated with increased allergen-induced lymphocyte proliferation [122]. In another cohort study, Klinnert and co-workers

[123] observed that children whose parents had parenting difficulties were more likely to develop asthma by 6 years of age. It has been proposed that stress may have immune-system effects that promote activation of T_H2 pathways, thereby leading to allergic sensitization and asthma [124]. This risk factor could be potent in inner-city children, who face the combination of stress, passive exposure to ETS, and a high degree of exposure to indoor allergens, such as cockroach allergens.

Summary

The exact causes of the excess asthma morbidity in inner-city communities and increasing asthma morbidity and prevalence over time are uncertain; multiple factors undoubtedly contribute to these epidemiologic patterns. Potential contributing factors can be classified broadly as health care related, environmental, socioeconomic, and genetic. Factors that aggravate existing asthma may be risk factors for the de novo development of asthma. Many of these factors may act together to induce development of asthma instead of acting alone.

Some studies have implicated the indoor environment as the cause of asthma in over half of all asthma cases [125]. Environmental problems in inner cities, including higher rates of cockroach infestation, older housing stock, and more outdoor diesel pollution, may explain part of this area's increased risk for asthma. Excess asthma morbidity in low-income, urban communities remains a major public health problem in the United States despite currently available asthma medications, widely disseminated asthma care guidelines, asthma education and case-management programs, and growing knowledge about the environmental factors that aggravate asthma. Avoiding the multiple indoor allergens and other potentially harmful environmental factors that aggravate asthma often is difficult and not feasible for inner-city asthmatics. In light of the enormous burden of asthma in the inner-city community, new research must focus on reducing these environmental exposures and modifying the harmful effects of exposures that cannot be avoided in this high-risk population.

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