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Atopic dermatitis and aeroallergen contact sensitivity

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Atopic dermatitis (AD) may be worsened by ingested foods or contact with irritants. We have identified 10 patients (six male and four female subjects, aged 1 to 54 years) with AD and contact sensitivity to a variety of aeroallergens. Marked improvement in skin symptomatology was noted when these patients were removed from their usual environment. The patients had markedly positive immediate wheal-and-flare reactions to a variety of aeroallergen extracts in response to prick tests and were subsequently patch tested on uninvolved skin with aeroallergen extracts (1:20 wt/vol, 50% glycerine) that elicited positive prick tests. Patch tests were applied for 48 hours, removed, and then were interpreted 24 hours later. Fifty percent glycerine was used as a negative control. Significant delayed cutaneous responses to a variety of aeroallergens were noted: house dust mite, tree, grass and weed pollens, animal danders, and various molds. Positive delayed cutaneous responses correlated strongly with aeroallergens identified in the patient's environment and/or suspected by the patients as provocateurs of their AD. Delayed cutaneous reactions were negative to aeroallergens not historically relevant to their AD. We conclude that aeroallergen contact may play an important role in selected patients with AD. The demonstration of immediate and delayed cutaneous responses in AD suggests both IgE and cell-mediated hypersensitivity as contributory mechanisms. (J ALLERGY CLIN IMMUNOL 1988;81:736-42.)

The pathogenesis of AD is unknown. Several mechanisms to explain the nature and etiology of this disorder have been proposed, including abnormal cutaneous autonomic vascular responses to physical and

Abbreviation used
AD: Atopic dermatitis

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chemical stimuli, enhanced β -adrenergic blockade, and a variety of immunologic abnormalities.¹ Included in this latter category are the findings of elevated levels of serum IgE that are occasionally related to the severity of the disease and suggest an allergic cause to the disorder.²⁻⁴ Numerous studies of sensitivity to com-

TABLE I. Demographics of patients with AD

Patient No.	Age/Sex (yr)	Associated diseases	TEC ³ *mm	IgE (IU/ml)
1	1/M	Sinusitis	246	183
2	1/M	Rhinitis	475	38
3	2/M	Rhinitis	589	2,931
4	6/M	Rhinitis/asthma	1981	1,402
5	7/F	Rhinitis	1950	4,350
6	24/F	None	225	125
7	27/F	Rhinitis/asthma	ND	ND
8	28/F	None	900	ND
9	30/M	Asthma	500	10,936
10	54/M	Rhinitis/asthma	890	158

ND = not done.

*Total eosinophil count.

mon aeroallergens as an etiology of AD appeared in the allergy and dermatology literature in the 1930s, 1940s, and 1950s.⁵⁻¹¹ Subsequently, the interest in aeroallergen contact or food as important in the etiology of AD waned. Major text books presented scant mention to allergic precipitants as important in AD. Recently, however, the role of immediate food hypersensitivity in the pathogenesis of childhood AD has been demonstrated by double-blind food challenges.^{12, 13} In addition, two studies^{14, 15} have demonstrated positive immediate prick tests and delayed hypersensitivity patch tests to aeroallergens in patients with AD. Chapman et al.¹⁶ have also demonstrated that patients with AD have increased specific IgE and IgG antibodies to antigen P₁ from *Dermatophagoides pteronyssinus* (house dust mite) and Rye 1 from grass pollen. The latter three studies support the view that specific sensitivities to common aeroallergens may be important etiologic factors in certain patients with AD.

In this article, we describe 10 selected patients with AD who demonstrated positive immediate prick test and delayed cutaneous patch test responses to aeroallergens on normal appearing, unmanipulated skin. Patch tests positivity correlated strongly with aeroallergens identified in the patient's environment and/or suspected by the patients as provocateurs of their AD. We believe these observations suggest that contact with aeroallergens may cause flares of AD in selected patients.

MATERIAL AND METHODS

Ten patients, six male and four female subjects, aged 1 to 54 years, evaluated at the National Jewish Center for Immunology and Respiratory Medicine, fulfilling the criteria of Hanifin and Lobitz¹⁷ for the diagnosis of AD, were identified. AD was moderate to severe in nature in that the patients required daily topical treatment and occasional oral corticosteroids. Complete history and physical examinations

were performed, paying particular attention to associated allergic disorders including asthma, allergic rhinitis, and sinusitis. Total IgE levels were obtained on serum samples by solid-phase ELISA (Abbott Laboratories, Chicago, Ill.). Total eosinophil counts were performed on whole blood samples (Unopette, Becton-Dickinson, Rutherford, N.J.).

