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# RISK FACTORS FOR INNER CITY ASTHMA PATIENTS

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**Abstract**

Asthma patients in inner cities have the highest prevalence of asthma and the highest death rates in America. The purpose of this study was to evaluate sensitization and exposure to common indoor allergens seen for asthma treatment at Grady Memorial Hospital, Atlanta, Ga. Eighty asthma patients were enrolled in the emergency department and 64 in hospital clinics in this study. Neither sensitization nor visibility In this population, cat allergens are prevalent. The findings show that black asthma patients are exposed to high levels of mite and cockroach allergens in downtown Atlanta and that a high percentage of patients with asthma are sensitized to these allergens; a significant risk factor for asthma in this population is the combination of sensitization and exposure.

**Keywords:** asthma; mechanical ventilation, status asthmaticus, Pathophysiology epidemiology mechanisms.

## Introduction

Hospital admission rates for patients with asthma as well as the asthma mortality rate among the black population, particularly among patients in the inner city, are rising. Studies have concentrated on the growing morbidity of asthma, Overuse of beta-agonist for reasons such as air pollution, medication, health care inadequacy, and inflammation inhaled allergens after exposure [1]. There is also a high incidence of dust mite sensitization in patients from many countries that have asthma. Confirmed in a longitudinal sample, the sensitization of mites is a significant risk factor for asthma and indicated that sensitivity of babies to elevated mite allergen levels could contribute to earlier onset of asthma. In terms of atmosphere and living environments, the degree and form of allergens present in houses in the United States differ greatly. Dust mite is the primary indoor allergen in New Orleans and is present at elevated levels during the year. Houses in the inner city of Wilmington had elevated mite and cockroach levels with low cat allergen levels in the sample of adult patients, while dust mite and cat allergen levels were elevated in the suburbs and cockroach levels were low. Sensitization or sensitivity data are currently limited. It has been shown that allergens by asthma patients of the inner cities of the United States has increased [2]. Our key aim was to compare the amounts of allergens in soil. And the basic serum levels of IgE antibodies in asthma patients with and monitor to decide whether these lifestyle factors for asthma in patients from the inner city is. Furthermore, we used a questionnaire to test other questions and factors which contribute.

## Material and Methods

From the human inquiry committee at Grady Memorial Hospital, Atlanta, Ga. we gained consent. Between 1985 and 1985, a total of 144 asthma patients were enrolled in the research. 1990-1990. All of the Asthma patients came from black families, in keeping with the normal population seen at this hospital. During the fall of 1990, forty patients seen with acute wheezing and 40 patients seen with any other diagnosis were registered in the pediatric emergency room. All patients who agreed to be studied were enrolled, history and physical examinations were used to diagnose asthma. Furthermore, between 1985 and 1987, 41 patients from the Grady Memorial Hospital asthma clinic and 23

patients who were control subjects from the surgery clinic were admitted. Blood was collected from the infant after informed consent was granted and specifics of accommodation and socioeconomic data were gathered from a parent by questionnaire. Asthma, which had been already diagnosed by a psychiatrist, was also found in six of the control patients. The serum was preserved until measured at -20 ~ C.

**Home visits and assays for dust.**

House dust was collected for a total of 2 minutes from 57 homes by vacuuming each site with a hand-held cleaner. Every household has a bedroom. Sample collected, most of which also had a living room and a kitchen sample collected. Dust from the bedroom was obtained from mattresses, pillows, rugs, and any existing furniture. For the living room samples, rugs and furniture were vacuumed; floors, cabinets, and countertops were vacuumed for the Dust from the kitchen. At -20 ~ C, dust was stored until tested. Each dust sample was sieved and 100 mg was collected overnight at 4 ~ C in 2 ml of borate buffered saline solution. Group 1 of dust mite allergens, Bla g II of German were each measured separately in the dust extracts by using a two-site monoclonal antibody-based ELISA as previously described. Twenty six households of white patients from suburban Atlanta who were seen with asthma at Eggleston hospital had dust gathered for cockroach allergen level determination as an additional monitor [3-5].

