



Food intolerance and allergy: increased incidence or contemporary inadequate diets?

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Purpose of review

The role of nutrients in the study of allergic disease has been studied for many years, but recent evidence suggests that it is the quality and variety of the whole diet which affects the development of food allergy. This review seeks to understand whether food allergy prevalence is increasing and the role of diet in the development of the disease.

Recent findings

New data on the European and global prevalence of food allergy suggest that both reported and actual rates are increasing. Recent findings from studies in infants and children indicated that a Western diet could enhance the development of allergic disease, possibly due to an effect on the homeostasis of the gut microbiome, impacting the immune status of individuals.

Summary

With overall prevalence rates for food allergy increasing, it is important to ascertain the relevance of dietary influences. Attention has shifted from single-nutrient prevention studies to evaluation of the effect of different dietary patterns on allergic outcomes. Evidence suggests that introduction of complementary foods should not be delayed, and the diversity of the diet in early life is important. The increasingly processed nature of the Western diet, in combination with influences on the gut microbiome, may contribute to the rising prevalence of allergic disease, including food allergy.

Keywords

allergy, diet, microbiome, nutrition, prevalence

INTRODUCTION

Although the media portrayal of food allergy and food intolerance appears to show that they are reaching epidemic proportions, the reality is less certain. Food allergy [1*,2] is defined as ‘an adverse health effect arising from a specific immune response that occurs reproducibly on exposure to a given food’, and usually involves the production of food-specific IgE antibodies, although other cells of the immune system can also be implicated. Food intolerance [1*,2] is a nonimmune reaction mediated by metabolic, toxic, pharmacologic or undefined mechanisms. A significant proportion of the population reports adverse reactions to foods, but only a small percentage will have food allergy. The dietary norms of many populations have changed over the last few decades. Could an inadequate, nutritionally poor diet result in the development of food allergy or food intolerance, or are symptoms attributed to foods incorrectly interpreted and merely the result of a diet that is not conducive to good digestion? One area of interest has been the

relationship between diet in early life and allergy prevention, and more recently focus has also moved to the influence of contemporary diets on the development of allergy. This review will commence with a summary of the reported and actual prevalence of food allergy and food intolerance, and then address the role of diet and nutrition in the development of adverse food reactions and whether changes in the composition of the contemporary diet could account for the increase in adverse food reactions.

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KEY POINTS

- Although there is global variation and lack of data, the prevalence of diagnosed food allergy appears to be increasing, especially in some continents with apparent links to Westernization.
- Gut microbiota are important in early-life immune development; diet directly affects the microbiota; therefore, a poor nutritional intake could be linked to loss of gut-wall epithelial integrity and the development of IgE-mediated food allergy.
- Prevention guidelines have changed; they no longer recommend delay in the introduction of high allergic complementary foods, rather exclusive breastfeeding for 4–6 months, with the introduction of complementary foods taking place from 4 months onwards.
- Dietary influences on food allergy have previously concentrated on single nutrients, but evidence suggests that dietary patterns are more important, with a Western diet linked to poor allergic outcomes, whereas increased diversity is associated with a lower incidence of food allergy.

INCIDENCE/PREVALENCE OF FOOD ALLERGY/INTOLERANCE

An indication of the prevalence of food allergy and food intolerance can be understood from population studies on reported adverse reactions to food. A systematic review of food allergy prevalence, commissioned by the European Academy of Allergy and Clinical Immunology (EAACI) [3^{***}], evaluated 56 primary studies published between 2000 and 2012. The pooled point prevalence for food allergy diagnosed by self-report was 5.9% [confidence interval (CI) 95%: 5.7–6.1], similar to the overall rate of 8.96% (95% CI: 8.32–9.60%) extrapolated from the US National Health and Nutrition Examination Survey (NHANES) [4]. The rate of food allergy diagnosed by oral food challenge (OFC) in Europe was 0.9% (95% CI: 0.8–1.1) [2], although, since studies using OFC are rare, a revised estimate on the basis of studies in which subjects had a convincing clinical history and/or positive OFC was 2.6% (95%CI: 2.1–3.1) in children. There were no comparable data for adults, although one UK adult population survey [5] using a diagnostic questionnaire validated against OFC, reported a 2% prevalence of oral allergy syndrome, the predominant food allergy in adults, characterized by pollen sensitization provoking reactions to homologous allergens in plant foods.