All patients were prick tested with aeroallergens relevant to their usual environment. For these immediate hypersensitivity tests, we used aeroallergen extracts (Hollister-Stier, Spokane, Wash.) at 1:20 wt/vol in 50% glycerine. Tests were performed on patients' backs that were clinically free of eczema. Some patients were tested to materials from dusts within their home or occupational environments. The dust was extracted in a modification of Coca's extraction fluid (sodium chloride, 5 gm/L, and sodium bicarbonate, 5.4 gm/L). Extracts were made 1 gm in 10-ml extraction fluid (1:10 wt/vol). Material was extracted in a tumbler for 24 hours at 4° C to minimize bacterial growth. Material was then centrifuged gently to remove particulate matter and passed through a Millipore (Millipore Corp., Bedford, Mass.) filter. An equal volume of glycerine was added as a preservative. Wheal and erythema diameters were measured in millimeters and reported here as positive if the wheal diameter was 4 mm greater than that obtained with the 50% glycerine control. Aeroallergens that produced positive reactions on prick testing were selected for patch testing. These were performed without tape stripping on clinically uninvolved skin. Two drops (0.08 cc) of 1:20 wt/vol extracts were placed on 5-mm circular cotton gauze A1-Test patches (Sodertage, Sweden), occluded with 25 by 25-mm aluminum squares, and affixed to the skin with Scanpor porous tape (Norgesplaster, Norway) for 48 hours, removed and interpreted 24 hours later. Positive reactions produced at least erythema and papules or vesicles. Fifty percent glycerine was used as a negative control. Skin test observers were not aware of the patients' clinical history.

Thirty-two subjects (13 male and 19 female control patients) ranging in age from 5 to 58 years (mean 30 years) with seasonal or chronic rhinitis, but without histories of AD, received panels of 50 immediate prick tests and patch tests to Bermuda grass, timothy or rye grass, house dust mite (*Dermatophagoides farinae*), cat, and *Alternaria*.

TABLE II. Aeroallergens inciting AD

Patient No.	Historical precipitating factors	Positive prick test	Positive patch test	Environmental challenge
1	Patient's home	8/9 (89%) HD,* animals, molds	5/9 (62%) Rabbit, sheep, <i>Aspergillus</i> , <i>Cladosporium</i> (<i>Hor-modendrum</i>)	Patient's home (sheep hides, rabbits)
2	Patient's home	3/14 (21%) HD, cat, <i>Alternaria</i>	2/3 (67%) Cat, <i>Alternaria</i>	Patient's trailer home (cats in dirt crawl space, <i>Alternaria</i>)
3	Humid environment	1/17 (6%) HD mite	1/1 (100%) HD mite	Puerto Rico (humid)
4	Spring, dust	18/29 (62%) Trees, grasses, weeds, HD mite, cat	5/18 (28%) Grasses, HD	Haifa, Israel (humid)
5	Humid environment	4/27 (15%) HD, HD mite, Johnson, <i>Kochia</i>	1/4 (25%) HD mite	Hawaii (humid)
6	Dog contact	9/41 (22%) Dog, molds	1/9 (11%) Dog	Dog contact
7	Humid environment	5/9 (55%) HD, HD mite, cat, timothy, ragweed	1/5 (20%) HD mite	Pillow from Boston
8	Seattle, Wash., cat	15/36 (42%) Cat, trees, grasses, weeds, molds	7/16 (44%) Cat, trees, weeds	Furniture from Seattle in Denver, cat
9	Spring and fall	31/40 (78%) Trees, grasses, weeds, animals, HD mite, molds	2/21 (9%) Oak, ragweed	Oak- and ragweed-pollen season
10	Ivory Coast, Africa	6/41 (15%) Cat, HD mite, trees, <i>Alternaria</i>	1/6 (17%) HD mite	Car from Ivory Coast to Denver
Total		100/270 (37%)	26/91 (29%)	

*HD, house dust.