**Immnnoassays for total IgE and specific IgE Antibodies**

With a two-site ELISA, complete serum IgE was assessed. In microtiter plates (Removawell; Dynatech Laboratories, Inc., Chantilly, Va.), the monoclonal anti-IgE antibody (CIA/7.12 and CIA/E4.15, kindly provided by Dr. Andrew Saxon, University of California, Los Angeles) was applied to the unknown or standard, and the bound IgE was detected with an enzyme-linked colorimetric change. Quantitative RAST with allergen attached to cyanogen bromide-activated cellulose disks (catalog No. 541, 0.6 em in diameter, Whatman, Inc., Clifton, N.J.) was used to calculate the unique IgE antibody. Allergen extracts were thoroughly dialyzed (Miles Inc., Hollister-Stier Division, Spokane, Wash.) and were unified by major measurement measurements Allergenic medications [6]. The dust mites Dermatophagoides farinae and Dermatophagoides pteronyssinus, cat epitherlium, short ragweed pollen, ryegrass pollen, and mixed cockroach extract were allergens used with ~0.1 to 0.5 #g per disk of main allergen (i.e. Der p I, Der f I, Fel d I, Amb an I, or Lolp I). With D, a regular curve was used in each assay. Farinae disks and serial repeat serum dilutions containing 2500 RAST units of IgE to D antibody. Farina. Farinae. Fifty percent horse serum was used as a context reduction diluent. The RAST units for specific IgE were previously defined and standardized by reference to a serum pool developed at the National Institute of Biological Standards and Regulation, London, England (NIBSC No. 82/528) containing 1,800 RAST units of D-D antibody IgE. Furthermore, three negative serums and three positive serums were operated. For each assay, in parallel. In the two assays, direct comparison of the antiIgE antibody bound indicated that the IgE antibody unit in the RAST was equal to ~0.1 ng of IgE [7].

**Table I.** Total IgE in 81 children with asthma and 63 control subjects seen at Grady Hospital, 1985 through 1987 and 1990

| Total IgE (IU/ml) | Asthma group (n = 81) | | Control group (n = 63) | |
|---|---|---|---|---|
| | No. | % | No. | % |
| <40 | 5 | 6 | 26 | 41 |
| ≥40 <199 | 29 | 36 | 25 | 40 |
| ≥200 | 47 | 58* | 12 | 19* |

*$p$ <0.001.

**Statistical Analysis**

The prevalence of igE antibodies was correlated with the chi-square test in groups of patients and in control subjects. As defined by Schlesselman, the odds ratio that reflects the odds that the disease will occur in exposed individuals compared to the odds that the disease will occur in unexposed individuals was determined [8].

**Results**

**Total IgE and specific IgE antibodies.** The geometric average for overall IgE was 270 IU/ml among the patients with asthma seen at Grady Hospital, while the comparable figure was 60 IU/ml for control subjects. 58% of the asthma patients had a total IgE of <200 IU/ml; 81% of the control subjects had a total IgE of <200 IU/ml (Table I). In both serum groups, specific IgE antibodies to dust mites, cockroaches and cat allergens were measured (i.e., from 1985 through 1987 and from 1990) [9]. As a group, the outcomes for the indoor allergens showed similar Results for both parts of the study indicated that >200 RAST units had the highest specificity level for the separation of asthma patients from control subjects (Table II). Forty percent of patients with asthma (32/81) had IgE dust mite antibodies alone, and another 25 percent had dust mite and cockroach IgE antibodies. Cat sensitivity was present in three asthma-positive patients and two control subjects, but the patients were also sensitive to one of the other indoor allergens in each case. Therefore, cat sensitivity was uncommon. Since allergic patients were not excluded from the control group, this group included some highly allergic children; 12 out of 63 control patients had a total IgE level of >200 IU/ml and 17 had >200 IgE antibody RAST units [10].

