DEVELOPMENT OF PEANUT ALLERGY IN FACTORS ASSOCIATED WITH THE
limited their use to research settings to date. Food and Drug Administration approval, their cost has
haps some of these asymptomatic atopic patients are evoking asthmatics. Although eNO devices have received
matics, but this study demonstrated an association between atopy and eNO levels. It has been suggested that
eNO may be a useful screening test for asthma, and perhaps some of these asymptomatic atopic patients are evolving asthmatics. Although eNO devices have received

FOOD ALLERGY

FACTORS ASSOCIATED WITH THE DEVELOPMENT OF PEANUT ALLERGY IN CHILDHOOD


Purpose of the Study. Because peanut allergy has increased in prevalence and is an important cause of life-threatening reactions, the authors sought to investigate possible determinants of peanut allergy.

Study Population. Data were obtained from the Avon Longitudinal Study of Parents and Children. This geographically defined cohort included 13,971 preschool-aged children. Forty-nine of those children had a history of peanut allergy. Thirty-six of those 49 underwent skin testing, and 29 demonstrated positive results. Peanut allergy was confirmed for 23 children with double-blind, placebo-controlled, food challenge.

Methods. Pregnant women were enrolled and questioned about their allergy history before delivery and were given serial questionnaires throughout their children’s infancy and childhood. The authors prospectively identified 49 children with a history of reactions to peanuts. Twenty-three children were then confirmed as being allergic to peanuts with skin testing and double-blind, placebo-controlled, food challenge. There were 2 control groups, including children with eczema in the first 6 months of life whose mothers also had eczema and 140 children without peanut allergy who were randomly selected from the cohort. Cord blood samples stored at birth were retrieved and analyzed for peanut-specific and total immunoglobulin E (IgE) for the children with peanut allergy. Retrospective data on maternal consumption of peanuts during pregnancy and lactation, family history of peanut allergy, and the use of specific lotions and creams (the interviewer was not aware of which products contained peanut oil) were then obtained.

Results. Peanut allergy was found to be independently associated with eczematous dermatitis (rash over joints and creases or oozing crusted rash) in the first 6 months of life, intake of soy products, family history of peanut allergy, and the use of skin preparations containing peanut oil. Neither maternal peanut consumption during pregnancy and lactation nor duration of breastfeeding was found to be associated with the development of peanut allergy. Additional evidence not supporting previous concepts of in utero sensitization came from undetectable peanut-specific IgE and normal total IgE levels in cord blood.

Conclusions. Sensitization to peanut antigens appeared to be through inflamed atopic skin, rather than via the gastrointestinal tract, possibly from the use of skin preparations with even trace amounts of peanut oil. With respect to the independent association between intake of soy products and peanut allergy, soy protein fractions have shown homology to major peanut proteins and cross-sensitization could result from exposure to a common T cell epitope.

EFFECT OF ANTI-IMMUNOGLOBULIN E THERAPY FOR PATIENTS WITH PEANUT ALLERGY


Purpose of the Study. To determine whether subcutaneous administration of a humanized anti-immunoglobulin E (IgE) antibody, TNX-901, raises the threshold of sensitivity to peanuts among patients with peanut allergy.

Study Population. Eighty-four patients between 12 and 60 years of age, with a history of allergic reactions to peanuts, total IgE levels between 30 and 1000 IU/mL, positive skin prick tests for peanuts, and documented reactions with formal peanut challenge at the start of the study, were studied.

Methods. A randomized, double-blind, placebo-controlled, dose-range study was performed. During the screening process, peanut allergy was confirmed and the threshold for reactivity was determined with a double-blind, placebo-controlled, oral food challenge with encapsulated peanut flour. Patients were subsequently randomized to receive subcutaneous injections of placebo or TNX-901 (150, 300, or 450 mg) at 4-week intervals, for a total of 4 doses. Two to 4 weeks after the final injection, a final peanut challenge was performed, to determine the threshold of reactivity to peanuts after the treatments. Serum samples were obtained at 4-week intervals, to monitor trough total IgE levels.

Results. From mean baseline thresholds of sensitivity of 178 to 436 mg of peanut flour in the various groups, the mean increases in the oral food challenge threshold were 710 mg in the placebo group, 913 mg in the group given 150 mg of TNX-901, 1650 mg in the group given 300 mg of TNX-901, and 2627 mg in the group given 450 mg of TNX-901 (P < .001 for comparison of the 450-mg dose with placebo; P < .001 for trend with increasing dose). Patients who received 450 mg had a mean threshold of reactivity of 2805 mg of peanut protein (equivalent to ~9 peanuts), compared with 178 mg (equivalent to one half of a peanut) before the injections.

Conclusions. Subcutaneous administration of TNX-901 increases the threshold of reactivity to peanuts in a dose-dependent manner, which may translate into protection against most accidental ingestions of peanuts.
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Jordan Scott and Lynda C. Schneider

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