aeroallergen avoidance. This study suggests that eczema severity, HDM-specific IgE, and CD30 are significantly decreased with occlusive bed covers, however, there was not a statistically significant difference between the study populations. Additionally, HDM allergen in this study was not common and may not be an important factor in the disease process of this population. Future studies should include participants with both sensitization and significant exposure.

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**MONTELUKAST IN THE TREATMENT OF CHILDREN WITH MODERATE-TO-SEVERE ATOPIC DERMATITIS: A PILOT STUDY**


**Purpose of the Study.** The role of leukotrienes in the pathogenesis of atopic dermatitis (AD) is uncertain. This double-blind, placebo-controlled crossover study addressed the efficacy of the leukotriene receptor antagonist montelukast in moderate to severe AD.

**Study Population.** Fifteen patients (ages 6–16) with moderate to severe AD despite conventional therapy consisting of at least a class II steroid, soap substitutes, and emollients.

**Methods.** Disease severity was evaluated by grading 8 areas of the body (head/neck, front of the trunk, the back, genitalia, and 4 limbs) on a scale of 0 to 3. A score of at least 40 was required to be enrolled in the study. The extent of disease was calculated by estimating the percentage of the body surface involvement. Patients were examined by the same physician on a biweekly basis and completed questionnaires to assess the impact of AD on daily life, as well as the effect of disease on relationships with family members and social life. There was a 2-week run-in period during which standardized topical treatment was initiated. Patients were randomized to receive either 5 mg montelukast or placebo daily for 4 weeks. There was a 2-week washout period before crossover for the second phase.

**Results.** Eleven patients completed the study with 6 in group A (placebo first) and 5 in group B (drug first). Despite randomization, the baseline median disease severity score between groups was significantly different, group A 52 and group B 78 ($P = .018$). Group B had a significant decrease in the disease severity ($P = .05$) during the drug phase. There was also an increase in disease severity during the placebo phase, however, severity scores did not return to baseline. Group A had improvement during both the placebo and drug phase ($P = .075$ and .029, respectively). Patient index scores and extent of disease did not change significantly for either group.

**Conclusion.** This pilot study shows that leukotrienes may be important mediators in AD and leukotriene receptor antagonists (LRAs) may be suitable adjuvant therapy in those patients with severe disease.

**Reviewers’ Comments.** Although leukotrienes are important chemical mediators in asthma and allergic rhinitis, their role in the pathogenesis of AD is not as clear. This study suggests that they may have a role in AD and that LRAs may provide some clinical benefit. In practice, some patients do seem to improve although the responses have not been overwhelming, which is consistent with the results of this study. It may be worth a try in patients with severe disease and, in addition, patients who are started on an LRA for their asthma may experience some improvement in their AD.


**Purpose of the Study.** To identify susceptibility loci for atopic dermatitis.

**Study Population.** A total of 839 individuals from 199 families in Germany, Italy, Sweden, and the Netherlands with moderate to severe atopic dermatitis with at least 2 affected siblings.

**Methods.** A genome-wide linkage study for atopic dermatitis and immune evidence of atopy (ie, serum evidence of either specific allergen sensitization by CAP-radioallergosorbent test or elevated immunoglobulin E levels).

**Results.** Chromosome 3q21 was strongly linked with atopic dermatitis and immune evidence of atopy.

**Conclusions.** Atopic dermatitis and immune evidence of atopy have been linked to the same locus on chromosome 3q21.

**Reviewers’ Comments.** Previous genetic studies of atopic diseases have revealed different chromosome linkages to a variety of atopic disease features. Candidate genes in chromosome 3q21 include costimulatory signals for T lymphocyte activation, CD80, and CD86. Therefore, the finding of a major susceptibility locus for atopic dermatitis on chromosome 3q21 supports a pivotal role of T cell costimulatory signals in mediating allergic inflammation. A larger genetic cohort study of atopic dermatitis in the future will reveal other important loci with affecting and contributing of the disease.

William Cookson and colleagues also recently published a genome screen for atopic dermatitis and have identified additional atopic dermatitis linkages with chromosoms 1q21, 17q25, 20p (Nature Genet. 2001;27:372–373). Interestingly, Cookson’s linkages are closely coincident with some major psoriasis loci previously reported. Furthermore, the 3q21 linkage to atopic dermatitis found by Lee has also been linked with psoriasis. Therefore, Lee’s and Cookson’s studies imply that similar genetic susceptibilities influence both atopic dermatitis and psoriasis.

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**DRUG ALLERGY**

**IMMEDIATE ALLERGIC REACTIONS TO CEPHALOSPORINS: CROSS-REACTIVITY AND SELECTIVE RESPONSES**


**Purpose of the Study.** Beta lactams are the most common agents involved in allergic reactions to antibiotics. There appears to be less cross-reactivity between penicillin and the newer generation cephalosporins. These latter agents might be associated with specific immunologic responses that do not cross-react with penicillins or other cephalosporins. These investigators examined the scope of immunoglobulin E (IgE) responses to various cephalosporins and penicillins in children and adults with histories compatible with cephalosporin allergy.

**Study Population.** Six children and 24 adults who had experienced urticaria/angioedema or anaphylaxis after ex-
A Major Susceptibility Locus for Atopic Dermatitis Maps to Chromosome 3Q21
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