Timing of Initial Exposure to Cereal Grains and the Risk of Wheat Allergy

PURPOSE OF THE STUDY. To ascertain the relationship between cereal-grain exposure, wheat, barley, rye, and oats in the infant diet and the subsequent development of wheat allergy.

STUDY POPULATION. A birth cohort of 1612 children followed to a mean age of 4.7 years.

METHODS. A total of 1612 children were enrolled in a birth cohort with questionnaire data, and dietary exposures were determined at 3, 6, 9, 15, and 24 months and annually thereafter. The primary outcome measure was apparent report of wheat allergy. Wheat-specific immunoglobulin E (IgE) levels in children reported to have wheat allergy were obtained. Children with celiac disease with autoimmunity were excluded.

RESULTS. Sixteen children (1%) noted wheat allergy. Children who were first exposed to cereal after 6 months of age had a >4 times prevalence of wheat allergy when compared with children who were first exposed to cereals before 6 months of age, after controlling for family history and allergic disorder and history of food allergy before 6 months of age. All 4 children with detectable wheat-specific IgE were initially given cereal grains after 6 months. Having a first-degree relative with a history of atopy (ie, asthma, eczema, or hives) was also independently associated with an enhanced risk of subsequent development of wheat allergy.

CONCLUSIONS. Delaying first exposure to cereal grains until after 6 months enhanced the risk of subsequent development of wheat allergy. These results do not support delaying introduction of cereal grains for protection against food allergy.

REVIEWER COMMENTS. This study was limited by its dependence of parental report of wheat allergy, although IgE was obtained as well. No formal diagnostic oral food challenges were performed. The possibility of reverse causality was addressed by eliminating confounding factors related to history of food allergy but could still be a potential explanation. The birth cohort also was selected on the basis of HLA-genotype screening and family history of diabetes, which may limit its generalizability. However, the results, that delaying exposure to cereal grains until after 6 months is associated with increased risk of wheat allergy, not a protective outcome, are still provocative. Additional large prospective studies using double-blind, placebo-controlled food challenge to confirm these findings are needed.

Fish Consumption During the First Year of Life and Development of Allergic Diseases During Childhood

PURPOSE OF THE STUDY. To assess the association of fish consumption during the first year of life with development of allergic diseases by age 4.


METHODS. Parental questionnaires at child ages 2 months and 1, 2, and 4 years were administered to collect information on atopic heredity, diet, exposures, and allergic symptoms. When the children were 4 years old, a clinical investigation, including blood sampling for analysis of specific immunoglobulin E (IgE) to common inhalant and food allergens, was performed.

RESULTS. The mean age of introduction of fish in the child’s diet was 8.3 months. History of parental allergic disease and onset of eczema or wheeze during the first year of life were associated with delayed introduction of fish in the child’s diet. Regular fish consumption during the first year of life was associated with a reduced risk for allergic disease by age 4 (adjusted odds ratio: 0.76; 95% confidence interval: 0.61–0.94) and sensitization (adjusted odds ratio: 0.76; 95% confidence interval: 0.58–1.0) after exclusion of children with onset of eczema or wheeze during the first year of life to avoid disease-related modification of exposure. A dose-dependent reduced risk was observed for all outcomes (asthma, eczema, allergic rhinitis, and sensitization). IgE sensitization to fish was present in 18 of the 2614 children.

CONCLUSIONS. Regular fish consumption before the age of 1 seems to be associated with a reduced risk of allergic disease and sensitization to food and inhalant allergens during the first 4 years of life.

REVIEWER COMMENTS. In recent decades, a decrease in consumption of omega-3 polyunsaturated fatty acids, prevalent especially in oily fish, has been proposed to contribute to the increased prevalence of allergic diseases. The authors postulated that dietary polyunsaturated fatty acids might influence the development of allergic sensitization by increasing the formation of prostaglandin E2, which in turn promotes the formation of a T-helper 2 response and stimulates the formation of IgE. The authors attempted to control for the disease-related modification of exposure by excluding children with very early symptoms of eczema or recurrent wheeze. However, this attempt to control for “reverse causation” added limitation, because the most atopic infants were excluded. As the authors admitted, fish consumption in early life may be associated with a lifestyle that reduces...
Timing of Initial Exposure to Cereal Grains and the Risk of Wheat Allergy
Christopher Randolph
Pediatrics 2007;120;S109
DOI: 10.1542/peds.2007-0846L

Updated Information & Services
including high resolution figures, can be found at:
/content/120/Supplement_3/S109.1

Permissions & Licensing
Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
/site/misc/Permissions.xhtml

Reprints
Information about ordering reprints can be found online:
/site/misc/reprints.xhtml

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2007 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.

American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN™

Downloaded from by guest on April 19, 2017
Timing of Initial Exposure to Cereal Grains and the Risk of Wheat Allergy
Christopher Randolph

Pediatrics 2007;120;S109
DOI: 10.1542/peds.2007-0846L

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/120/Supplement_3/S109.1