A more global picture of the prevalence of paediatric allergy comes from a questionnaire survey of 93 member societies of the World Allergy Organisation [6^{***}]. Out of the 89 who responded, 50% had no

prevalence data, and there was considerable global variation amongst those who did. For reported prevalence, the lowest rates, around 5% or less, were noted in Japan, Hong Kong, Korea, Slovenia, Switzerland and Greece. The USA and Canada had rates of 7 and 8%, compared with 15% in the UK and 19% in Mozambique; lifetime rates of reported food allergy were highest in African nations. Only 10% of respondents had food allergy data on the basis of oral food challenge; these data found the true prevalence of food allergy in infants to range from 1% in Thailand to 10% in Australia. A key observation was that rates in South Asia were increasing, with the rate in China comparable to that of the UK and Denmark. The authors postulate that the higher rates of food allergy recorded in Hong Kong-born Chinese as compared with the rates of those born in mainland China indicate that food allergy development is associated with 'Westernized lifestyles'. Both European and global studies imply that whilst the incidence of food allergy is stable, prevalence appears to be increasing.

The huge spectrum of conditions under the umbrella of food intolerance means that prevalence is less well documented. However, attempts have been made to quantify the prevalence of wheat intolerance in particular noncoeliac gluten sensitivity (NCGS) (gastrointestinal symptoms on ingestion of wheat in the absence of coeliac disease). This condition is of global interest in both the popular press and academic journals, but the true prevalence of NCGS is unknown. A survey of Australian adults [7] reported that 7.3% of the population were avoiding wheat, although more robust data from the USA suggest the reported prevalence of NCGS to be 0.548% (95% CI 0.206–0.889), approximately half that of coeliac disease [8]. The mechanisms of NCGS are unknown [9] and clarification is needed as to whether the beneficial effect of wheat withdrawal is entirely because of the gluten, other wheat proteins or the inulin-type fructans found in the whole wheat [10[■],11,12[■]].

THE MICROBIOME AND DEVELOPMENT OF FOOD ALLERGY

Gut microbiota influence the immune and metabolic systems, and are now recognized as important determinants for the development of allergic disease in parallel with host genotype and diet [13–16,17^{***}]. It is hypothesized that lack of exposure to beneficial microbes in the environment induces an altered gut microbiome that has modulating effects on the gut immune function, leading to a lack of tolerance and inflammation and hence allergic disease and obesity [14,15,18]. Gut microbiota play a vital role in early life immune development and function, and

maintenance of gut-wall epithelial integrity, which might explain the link with IgE-mediated food allergy and its association with immune dysregulation and impaired gut epithelial integrity [18]. Ling *et al.* [19] found that changes in key bacterial phylotypes in infancy were associated with the development of food allergy, with a specific microbiota signature distinguishing infants with IgE-mediated food allergy from those without IgE-mediation. In a systematic review, Marss *et al.* [20] found that caesarean delivery increased the risk of challenge-proven or doctor-diagnosed food allergy, whereas having siblings and attending childcare before 6 months decreased the risk of challenge-proven food allergy. These data are supported by the results from the HealthNuts study [21]; this population-based cohort study found that food allergy was twice as common in infants without siblings compared with those with siblings (10.2% vs. 5.6% $P=0.001$), unless the siblings were allergic.

The pre and postnatal periods provide important windows of opportunity for gastrointestinal microbial colonization and modulation of the immune and metabolic systems. Nutrition during pregnancy and early postnatal life is, therefore, important; diet modulates the gut microbiome by providing substrates (e.g., dietary fibre, fat, proteins and prebiotics), regulating transit time, pH, bile and enzyme secretions, and regulating gene expression in the host and the microbes. Diets can also provide pre and probiotics. In a mouse model of allergy it was shown that prebiotic exposure during perinatal and post-weaning periods induced the highest expression of biomarkers related to tolerance without affecting biomarkers related to allergy [22]. The immune-modulating effects of these oligosaccharides are likely assisted by alterations of the intestinal microbiota, in a microbiota-independent manner by direct interaction with immune cells, or both [23].

Although diet modulates the microbiome, medications used to treat gastro-oesophageal reflux disease (GORD) or hyperacidity might also have an adverse effect on the development of food allergy, irrespective of the quality of the diet. Trikha *et al.* [24] compared 4724 children aged 0–18 years diagnosed with GORD and treated with gastric acid suppressive medication (GAS) to 4724 children with GORD but not treated with GAS. The children with GORD treated with GAS were more likely to be diagnosed with a food allergy [Hazard Ratio: 3.67, 95% CI 2.15–6.27].