TABLE III. Patterns of aeroallergen skin testing

	Positive prick tests	Negative prick tests	Positive patch tests	Negative patch tests
Trees	26	50	3	17
Grasses	11	23	4	7
Weeds	22	47	4	14
Mites/dusts	15	5	6	8
Animals	13	5	6	6
Molds	13	40	3	13
Total	100	170	26	65

RESULTS

Characteristics of the 10 patients with AD are listed in Table I. Total circulating eosinophil counts and serum IgE levels were characteristically elevated. Table II lists the multiple factors that the patients or

their parents, identified as important precipitants of their eczema, the results of prick and patch testing, and features of the patients' local environment and home. A careful history was taken for contact with animals, pollens, dust, irritants, and for variations in

dermatitis with changes in climatic conditions and seasons. In most cases disease activity varied with changes in environment. Removal from their home or their humid local environment resulted in marked improvement of skin symptoms in many patients. This improvement occurred with nonintensive and often minimal changes in skin care. Additionally, many patients were able to associate contact with specific allergens and exacerbations of their AD, or noted seasonal variability in their disease severity. Hospitalization had little effect on mite-sensitive patients when they were hospitalized in a region known to contain a high mite density.

One hundred of 270 prick tests were positive (37%). A wide variety of aeroallergens produced positive reactions, including tree, grass, and weed pollens, house dust, house dust mite (*D. farinae*), animals, and molds. The percent positive reactions with pollens was 30%; mold, 20%, dust, mites, and animals, 75%. Ninety-one of 100 allergens producing positive prick tests were applied as patch tests, resulting in 26 positive reactions (29%). The positive delayed cutaneous responses by patch testing included tree, grass, and weed pollens, house dust, house dust mites (*D. farinae*), animals (cat, dog, rabbit, and sheep), and molds (Table III). Patch tests to trees, weeds, and molds produced positive reactions in 15% to 22% of tests applied, grasses, 36%; mites, 43%; and danders, 50%. An example of positive patch tests to house dust mite and three grasses obtained on patient No. 4 from Haifa, Israel, is illustrated in Fig. 1. The 50% glycerine and petrolatum controls are also presented. The papular reactions produced by the provocative contact challenges can be appreciated. The extension of poorly margined reactions beyond the 5-mm circular cotton gauze patches was typical and is probably secondary to perspiration causing antigens to leach out of the gauze and diffuse over the entire occluded area surrounding the gauze.

Positive patch tests correlated with aeroallergens identified in patients' environment or suspected by patients as provocateurs of their eczema. For example, patient No. 1 was a 1-year-old Wyoming boy whose parents operate a trading post in the home in which there were pet rabbits and sheep skins. Positive prick and patch tests to these allergens were identified. The parents refused to remove these allergens from the home, and the patient's eczema continues to flare at home but remits away from home. Patient No. 2 lived in a mobile home with a dirt crawl space and heating intake on the floor. A colony of cats inhabited the space, and an *Alternaria* species was cultured from Sabouraud's agar plates placed there. Positive prick and patch tests to these allergens were also found. The patient's eczema is consistently worse at home.



FIG. 1. Cutaneous reactions to five aeroallergens on the back of patient No. 4 at 72 hours after application of seven patches. Specific aeroallergens eliciting papular eczematous reactions are Johsson, Bermuda, and perennial rye grasses (top left to bottom left, respectively), and house dust mite (second from top right). Timothy grass elicited no reaction (top right) nor did 50% glycerine in petrolatum (lower right). Dermographic reactions from pen outlines are observed, surrounding the timothy grass, 50% glycerine, and petrolatum patch sites.

Patients Nos. 3, 4, and 5 from humid environments, known to contain high densities of house dust mite,¹⁸ revealed markedly positive prick and patch test responses to this allergen. Their AD resolved in Denver, whether in or out of the hospital, and worsened on return to their homes. Denver is known to be almost completely free of house dust mites.^{19, 20} The patient from Israel now lives in Denver, and his eczema is resolved. The patient from Hawaii now lives in southern California. Her eczema is easily controlled and limited only to the flexoral areas. Patient No. 7 had uncontrolled eczema in Boston that cleared in Denver, except from her face and neck. Questioning revealed that the only piece of furniture she brought from Denver to Boston was her pillow. Replacement of her pillow resulted in clearing of her skin. Mites can be transmitted in home furnishings.¹⁹ Positive prick and patch tests to mites were found. Patient No. 8 had flares of eczema on exposure to dogs. Positive prick

and patch test to dog extracts were demonstrated. Patient No. 9 has severe exacerbations of eczema in spring and fall, correlating with oak- and ragweed-pollen season, respectively. Positive prick and patch tests to these allergens were identified. Patient No. 10 had severe eczema in the Ivory Coast, which cleared in Maryland, until his car from Africa arrived in Maryland. Positive prick and patch test to house dust mite were found. Although many allergens produce markedly positive wheal-and-flare skin test reactions, allergens not relevant by history to the patient's AD did not produce positive patch test responses.

