Peanut Protein in Household Dust Is Related to Household Peanut Consumptions and Is Biologically Active


**PURPOSE OF THE STUDY.** The goal of this study was to compare household peanut consumption (HPC) with the level of peanut in the home and to determine if ambient peanut in the home was biologically active.

**STUDY POPULATION.** Forty-five families were recruited from pediatric allergy clinics. Three peanut-allergic and 3 non-allergic children were recruited for basophil activation tests. Basophil activation tests were conducted by using extracted dust samples of low or high peanut protein content. Samples containing <0.375 ng/mL did not upregulate CD63 expression (a marker of basophil activation) in samples from peanut-allergic children. However, at higher peanut protein concentrations in extracted dust samples, there was a dose-dependent increase in CD63 expression.

**RESULTS.** Peanut consumption by each parent and their cumulative consumption correlated with levels of environmental peanut protein in the parental beds (mother: $r_s = 0.698$, $P < .001$; father: $r_s = 0.672$, $P < .001$). Only 6 infants were eating peanut, but their consumption correlated with levels in the play area and crib, and all levels of peanut protein were high ($>$10 μg/g of dust). HPC was most highly correlated with ambient peanut protein levels in the infants’ cribs and play areas ($r_s > 0.700$, $P < .001$ in all cases and over all durations of HPC measurements). Paternal consumption correlated with levels in the crib to a greater extent than did maternal consumption. Peanut protein was found in the cribs of infants who did not eat peanut. Levels were high when parental consumption was moderate to high even if the child did not eat peanut. HPC correlated with peanut protein levels from wipe samples from all sites. Basophil activation tests were conducted by using extracted dust samples of low or high peanut protein content. Samples containing <0.375 ng/mL did not upregulate CD63 expression (a marker of basophil activation) in samples from peanut-allergic children. However, at higher peanut protein concentrations in extracted dust samples, there was a dose-dependent increase in CD63 expression.

**CONCLUSIONS.** Peanut protein levels in homes correlate with HPC. Peanut protein in household dust is biologically active and is a possible route of sensitization.

REVIEWER COMMENTS. Should families with infants ban peanut from the home? I don’t know, and I wouldn’t venture a guess. Previous research by this group found that HPC was a risk factor for the development of peanut allergy. Conversely, with all the HPC in this study, only 6 infants had peanut allergy. Maybe there is a “window” of ambient peanut protein concentration that increases risk of peanut allergy and a different “window” that increases the likelihood of tolerance. As the authors very importantly note, the levels of household peanut protein found in this study might be enough to induce sensitization but are not likely to be high enough to trigger clinical reactions in sensitized individuals.
CONCLUSIONS. Placing a child on EHCF accelerates tolerance acquisition in children with cow’s milk allergy compared with other formula choices and is further improved with the addition of LGG.

REVIEWER COMMENTS. The results shown in this study with EHCF resulting in a greater rate of tolerance acquisition is likely due to the immunomodulatory effect induced by hydrolyzed casein peptides, as suggested from previous animal studies. This benefit will have to be factored in when specifically considering starting patients on EHCF versus AAF because some patients will react even to EHCF. The difference in the rate of tolerance acquisition between EHCF and AAF may be that those requiring AAF have a more severe form of milk sensitivity, thus requiring them to be on AAF initially.


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Sustained Unresponsiveness to Peanut in Subjects Who Have Completed Peanut Oral Immunotherapy


PURPOSE OF THE STUDY. The goal of this study was to determine if long-term treatment with oral immunotherapy (OIT) will result in sustained unresponsiveness to peanut.

STUDY POPULATION. The study enrolled 39 children, ages 1 to 16 years, with peanut allergy defined as a clinical history of a reaction to peanut within 60 minutes of ingestion, a positive peanut skin prick test (SPT) result ≥3 mm, and peanut-specific immunoglobulin (Ig)E ≥15 kU/L or with a positive SPT result and peanut IgE ≥7 kU/L and a clinical reaction within the previous 6 months. Subjects with poorly controlled or severe asthma and those with a history of severe anaphylaxis were excluded.

METHODS. Daily OIT was administered by using a standard protocol with initial dose escalation, build-up every 2 weeks to 4000 mg, and daily maintenance dosing for up to 5 years of therapy. Blinded oral food challenges (OFC) to 5000 mg of peanut were performed during and at the end of the study, including after 4 weeks of OIT cessation to assess sustained unresponsiveness. Peanut SPT, IgE testing, and immunologic assays were conducted throughout the study. A telephone survey was conducted at the end of the study to assess peanut consumption and symptoms noted.

RESULTS. Twenty-four of the 39 subjects completed the protocol. Six subjects withdrew due to allergic adverse effects, and 9 withdrew for other reasons. Twelve subjects demonstrated sustained unresponsiveness after the final OFC and were deemed treatment successes (TS) and were instructed to add peanut ad libitum to their diet; those not passing the final OFC were considered treatment failures (TF) and were instructed to continue dietary avoidance of peanuts. At baseline and final OFC, TS had smaller SPT results (P < .01) and lower peanut-specific IgE (P < .01), Ara h 1 (P < .05), and Ara h 2 (P < .01) levels plus a reduced peanut-specific IgE/total IgE ratio (P < .001) compared with TF. Peanut IgG4 levels did not differ between the 2 groups. Survey results showed that none of the TS reported allergic reactions to peanut compared with 14% of TF. TS consumed a median of 555 mg/d (0–4000 mg/d) of peanut 3 days per week (0–7 days/week).

CONCLUSIONS. This study is the first to show sustained unresponsiveness after peanut OIT noted in ~50% of subjects treated for up to 5 years, results that persisted after OIT. Smaller SPT results and lower peanut-specific IgE levels strongly correlated with success of treatment.

REVIEWER COMMENTS. Sustained unresponsiveness in peanut OIT is a vital step in the treatment of peanut food allergies. This advancement in OIT is associated with direct benefit that likely requires ongoing antigen exposure for persistence. Further study in larger populations is needed to advance OIT as a viable treatment option for widespread use.


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Assessing the Efficacy of Oral Immunotherapy for the Desensitisation of Peanut Allergy in Children (STOP II): A Phase 2 Randomised Controlled Trial


PURPOSE OF THE STUDY. The goal of this study was to establish the efficacy of oral immunotherapy (OIT) for the desensitization of children with peanut allergy.

STUDY POPULATION. Children ages 7 to 16 years with an immediate peanut hypersensitivity reaction and a positive result on skin prick test and double-blind, placebo-controlled food challenge (DBPCFC) to peanut were recruited from allergy clinics and national patient support groups.

METHODS. In phase 1 of this unmasked, randomized, placebo-controlled trial, children in the active OIT group received 2 to 800 mg/d of peanut protein, and those in the control group continued to avoid peanut. The primary outcome was desensitization, defined as passing a DBPCFC to 1400 mg of peanut protein after 6 months. During phase 2, control participants remaining allergic to peanut received OIT and had a repeat DBPCFC.
Formula Selection for Management of Children With Cow's Milk Allergy Influences the Rate of Acquisition of Tolerance: A Prospective Multicenter Study
Sandy Jung-Wu

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