**Table II.** IgE antibody to any of three indoor allergens in 1990 and 1985 through 1987

| IgE (RAST units/ml) | Asthma group A (n = 40) No. | % | Asthma group B (n = 41) No. | % | Control group A (n = 40) No. | % | Control group B (n = 23) No. | % |
|---|---|---|---|---|---|---|---|---|
| <40 | 8 | 20 | 11 | 27 | 22 | 55 | 12 | 52 |
| 40-199 | 3 | 7 | 3 | 7 | 7 | 18 | 5 | 22 |
| ≥200 | 29 | 73 | 27 | 66 | 11 | 27 | 6 | 26 |

*A*, 1990 assessments; *B*, 1985–1987 assessments.

**Allergen levels in house dust.** The homes of 57 patients were got to visit and their house dust was tested for allergens of dust mite, cockroach and cat; the results are expressed as micrograms per gram of sieved dust for allergens of cat and dust mite and as units per gram for allergens of cockroach (Figure) [11]. For patients with asthma and control subjects, the percentage of homes with high levels of each allergen (and, in particular, the Mlergen cockroach) was very similar. There was a cat in the house for none of the patients or control subjects, and only two houses had >8 ug Fel d I/gin dust. Some patients did not have dust samples taken because they did not agree to visits or it was not safe to visit their neighborhoods [12]. Dust was also available at Eggleston Hospital from the homes of 26 patients seen for asthma in the pediatric pulmonary clinic. None of those samples (n = 85) had significant cockroach allergen (0/85 >2 units Bla g II per gram of dust).

**Sensitization and exposure,** There was both sensitization and significant exposure to at least one of the three indoor allergens in 21 out of 35 asthma patients and in only 3 out of 22 control subjects (Table III); this combination yielded a 9.5 odds ratio (confidence interval, 2.0 to 34) for asthma patients compared to control subjects. Those with sensitization in this sample of inner-city patients exposure and exposure were about nine times more likely to without this combination, there was wheezing as patients were [13].

**Table III.** Subjects with both sensitivity to indoor allergens (IgE antibody ≥200 RAST units/ml) and significant exposure at home to the relevant allergen

| | Mite | Cat | Cockroach | Any indoor allergen |
|---|---|---|---|---|
| Asthma group (n = 35) | 14 | 1 | 9 | 21* |
| Control group (n = 22) | 3 | 0 | 1 | 3* |

*$p$ <0.001.

**Questionnaire;** Medicines taken from the emergency department questionnaire were predominantly beta agonists and theophylline (64 percent and 33 percent , respectively). Inhaled steroid therapy was received by one patient, and cromolyn was received by one. Twenty-eight percent of the patients received no medication at all, and only eight percent of the patients received a new asthma diagnosis. Approximately half of the asthma patients enrolled had at least one household relative who smoked, but this was similar for patients with asthma (19/37) and control subjects (19/36) [14].

**Discussion**

Our research showed that 86% of the sampled inner city houses had significant concentrations of either mite or cockroach allergens [15]. Therefore, most patients with asthma and most control patients lived in homes that had previously recognized allergen levels as high enough to induce sensitization. Given the high average humidity levels in Atlanta, the use of wall-to-wall carpeting in government-subsidized housing, and the lack of a vacuum cleaner in many of these dwellings, the high levels of dust mite allergens found are not surprising. Previous suburban Atlanta data showed a very high content of dust mite allergens in carpets, sofas, mattresses and pillows [16]. Cockroaches were mainly observed in the kitchen area, and the highest levels of allergens were found in kitchen samples, but high levels were also observed in other rooms. Because none of the families said they had an indoor cat, it was not surprising to find a low prevalence of cat allergens. Two houses were found to contain significant cat allergens, which may have remained from a previous occupant or resulted from passive clothing transfer [17]. The low prevalence of cat allergen sensitization in these patients indicates that the levels found were not generally sufficient to trigger sensitization. IgE antibodies to ragweed and ryegrass were found in sera in some of the patients seen in spring and early summer. Sensitization to non-cockroach fungi, rodents, or insects may also have been present in these children, but we focused on those indoor allergens for which precise tests are available. Our findings support the results of previous studies showing a correlation between dust mite and cockroach allergens at more than threshold levels at home and asthma in patients sensitized to one or both of these allergens. Data from outside the United States indicate that a reduction in asthma symptoms occurred when the level of dust mite allergens was significantly reduced.

