was noted to be highly associated with this hypersensitivity reaction. The purpose of this study was to evaluate the effectiveness of prospective HLA-B*5701 screening to avoid these reactions.

STUDY POPULATION. A total of 1956 HIV-infected patients from 19 countries who had not previously received abacavir were enrolled.

METHODS. Individual patients were randomly assigned to undergo prospective HLA-B*5701 screening, with exclusion of previously known HLA-B*5701 positive patients from abacavir treatment, or to undergo a standard approach of abacavir use without screening. All subjects who started abacavir were observed for 6 weeks, the time frame in which a large majority of hypersensitivity reactions occur. The clinical diagnosis of hypersensitivity reaction to abacavir was assessed further by epicutaneous patch testing.

RESULTS. The prevalence of HLA-B*5701 was 5.76% (109 of 1956 subjects) of the subjects assigned to receive abacavir. Seventy-two percent were men, 84% were white, and 18% had not received antiretroviral therapy previously. Screening effectively eliminated patch-test–confirmed hypersensitivity. None of the subjects in the prospectively screened group had reactions, compared with 2.7% in the control group. This yielded a negative predictive value of 100% and a positive predictive value of 47.9%. Hypersensitivity reactions to antiretroviral therapy were clinically diagnosed in 93 patients, with a significantly lower incidence in the prospectively screened group (3.4%) than in the control group (7.8%) (P < .001).

CONCLUSIONS. HLA-B*5701 screening dramatically reduced the risk of immunologically confirmed hypersensitivity to abacavir. This pharmacogenetic test is useful for reducing the incidence of immunologically mediated hypersensitivity reactions to abacavir.

REVIEWER COMMENTS. This expansive study demonstrated the clinical usefulness of pharmacogenomics testing for immunologically mediated hypersensitivity to a particular drug. This is the first such demonstration for reactions to abacavir. This pharmacogenetic test is useful for reducing the incidence of immunologically mediated hypersensitivity reactions to abacavir.

ATOPIC DERMATITIS AND ALLERGIC SKIN DISEASE

Pollen Grains Induce a Rapid and Biphasic Eczematous Immune Response in Atopic Eczema Patients


PURPOSE OF THE STUDY. To investigate the relationship between eczematous reactions in subjects with immunoglobulin E–mediated allergy to pollen grains.

STUDY POPULATION. Two populations were evaluated: one population included subjects with atopic eczema who were sensitized to grass or birch pollen and who had a positive atopy patch test, and the second population included subjects without atopic eczema or atopy but who had a positive atopy patch test to nickel. In addition, as control populations, healthy volunteers and atopic subjects with eczema and sensitization to grass or birch but without a positive atopy patch test were included.

METHODS. Atopy patch tests with native pollen grains were performed to delineate the effect of native pollen grains on human skin of healthy and atopic individuals. A nickel patch test served as an established model of contact dermatitis. Skin-site samples were taken for biopsy 6 to 96 hours after allergen application and investigated immunohistochemically.

RESULTS. Histology of positive patch tests showed an influx of mononuclear cells (predominantly CD4+, CD25+, and CD45RO+). This influx was detected earlier in the pollen patch-test reactions compared with the immune response to nickel. A biphasic cytokine response could be detected in skin samples from the pollen patch test: interleukin 5 dominated in the early phase, and interferon γ dominated in the late phase. The nickel patch test was continuously dominated by interferon γ.

CONCLUSIONS. Pollen grains induce eczematous reactions in susceptible individuals. This reaction seems clinically and immunohistochemically similar to the contact hypersensitivity reaction to nickel but follows a faster kinetic and a biphasic course; T-helper 2 and immunoglobulin E in the early (24-hour) phase and T-helper 1 predominance in the late (96-hour) phase.

REVIEWER COMMENTS. This article highlights the importance of understanding that type 1 allergy-inducing antigens...
s such as pollens can exacerbate atopic eczema in susceptible individuals. Physicians should remember to discuss this with their atopic patients before the start of pollen season.

