(egg-containing products such as cakes or biscuits) was not. The lowest risk for egg allergy was found in infants introduced to cooked egg at 4 to 6 months (OR: 0.2 [95% CI: 0.06–0.71]; $P = .012$). There was no association of egg allergy with duration of breastfeeding (after adjustment for family and personal history of allergy) or age of introduction of other solid foods.

CONCLUSIONS. Introduction of cooked egg (boiled, scrambled, fried, or poached) at 4 to 6 months of age might protect against egg allergy irrespective of family or personal history of allergy. Duration of breastfeeding and age of introduction of other solids does not seem to affect development of egg allergy.

REVIEWER COMMENTS. In light of the changing perception that early instead of delayed exposure of commonly allergenic foods might lead to tolerance, this study is an important step in determining how the timing of introduction and form of food introduced (eg, cooked versus baked) might influence the development of food allergy. A large population was studied, and 75% of positive skin-prick-test results were confirmed with oral food challenges; however, egg-introduction history was retrospective and might have been subject to recall bias. The next step would be a prospective study on egg introduction to confirm these observations and to determine if the protective effect is limited only to egg or affects other food allergies such as those to peanut.

URL: www.pediatrics.org/cgi/doi/10.1542/peds.2011–2107V

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Early Exposure to Cow's Milk Protein Is Protective Against Immunoglobulin E (IgE)-Mediated Cow's Milk Protein Allergy

Katz Y, Rajuan N, Goldberg MR, et al. *J Allergy Clin Immunol.* 2010;126(1):77–82

PURPOSE OF THE STUDY. The investigators determined the prevalence of cow milk allergy, the cross-reactivity with soy, and risk factors for the development of cow milk allergy in a large-scale, population-based prospective study.

STUDY POPULATION. All infants (13 234) born from June 10, 2004, to June 30, 2006, at the Assaf-Harofeh Hospital in Zerifin, Israel, were eligible for enrollment. The feeding history was obtained for 98.4% (13 019) of these infants, mostly by telephone interview.

METHODS. In the newborn period, after routine anticipatory guidance, in which breastfeeding was encouraged and other alternative cow milk–based feeding programs were reviewed, parents were asked to either fill in a questionnaire or contact the allergy clinic immediately after any suspected adverse reaction to the initiation of cow milk–protein feeding. If no unusual event was noted, the families were asked to contact the allergy clinic 14 to 30 days after initiation of cow milk–based feeding. Any parents who noted a possible adverse reaction were interviewed by an investigator and invited for examination and testing. Final diagnosis of immunoglobulin E (IgE)-mediated cow milk–protein allergy was made independently by 2 investigators, and any disagreement (2 cases) was resolved with conjoint discussion. Skin-prick testing to cow's milk and soy was conducted, as were open cow milk challenges.

RESULTS. The cumulative incidence of IgE-mediated cow milk allergy was 0.5% (66 of 13 019). The mean age of cow milk introduction was significantly different ($P < .001$) between healthy infants (61.6 ± 92.5 days) and those with IgE-mediated cow milk allergy (116.1 ± 64.9 days). Only 0.05% of the infants who were started on regular cow milk–protein formula within the first 14 days versus 1.75% who were started on formula between the ages of 105 and 194 days had IgE-mediated cow milk allergy ($P < .001$). None of the patients with IgE-mediated cow milk allergy proved to have an IgE-mediated soy allergy.

CONCLUSIONS. In this patient population, IgE-mediated cow milk allergy is less prevalent than previously reported. Early exposure to cow milk protein seemed to be protective against cow milk allergy.

REVIEWER COMMENTS. The results of this study, as well as those of other recent investigations, go against the previous mantra that prolonged restriction of specific food allergens might be helpful in the prevention of food sensitivity in the early years. Early introduction of specific dietary proteins seems to lead to tolerance, although the exact timing and dose required have not been determined. It is remarkable that none of the subjects with IgE-mediated milk allergy proved to have soy allergy, contrary to a reported co-reactivity of 10% to 14%. Because the reported rate of IgE-mediated milk allergy is lower in this study population than has been reported previously, additional studies are required to confirm these findings.

URL: www.pediatrics.org/cgi/doi/10.1542/peds.2011–2107W

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Peanut Oil and Peanut Allergy, Foes or Folks?

Ho MH, Lee S, Wong WH, Lau Y. *Arch Dis Child.* 2011; 95(10):856–857

PURPOSE OF THE STUDY. Peanut allergy seems to be increasing among children in Hong Kong. The authors of this study report suggested that this increase might be a result of changes in edible oils. Crude peanut oil (protein content:

100–300 μg/mL) was "ubiquitous in maternal and infant diet in Hong Kong in the past" but has now largely been replaced by olive oil.