PREVENTION AND EARLY LIFE INFLUENCES

The modulation of dietary factors in early life could help prevent food allergy. A new evidence-based

guideline on the primary prevention of food allergy [25,26] published by EAACI advocates no dietary restrictions during pregnancy and lactation, and exclusive breastfeeding of infants for the first 4–6 months of life. If breastfeeding is insufficient or not possible, a hypoallergenic formula with a documented preventive effect should be given for the first 4 months. One of the main studies providing evidence for this guidance comes from the German Infant Nutritional Intervention study. Infants unable to breastfeed randomly received either partially hydrolyzed whey formula (pHF-W), extensively hydrolyzed whey formula (eHF-W), extensively hydrolyzed casein formula (eHF-C) or standard cow's milk formula for the first 4 months of life. The 10-year follow-up data [27] showed a significant persisting preventive effect on the cumulative incidence of allergic diseases, particularly atopic dermatitis, with pHF-W and eHF-C, whereas eHF-W showed, surprisingly, no significant risk reduction.

Thus, delayed exposure to cow's milk may particularly be useful for reducing the risk of atopic dermatitis. Complementary foods can be given from 4 months of age, with no delay to the introduction of potentially allergenic foods once weaning has commenced. Grimshaw *et al.* [28] found that continued breastfeeding during the introduction of solids, and delaying the introduction of solids until at least 17 weeks of age, was also associated with fewer food allergies. Prolonged avoidance of high allergenic foods might be a risk factor, because oral tolerance induction would be bypassed, whereas alternative sensitizing routes of exposure (e.g., cutaneous) were ongoing [1].

Single nutrients, such as omega-3 fatty acids, selenium, zinc, folate, vitamin A, vitamin C, vitamin D and vitamin E [16,17], have all been studied to assess their protective effects against the development of food allergy. The significance of vitamin A [29] in food allergy prevalence is only provided through a hypothetical association because of its role in the immune system, whereas there is some indirect evidence that variation in food allergy prevalence within countries might be linked with ambient ultraviolet radiation exposure and thus potentially with vitamin D levels. Recently published data [30] found that children with vitamin D insufficiency (≤ 50 nmol/l) were more likely to be peanut allergic [adjusted odds ratio (aOR), 11.51; 95% CI, 2.01–65.79; $P=0.006$] and/or egg allergic (aOR, 3.79; 95% CI, 1.19–12.08; $P=0.025$) than were those with adequate vitamin D levels, independent of eczema status. van den Elsen *et al.* [31], investigating the protective effect of omega-3 fatty acids derived from fish oil, found that acute allergic skin responses to whey were reduced by more than 50% in

whey-sensitized mice fed a fish oil diet, compared with a control diet. In addition to suppressing allergic sensitization to whey, the fish oil increased T regulatory cells, enhancing the tolerizing capacity of the adaptive immune system.

However, although intakes of omega-3 fats are still below the recommended level in many countries, the global daily intake of omega-3 fats has risen substantially in the last 10–20 years [32,33], suggesting that supplementation of a single nutrient is unlikely to affect the prevalence rates of food allergy. A systematic review by Saadeh *et al.* [34²²] concluded that although Westernization is associated with a rise in allergic disease, the evaluation of individual dietary factors has resulted in contradictory findings often of low significance. They reiterated the importance of evaluating the whole diet and evaluating nutritional patterns so that the complex interactions of nutrients eaten together could be evaluated.

THE INFLUENCE OF CONTEMPORARY DIETS ON THE DEVELOPMENT OF FOOD ALLERGY AND FOOD INTOLERANCE

Only a small percentage of populations are achieving what is considered to be a healthy diet. The data from the NHANES study on 5211 US participants found that only 16.5% of participants (weighted proportions) were classified as consuming a healthy diet and being sufficiently active [35]. Jahns and Kranz [36] showed that 60% of foods consumed in one US study contained solid fats and added sugars, which predicts low-diet quality and excess energy intake. National dietary data show that half of Canadian adults do not meet the minimum daily requirement for fruit and vegetables and more than 25% obtained more than one third of their energy from fat [37]. There is also an increase in the use by Canadians of ready-to-consume products to 61.7% in 2011 [38]. The authors concluded that the most important change in Canadian dietary patterns between 1938 and 2011 is the displacement of unprocessed or minimally processed foods and culinary ingredients used in the preparation of dishes and meals by ready-to-consume ultraprocessed products.

The 'Western diet' [39] which involves a high intake of fat, protein, sugar and salt, and frequent consumption of processed and 'fast foods', could promote autoimmune diseases, with T cells linking dietary influences to autoimmune disease. In contrast, the Mediterranean diet has a high nutritional quality, good quality of dietary fats, anti-inflammatory effects and higher quality antioxidants [40]. Consumption patterns in Brazilian children revealed a significant association between

the 'Western' pattern and wheeze [odds ratio (OR) = 1.77, 95% CI: 1.10–2.84] compared with the traditional Brazilian or 'Prudent' diet pattern [41²³]. High adherence to a 'Western' dietary pattern (refined grains, soups and sauces, savoury snacks, other fats, sugar-containing beverages and meat) was significantly associated with shortness of breath and wheeze at 3 years of age [adjusted response rate (aRR) 1.47: 95% CI 1.04–2.07] and frequent chest infections at 4 years of age (aRR 1.46, 95% CI 1.00–2.13). The same effect was not seen with the 'Health conscious' pattern associated with starchy foods, fruits, vegetables, potatoes, vegetable oils, fish, meat and legumes [42].