CONCLUSIONS. *S. aureus* is ubiquitous and was detected in dust samples from almost all homes regardless of disease state. However, house dust from participants with severe AD contained the most *S. aureus* DNA. The correlation between *S. aureus* DNA levels and AD severity is driven by proximity to the patient, as shown by the fact that the bed and bedroom floors from the patients with AD yielded the highest levels of *S. aureus* DNA. In the home and especially the bedroom, higher levels of *S. aureus* may contribute to disease severity and persistence in patients with AD.

REVIEWER COMMENTS. This is the first study to examine the environmental burden of *S. aureus* in the homes of patients with AD. This study concludes that patients with severe AD have higher environmental burdens of *S. aureus*. The source of the *S. aureus* may be shed bacteria from the skin of the patient with AD, and if live organisms persist in house dust, then they may be the source of recolonization of patients’ skin. However, to prove whether the relationship between high environmental load of *S. aureus* and severe AD is cause or effect, additional studies need to be performed, including quantification of bacterial load on the patient’s skin as correlated with home environmental burden and quantification of live bacteria in house dust.

**Severe Atopic Dermatitis Is Associated With a High Burden of Environmental *Staphylococcus aureus***


PURPOSE OF THE STUDY. It has been established that *Staphylococcus aureus* worsens atopic dermatitis (AD) by a variety of mechanisms. The purpose of this study was to quantify *S. aureus* burden in the homes of participants with AD of varying severity.

STUDY POPULATION. There were 62 volunteers aged 1 to 40 years. Participants were categorized as having mild (*n* = 18), moderate (*n* = 14), severe (*n* = 15), or no (*n* = 15) AD.

METHODS. Participants completed questionnaires about their asthma, allergies, and home environment. Patients with AD completed a Lung-Browder diagram, documenting the area and intensity of erythema, excoriation, papulation, and lichenification. From these diagrams, AD severity was calculated by using the Eczema Area and Severity Index (EASI). Subjects collected dust samples from their bed, the floor next to their bed, and the home vacuum bag and sent them to the laboratory for analysis. *S. aureus* DNA was extracted, and quantitative reverse-transcription polymerase chain reaction for the fem*B* gene (an *S. aureus*-specific genomic marker) was performed. Data were log-transformed and then analyzed with analysis of variance, student’s *t* test, and Spearman’s *r*.

RESULTS. Bed dust yielded the highest *S. aureus* concentrations. Participants with severe AD had significantly more *S. aureus* DNA (14.67 pg/mg dust) in bed dust than those with moderate (0.41 pg/mg dust; *P* < .0001), mild (1.42 pg/mg; *P* = .0051), and no (0.09 pg/mg; *P* < .0001) AD. The concentration of *S. aureus* DNA in bed dust strongly correlated with EASI scores. Similar patterns were observed for dust from bedroom floors for both DNA concentrations and EASI scores. The quantity of *S. aureus* DNA from the vacuum samples was significantly higher in participants with severe AD versus moderate, mild, and no AD. However, there was no correlation between EASI scores and concentrations of *S. aureus* DNA from vacuum dust samples.

**IgE Food Sensitization in Infants With Eczema Attending a Dermatology Clinic**


PURPOSE OF THE STUDY. Because community-based studies, which report immunoglobulin E food sensitization (IgE-FS) in >80% of infants with moderate atopic eczema, may be influenced by referral bias, the researchers assessed the prevalence of IgE-FS in a cohort of infants with moderate atopic eczema who were attending a dermatology department clinic.

STUDY POPULATION. Consecutive infants (*n* = 51 [39 boys]; median age: 34 weeks [range: 20–51 weeks]) with moderate atopic eczema severity were studied prospectively.

METHODS. Clinical history and eczema severity were documented. IgE-FS was assessed by the skin-prick test (SPT) (*n* = 51) and food-specific serum IgE antibody levels (CAP-FEIA test; *n* = 41). IgE-FS was diagnosed if the SPT or CAP-FEIA level exceeded the >95% predictive reference cutoff for positive food-challenge results.
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