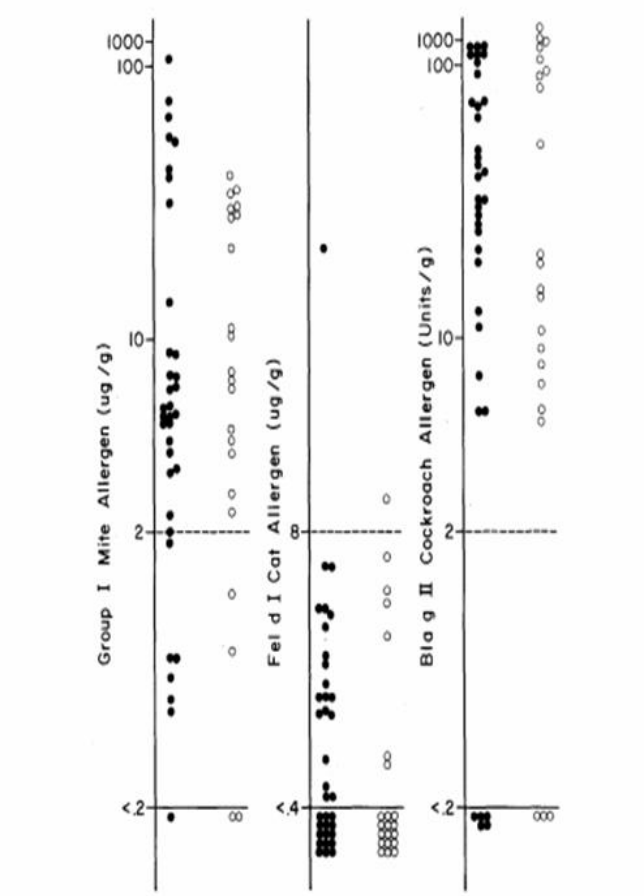


**Figure.** Dust mite (group I), cat (*Fel d* I), and cockroach (*Blo g* II) allergen levels in dust collected from the homes of 35 children with asthma (●) and 22 control subjects (○). *Dashed lines* indicate proposed threshold levels for each allergen; also indicated are the lower levels of sensitivity for each assay *(solid lines)*.

Our data shows that it will take several changes to control asthma morbidity and mortality rates among patients in inner cities. Before admission to the emergency department, many of the patients had inadequate health care. In addition to the use of action plans to respond to increased symptoms and to increased use of anti-inflammatory drugs (e.g., cromolyn sodium and inhaled steroids), adequate control of the disease is likely to require action plans to reduce exposure to allergens. Patients and their parents need to be given specific advice on the role sensitization plays in the disease and on approaches to exposure reduction. Further research is needed to identify effective regimens for reducing exposure to both cockroach and dust mite allergens. Advice will need to be tailored to suit patients' socio-economic conditions, and controlled studies of avoidance measures should be carried out in this population [18,19].

We would like to thank the doctors and nurses of the Pediatric Emergency Department of Grady Memorial Hospital for their assistance and Mary Sawyer for allowing us access to patients in the emergency department under her supervision.

**Conclusion**

Asthma patients in inner cities have the greatest incidence of asthma and the highest death rates in the United States. High levels of mite and cockroach allergens are exposed to black patients in downtown Atlanta and a high number of patients with asthma are sensitized to these allergens; a mix of sensitization and susceptibility is a significant risk factor for asthma in this community.