METHODS. The consumption of various oils was estimated from data on imports. Per-capita consumption was calculated on the basis of population over time.

RESULTS. Per capita consumption of crude peanut oil fell ~30-fold over the last 15 years, whereas consumption of olive oil increased ~30-fold over the same time period.

CONCLUSIONS. It is gaining consensus that avoiding consumption of peanut abrogates development of oral tolerance and increases risk of hypersensitivity through cutaneous exposure. The timing and perhaps the dosage and the balance of cutaneous and oral exposure determine whether a child will have allergy or tolerance. Crude edible peanut oil contains immunogenicity-competent protein fractions that might deserve further studies on its implication on peanut-allergy prevention.

REVIEWER COMMENTS. The authors suggested that oral consumption of crude peanut oil (contaminated with peanut protein) might have been protecting infants in Hong Kong from peanut allergy by tolerizing them and that now, without this early enteral exposure, more are becoming sensitized through cutaneous or respiratory routes. This concept is consistent with data from other studies that suggest that early feeding of food proteins is protective against the development of allergy and that early feeding avoidance (which leaves only cutaneous or respiratory exposure) might actually cause allergy. The American Academy of Pediatrics no longer advocates delayed introduction of any food past 4 months of age. Prospective, blinded, randomized trials are underway to better characterize the relationship between route, timing, and dose of food exposure and subsequent development of allergy.

URL: www.pediatrics.org/cgi/doi/10.1542/peds.2011–2107X

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Maternal Consumption of Peanut During Pregnancy Is Associated With Peanut Sensitization in Atopic Infants

Sicherer SH, Wood RA, Stablein D, et al. *J Allergy Clin Immunol*. 2010;126(6):1191–1197

PURPOSE OF THE STUDY. To identify factors associated with peanut sensitization.

STUDY POPULATION. The study population included 512 infants aged 3 to 15 months with likely milk or egg allergy but no previous diagnosis of peanut allergy.

METHODS. Enrollment criteria included history of immediate allergic reactions to cow's milk (and/or egg) and a positive skin-prick-test (SPT) result to milk (or egg if the clinical reaction was to egg) and/or moderate-to-severe atopic dermatitis and a positive SPT result to milk or egg. This longitudinal study was aimed at observing the development of peanut allergy; therefore, children with a known peanut allergy or known peanut-specific immunoglobulin E (IgE) level of ≥5 kU of antibody (kU_A)/L before enrollment were excluded. Maternal ingestion of peanut was queried retrospectively. In categorical analyses, frequent maternal peanut ingestion was defined as ≥2 times per week. A peanut-IgE level of ≥5 kU_A/L was used as the end point to signify a high likelihood of peanut allergy.

RESULTS. The 503 participants from whom blood samples were obtained were included. At enrollment, 140 (27.8%) of the participants were found to have a peanut-IgE level of ≥5 kU_A/L. A peanut-IgE level of ≥5 kU_A/L was associated with sensitization to egg or milk, male gender, nonwhite race, and frequent maternal peanut consumption during pregnancy. There was a dose-dependent association between frequent maternal peanut ingestion during pregnancy or breastfeeding and a peanut-IgE level of ≥5 kU_A/L, but only consumption during pregnancy was a significant predictor. Of the 71 infants who were never breastfed, frequent peanut consumption during pregnancy was related to a peanut-IgE level of ≥5 kU_A/L.

CONCLUSIONS. Maternal ingestion of peanut during pregnancy was strongly associated with peanut sensitization in infancy.

REVIEWER COMMENTS. Dietary advice for mothers during pregnancy and lactation is controversial because of conflicting results from previously published retrospective studies. This observational investigation of young atopic infants provides evidence that frequent consumption of peanut (≥2 times per week) during pregnancy is related to peanut sensitization; however, the development of clinical peanut allergy was not determined. The American Academy of Pediatrics (AAP) previously recommended peanut avoidance for pregnant and lactating women; however, the lack of scientific evidence to support such recommendations led to their withdrawal of that recommendation in 2008. Currently, neither the AAP nor the National Institute of Allergy and Infectious Diseases (NIAID) expert panel recommends maternal dietary avoidance of any foods, including peanut. The development of clinical peanut allergy among this study's cohort, as well as results of prospective investigations, will need to be examined before more definitive maternal dietary recommendations can be made.

URL: www.pediatrics.org/cgi/doi/10.1542/peds.2011–2107Y

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Peanut Oil and Peanut Allergy, Foes or Folks?

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Pediatrics 2011;128;S106
DOI: 10.1542/peds.2011-2107X

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Pediatrics 2011;128;S106
DOI: 10.1542/peds.2011-2107X