history of penicillin (PCN) or cephalosporin allergy, and was <18 years of age. The mean ± SD age was 5 ± 3.5 years, and 47.1% were female.

METHODS. This was a retrospective cohort study from the Mayo Clinic (Rochester, MN) in which medical records were reviewed for basic demographics (age and gender), type of adverse reaction (ADR) to penicillin or cephalosporin, PST results, time from original PCN ADR to testing, and adverse reactions to PCN after negative PST. The χ² test was used to compare the differences in the proportion of children and adults with a positive PCN skin test. P < .05 was considered statistically significant.

RESULTS. Among the study participants, 703 had a negative PST (90.4%), 66 had a positive test (8.5%), and 9 had equivocal tests (1.1%). Three hundred sixty-nine of 703 patients with negative PST (52%) were challenged with PCN; 14 of these 369 (3.8%) had an adverse drug reaction. Of the 14 with reactions, 12 were isolated to the skin, and 2 children had serum sickness. There were no adverse reactions to PST.

CONCLUSIONS. This study confirms that penicillin skin testing was safe and effective in the evaluation of children with a history of PCN allergy.

REVIEWER COMMENTS. Multiple studies in the adult population have shown that the prevalence of ADR after negative PST is low; however, studies in the pediatric population were lacking. This study confirmed that previous suspicion or diagnosis of “penicillin allergy” should be evaluated, that the testing is safe, and that reactions after negative tests are uncommon and typically not anaphylaxis. Importantly, skin testing excluded serious allergy for the vast majority in this study. The study is a good reminder to consider consulting an allergist for an evaluation rather than exclude this class of medications based solely on the history.

Randomized Trial of Peanut Consumption in Infants at Risk for Peanut Allergy

PURPOSE. To evaluate strategies of early peanut consumption or avoidance for prevention of peanut allergy in patients at risk.

STUDY POPULATION. The participants were between 4 and 11 months of age at randomization. They suffered from severe eczema, egg allergy, or both.

METHODS. A total of 640 patients were evaluated over a 60-month period. They were stratified according to their sensitivity to skin testing to peanut extract. Those with no measureable wheal were evaluated as not sensitized, those with wheal diameters 1 to 4 mm were considered sensitized, and participants with >4-mm wheal were excluded. Participants were randomized to receive an initial supervised feeding of peanut or to avoid peanut. Those tolerating an initial peanut feeding were instructed on incorporating peanut into the diet. Participants underwent peanut feeding tests at 5 years of age.

RESULTS. Of the 530 infants in the intent-to-treat population who initially had negative skin tests, the prevalence of peanut allergy at 60 months was 13.7% in the avoidance group and 1.9% in the consumption group. Of the 98 participants in the group who initially had positive tests, the prevalence of peanut allergy was 35% in the avoidance group and 10% in the consumption group at 60 months. There was no significant difference in the incidence of serious adverse effects. Increase in levels of peanut-specific immunoglobulin (Ig)G4 antibody occurred predominantly in the consumption group, and a greater percentage of participants in the avoidance group had elevated titers of peanut-specific IgE antibody. A larger wheal at skin prick test and a lower ratio of peanut specific IgG4:IgE were associated with peanut allergy. In the intent-to-treat group, the overall reduction rate of peanut allergy from 17.2% in the peanut avoidance group to 3.2% in the peanut consumption group relates to ~80% relative risk reduction in developing peanut allergy and a 14% absolute risk reduction. This corresponds to a number needed to treat of 7.1. A total of 7 children reacted to peanut at baseline before randomization, and 9 children in the consumption group developed peanut allergy during the study.

CONCLUSIONS. The early introduction of peanuts in a high-risk group significantly decreased but did not nullify the chance of developing peanut allergy.

REVIEWER COMMENTS. This is a landmark study indicating that early highly regulated consumption of peanut in a high-risk group of patients will bring a significant reduction in the occurrence of peanut allergy. It does not completely obviate the possibility of developing peanut allergy. It should also be noted that these data may not be directly extrapolated to patients other than those fitting the criteria of the study. Caveats and cautions include the following: whether eating peanut on a different schedule/amount than the study would have different results, whether discontinuation of eating peanut could result in loss of tolerance, and the concern that many peanut products may be choking hazards for infants and young children, requiring careful instructions about specific foods to use. These data are of great interest.
and should be evaluated in each individual practice setting to determine if the adoption of this protocol is possible.