The positive influence of whole dietary patterns may also be explained by the importance of dietary diversity, demonstrated by prospective birth cohort studies. A nested, case-control, within-cohort UK study of 41 cases and 82 age-matched controls found that an infant diet containing high levels of fruit, vegetables and home-prepared foods was associated with significantly less food allergy by the age of 2 years [43²⁴]. In a Finnish prospective birth cohort study of 3142 children, less food diversity during the first year of life was associated with an increase in the risk of asthma and allergies in childhood [44²⁵]. The same was true in 856 children from another birth cohort study; increased diversity of complementary foods introduced in the first year of life was inversely associated with asthma, with a dose-response effect [aOR with each additional food item introduced, 0.74 (95% CI, 0.61–0.89)]; a similar effect was observed for food allergy and food sensitization [45²⁶]. Dietary patterns appear to change as children get older; one study on obesity revealed a noticeable transition to less favourable dietary intakes for fibre, fat, fruit and vegetables, confectionery and snacks and sugar-sweetened beverages as children get older [46].

Food allergens are never in contact with the immune system in a purified form, but as part of a complex mixture of substances known as the food matrix. A review study from McClain *et al.* [47²⁷] reminds us that within the matrix, lipids can influence sensitization and severity of response, sugars can alter digestion and impact the modification of allergens during processing, and proteins may also affect allergen digestion. For example, the addition of medium chain triglyceride oil to the diet of mice promoted allergenic sensitization to simultaneously ingested antigens in peanut butter [48]. Medium chain triglyceride (MCT) oil is added to many products and unlike long chain fats MCTs do not form chylomicrons on digestion, which, the authors postulated, might be the key difference in protection against allergic sensitization.

OTHER FACTORS WHICH MAY AFFECT THE DEVELOPMENT OF FOOD ALLERGY

Although food allergens are normally consumed in the diet, there are other routes of exposure which may be relevant. Two studies by Brough *et al.* [49,50] have shown that biologically active peanut allergen is in the environment, being transferred by hands or saliva, with the quantity of peanut in the environment directly linked to the quantity of peanut consumed in the home. The authors considered that peanut protein is unlikely to sensitize through inhalation, because aerosolized peanut protein disappears quickly, but it is clear that in homes with a high peanut consumption, there is the possibility that peanut allergen in household dust could sensitize through cutaneous or oral routes. Food allergens from milk, peanut, egg and fish were detected in dust samples from the mattresses of Norwegian adolescents [51]. The authors concluded that although inhalational or skin exposure may only induce limited reactions, this mattress dust is an important consideration in the development of food allergy, not least because of the amount of time children spend in their bedrooms.

Although diet is a key factor in the development of food allergy, many other factors play a seminal role. The HealthNuts study confirmed a long held view of the relevance of a family history of allergy. The results [21[■]] showed that 70% of the children with diagnosed food allergy had a family history of allergy, the risk being much greater with more than one immediate family member being affected. The situation is less clear-cut with adults; a longitudinal study [52] found that new onset food sensitization in adults was not associated with age, sex, BMI, rhinitis, asthma, smoking or sensitization to aeroallergens, but persistent sensitization was related to higher total IgE levels at the baseline, but not to levels of specific IgE or aeroallergen sensitization. Their conclusion was that the incidence of food allergy in adults may be related to the atopic trait of an individual.

CONCLUSION

Although levels of food allergy and food intolerance vary globally, it appears that countries adopting Western lifestyles are especially vulnerable. For both food allergy and food intolerance, large-scale prevalence studies are needed to inform health economies, to enable appropriate support for the diagnosis of food allergy and the care of affected patients. Many dietary factors undoubtedly play a role in the development of food allergy; however, the quality and diversity of the whole diet and the influence of a Western dietary pattern on the development of

food allergy is possibly the most significant new finding. Key to our understanding of the interaction between diet and food allergy is more research into the effects of processing on the allergenicity of foods, further evaluation of food diversity and dietary patterns, and the effect of a typical Western diet on the gut microbiome in comparison to a healthy or traditional diet.

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Conflicts of interest

There are no conflicts of interest.

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