Nineteen of 32 control patients with rhinitis demonstrated positive immediate prick test to numerous common aeroallergens. All control patients (19 patients with positive prick tests and 13 patients with negative skin tests) were patch tested with five aeroallergens. Only one of the 160 patch tests applied on the 32 control patients was positive. The control subject with the positive patch test reacted to cat in the face of a negative prick test but a high continued exposure to the allergen (four cats at home).

DISCUSSION

We have identified 10 patients with severe AD who demonstrated immediate prick and delayed cutaneous patch tests to aeroallergens. It is important to stress that this is not an epidemiologic study of the prevalence of prick skin test or patch test reactivity in patients with AD, but rather an initial study suggesting that a subset of patients with AD may be triggered to flare in the presence of aeroallergens. The circumstantial evidence that aeroallergen contact might be an important etiologic factor in these patients is suggested by the patients' histories of worsening skin symptoms with allergen contact and marked improvement when they were removed from their usual environments. The improvement often occurred with minimal changes in skin care in or out of the hospital setting and has persisted for up to 3 years (patient No. 5) when patients avoided environments known to precipitate their disease state. Exacerbations invariably occur within 1 to 2 days when these patients return to their original environments. All patients have remained with their original families and thus the improvement is less likely attributable to making or breaking family ties.

Many of our patients demonstrated positive patch tests to house dust mites, which were believed to be significant provocateurs of their eczema. This was based on the observations that these patients had improvement of skin symptoms in semiarid Denver and/or exacerbations when they returned to humid

geographic areas or came in contact with furnishings brought from these humid environments. House dust mites are not found in significant numbers in Denver.^{19, 20} It is also known that mite prevalence is greater in more humid geographic areas than in dry ones^{21, 22} and that mite densities vary in relation to the relative ambient humidity.^{23, 24} Therefore, although we did not document increased densities of mites in these patients' homes or furnishings, we believe it is reasonable to infer, based on previous studies, that house dust mites were likely present.

Although these patients had positive prick tests to numerous aeroallergens, only aeroallergens that precipitated dermatitis by histories, or were identified in their home environments, elicited positive patch test reactions. Aeroallergen patch tests that were negative were not implicated as precipitating factors by history. In this manner, the patch test may be viewed as a direct provocative allergen challenge of the skin in patients with AD, much as inhalation bronchial challenge is used in allergic asthma. However, some aeroallergens, implicated by history as inducing eczema, elicited negative patch tests. These data suggest that the patch test technique described in this study is highly specific but may not be highly sensitive. The specificity of the patch test in this setting was further demonstrated by the fact that individuals with respiratory allergy but no dermatitis did not have positive patch testing to the same allergens that elicited markedly positive skin tests.

We cannot exclude the possibility that these flares of eczema might be secondary to inhalation of aeroallergens.^{10, 11} Although sensitization to these allergens may have occurred by inhalation, we would speculate that ongoing dermatitis is more likely due to direct skin contact. The observation that patients with AD demonstrated immediate and delayed cutaneous responses to aeroallergens suggests that both IgE-mediated immunity and cell-mediated immunity are important pathogenic mechanisms in this disease.

Recently, Mitchell et al.¹⁴ have demonstrated immediate prick tests and delayed hypersensitivity patch tests on abraded skin to aeroallergens in 17 patients with AD. Biopsy specimens of these lesions revealed infiltration with basophils, eosinophils, and mononuclear cells and appeared similar to cutaneous basophil hypersensitivity in guinea pigs. Details of the patients' histories and the relationships of their skin symptoms and allergy contact were not provided. Four of six patients who were dust-mite allergic, but without dermatitis, also demonstrated reactivity on patch testing, although the responses were less than responses with eczema. Our results are in agreement with the study by Mitchell et al.,¹⁴ demonstrating that