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Influence of Early-Life Exposures on Food Sensitization and Food Allergy in an Inner-City Birth Cohort

PURPOSE OF THE STUDY. To use clinical, serological, and environmental exposure data to evaluate the prevalence of food allergy (FA) and the contribution of environmental exposures on sensitization and allergy in a population of inner-city children.

STUDY POPULATION. The study enrolled 560 children from the Urban Environment and Childhood Asthma birth cohort (≥34 weeks’ gestation) with a parental history of allergic disease or asthma, cord blood sample, and food-specific immunoglobulin (Ig)E levels measured.

METHODS. Pregnant women were enrolled and their children followed from birth to age 5 years. Mothers completed prenatal environmental exposure questionnaires and yearly clinical history/exposure questionnaires, and children were seen annually for detailed history of symptoms and exposures. Serum IgE to milk, egg, and peanut was obtained at ages 1, 2, 3, and 5 years, and blood cytokines were obtained at birth, 1 and 3 years. Household dust samples were collected at age 3 months and annually for indoor allergens, endotoxin, and ergosterol. Children were grouped based on sensitization (IgE ≥0.35 kU/L to milk, egg, and/or peanut) including the following: (1) FA (sensitized on dietary avoidance and confirmed reaction to food), (2) possible FA (sensitization with either dietary avoidance or unknown consumption and no confirmed reaction), (3) sensitized but tolerant (consumed food without reaction), and (4) not sensitized.

RESULTS. Of those enrolled, 55.4% were in a food-sensitized group (47% milk, 31% egg, 21% peanut) and were classified as follows: 9.9% FA (2.7% milk, 4.3% egg, 6.0% peanut); 17% possible FA; 28.5% sensitized but tolerant. FA was associated with breastfeeding, recurrent wheeze, eczema, aeroallergen sensitization, male gender, and low environmental endotoxin exposure. Other environmental exposures were similar in groups with and without food sensitization/allergy. IgG4 levels were higher in sensitized and FA patients than in the nonsensitized group, and FA children had altered innate/adaptive immunity, per cytokine profiles.

CONCLUSIONS. This study found the incidence of FA in inner-city children to be higher than in the average pediatric population with potential environmental risk factors delineated.

REVIEWER COMMENTS. This study is the first to use prospective clinical and serological data along with environmental exposure history to demonstrate a high incidence of FA in inner-city children. The study was also among the first to look at longitudinal exposures common in an urban environment and their effect on development of FA and sensitization demonstrating that environmental endotoxin exposure was associated with a lower incidence of FA. This study leaves us with the opportunity for more directed research to discover modifiable risk factors associated with the development of FA and sensitization among high-risk urban populations.

Atopic Dermatitis Increases the Effect of Exposure to Peanut Antigen in Dust on Peanut Sensitization and Likely Peanut Allergy

PURPOSE OF THE STUDY. To evaluate whether environmental peanut exposure is a risk for peanut sensitization and allergy and whether this risk is modified by an impaired skin barrier.

STUDY POPULATION. Subjects were 359 atopic children, aged 3 to 15 months, who had been recruited to the National Institutes of Health-sponsored Consortium of Food Allergy Research (CoFAR) Observational Study and had dust samples available for analysis. Participants had a clinical history of cow’s milk and/or egg allergy with a positive skin prick test (SPT) to these allergens or had atopic dermatitis

METHODS. At recruitment into the CoFAR study, subjects were evaluated for history and severity of atopic dermatitis, peanut sensitization, and likely allergy based on peanut-specific immunoglobulin (Ig)E ≥5 kU/mL. Peanut protein in household dust (micrograms per gram) was measured.

RESULTS. An exposure-response relationship was observed between peanut protein in household dust and peanut skin prick sensitization and likely allergy based on peanut-specific IgE. The odds of peanut SPT sensitization were increased with an increase in 4 log2 environmental peanut exposure (EPE; 1.71-fold; 95% confidence interval [CI], 1.13- to 2.59-fold, P = .01) in the final multivariate