**References**

1. Weiss KB, Wagener DK. Changing patterns of asthma mortality. JAMA 1990;264:1683-7.
2. Birkhead G, Attaway N J, Strunk RC, Townsend MC, Teutseh S. Investigation of a cluster of deaths of adolescents from asthma: evidence implicating inadequate treatment and poor patient adherence with medications. J Allergy Clin Immunol 1989;84:484-91.
3. Friday GA, Fireman P. Morbidity and mortality of asthma. Pediatr Clin North Am 1988;35:1149-62.
4. Sears MR, Taylor DR, Print CG. Regular inhaled beta-agonist treatment in bronchial asthma. Lancet 1990;336:1391-6.
5. O'Hollaren MT, Yunginger J, Offord KP, et al. Exposure to an aeroallergen as a possible precipitating factor in respiratory arrest in young patients with asthma. N Engl J Med 1991;324:359-63.
6. Platt-Mills TAE, De Week AL. Dust mite allergens and asthma: A worldwide problem. J Allergy Clin Immunol !989;83:416-27.
7. Voorhorst R, Spiekma FThM, Varekamp H, Leupen M J, Lyklema AW. The house dust mite (Dermatophygoides pteronyssinus) and the allergens it produces: identity with the house dust allergen. J Allergy 1967;39:325-38.
8. Sporik RB, Holgate ST, Platts-Mills TAE, Cogswell JJ. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. N Engl J Med 1990;323:502-7.
9. Luczynska CM, Arruda LK, Platts-Mills TAE, Miller JD, Lopez M, Chapman MD. A two-site monoclonal antibody ELISA for the quantification of the major Dermatophagoides spp. allergens, Der p I and Der f I. Immunol Methods 1989;118:227-35.
10. Pollart SM, Smith TF, Morris EC, Gelber LE, Platts-Mills TAE, Chapman MD. Environmental exposure to cockroach allergens: analysis with monoclonal antibody-based enzyme immunoassays. J Allergy Clin Immunol 1991;87:505-10.
11. Chapman MD, Aalberse RC, Brown M J, Platts-Mills TAE. Monoclonal antibodies to the major feline allergen Fel d I: single-step affinity purification of Fel d I, N-terminal sequence analysis, and development of a sensitive two-site immunoassay to assess Eel d I exposure. J Immunol 1988;140:812-8.
12. De Blay F, Chapman MD, Platts-Mills TAE. Airborne cat allergen (Fel d I): environmental control with the cat in situ. Am Rev Respir Dis 1991;143:1334-9.

13. Pollart SM, Reid M J, Fling JA, et al. Epidemiology of emergency room asthma in northern California: association of IgE antibody to ryegrass pollen. J Allergy Clin Immunol 1988; 82:32-4.
14. Smith M J, Disney ME, Williams JD, Goels ZA. Clinical significance of skin reactions to mite extracts in children with asthma. BMJ 1969;2:723-6.
15. Witt C, Stuckey MS, Woolcock A J, Dawkins RL. Positive allergy prick tests associated with bronchial histamine responsiveness in an unselected population. J Allergy Clin Immunol 1986;77:698-702.
16. Dodge R, Cline MG, Burrows B. Comparisons of asthma, emphysema, and chronic bronchitis: diagnosis on a general population sample. Am Rev Respir Dis 1986;133:981-6.
17. Schlesselman JJ. Case control studies. Oxford: Oxford University Press, 1982:32-4.
18. Wood RA, Eggleston PA, Lind P, et al. Antigen analysis of household dust samples. Am Rev Respir Dis 1988;137:358-63.
19. Smith TF, Kelly LB, Heymann PW, Wilkins SR, Platts-Mills TAE. Natural exposure and serum antibodies to house dust mite of mite-allergic children with asthma in Atlanta. J Allergy Clin Immunol 1985;76:782-8.