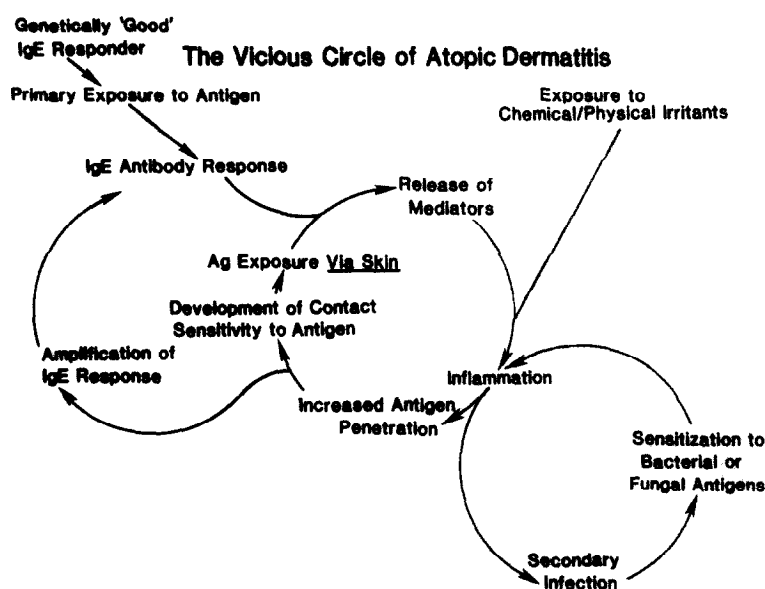


FIG. 2. Vicious cycle of AD.

patients with AD may have positive immediate prick and delayed cutaneous patch tests. However, we have extended their observations by demonstrating that the positive patch tests can be elicited on normal appearing, unmanipulated skin and are likely relevant to the disease process, since they correlated strongly with aeroallergen exposure. In addition, we did not observe positive reactions to patch tests in patients with respiratory allergy but without dermatitis. Since abrasion of the skin removes the upper layers of epidermis, the natural barrier to allergen penetration, and thereby enhances the likelihood of antigen presentation to immunoreactive cells, it is remarkable that our patients with AD produced positive patch tests to large protein molecules without skin abrasion. Reitamo et al.¹⁵ demonstrated six positive delayed hypersensitivity patch tests to birch pollen or house dust mite in 17 patients with AD. Their study used a 500-fold concentration of extract for patch testing as compared to the prick test standardized to one histamine-equivalent prick. Our findings extend their observations by demonstrating that contact sensitivity can be demonstrated to a wider variety of aeroallergens and correlates with a history of precipitating environmental factors. Additionally, our use of a much less concentrated extract for patch testing underlines the fact that these patients can be exquisitely sensitive to topically placed aeroallergens. Our results uniquely demonstrate the ability of aeroallergens in modest concentration to penetrate normal appearing skin and cause eczematous reactions as might likely occur with natural exposure. This suggests that part of the defect in AD may lie in an inherent abnormality in the epidermal permeability

barrier. Support for this possibility comes from a recent study that demonstrated patients with AD had increased transepidermal water loss through both dry and clinically normal skin.²⁵

It has been suggested that patients with AD have a variety of defects in antigen and nonspecific cell-mediated immune responses,¹ which would mitigate against the expression of cutaneous delayed hypersensitivity. For example, it was found that significantly fewer patients with AD had *Rhus oleoresin* sensitivity than normal control subjects.²⁶ Careful examination of the patient data revealed that 90% of the patients had active dermatitis at the time of patch testing. In subsequent studies, it has been demonstrated that nearly all immunologic abnormalities (both in vivo and in vitro) identified in patients with eczema returned to normal when the AD was controlled or resolved.^{27, 28} This suggests that many of the immunologic abnormalities associated with AD may be epiphenomena but nevertheless may be pathogenically important to the disease. It also suggests that patients with atopic dermatitis may express delayed cutaneous hypersensitivity responses, as was demonstrated in our patients. In fact, a recent large survey of patients with AD found that the prevalence of delayed hypersensitivity reactions to common contact chemicals was not significantly different from the prevalence in normal subjects.²⁹

Based on current information, we would propose a possible mechanism for the development and perpetuation of the lesions of AD (Fig. 2). Patients with an atopic genetic predisposition produce IgE antibodies after antigen exposure. Subsequent exposure

of "armed" mast cells to these allergens leads to mediator release and the inflammatory response. The skin inflammation results in a further increase of epidermal permeability, allowing increased allergen penetration, presentation to immunoreactive cells, and the development of contact sensitivity. A vicious cycle of dermatitis would ensue with further continued skin contact with allergen. This cycle of inflammation and sensitization is enhanced with exposure to chemical or physical irritants and secondary infection.

In conclusion, we suggest that aeroallergen contact may play an important role in selected patients with AD. Patch testing may be a useful provocative challenge to the skin in these patients. Last, the observation that patients with AD may demonstrate immediate and delayed cutaneous responses to aeroallergens suggest that IgE and cell-mediated immune responses are important pathogenic mechanisms in this disease. We are currently involved in prospective studies addressing the questions of the prevalence of aeroallergen contact sensitivity in patients with eczema, the time course of that sensitizing reaction, and the histology of the induced dermatitis.

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