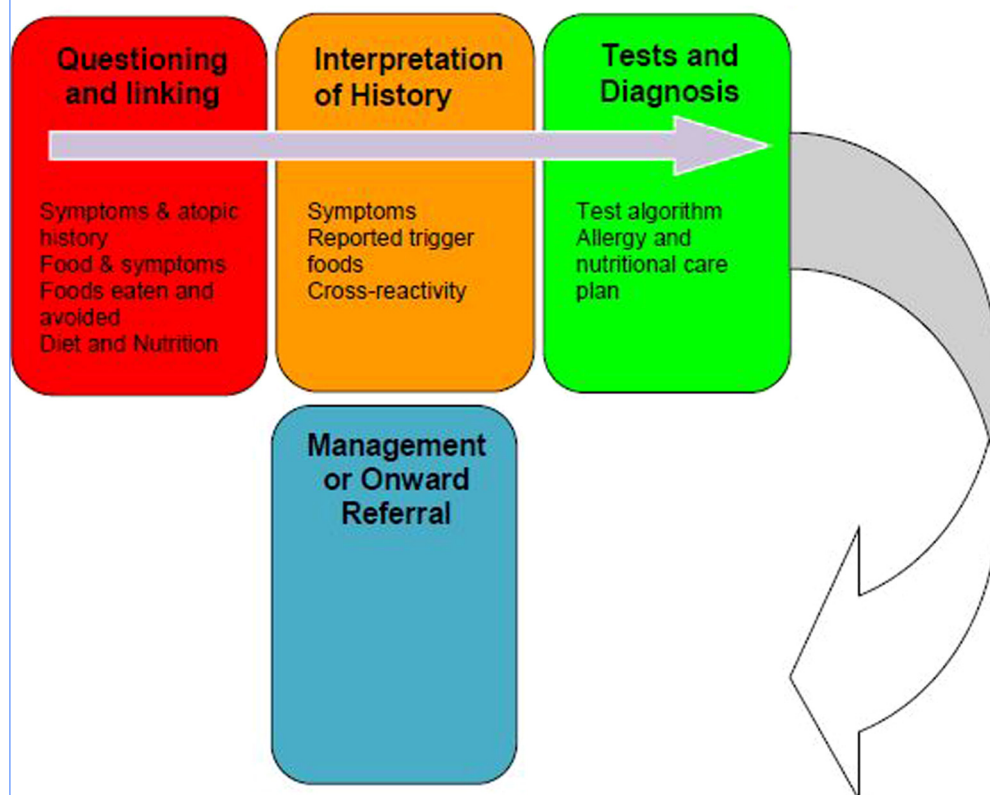




## Allergy-Focussed Diet History Paediatric version



# The development of a standardised diet history tool to support the diagnosis of food allergy

Skypala *et al.*

REVIEW

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# The development of a standardised diet history tool to support the diagnosis of food allergy

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## Abstract

The disparity between reported and diagnosed food allergy makes robust diagnosis imperative. The allergy-focused history is an important starting point, but published literature on its efficacy is sparse. Using a structured approach to connect symptoms, suspected foods and dietary intake, a multi-disciplinary task force of the European Academy of Allergy and Clinical Immunology developed paediatric and adult diet history tools. Both tools are divided into stages using traffic light labelling (red, amber and green). The red stage requires the practitioner to gather relevant information on symptoms, atopic history, food triggers, foods eaten and nutritional issues. The amber stage facilitates interpretation of the responses to the red-stage questions, thus enabling the practitioner to prepare to move forward. The final green stage provides a summary template and test algorithm to support continuation down the diagnostic pathway. These tools will provide a standardised, practical approach to support food allergy diagnosis, ensuring that all relevant information is captured and interpreted in a robust manner. Future work is required to validate their use in diverse age groups, disease entities and in different countries, in order to account for differences in health care systems, food availability and dietary norms.

**Keywords:** History, Diet, Tool, Allergy, Food, Diagnosis

## Introduction

Adverse reactions to foods are frequently reported, however only those involving immunological mechanisms, including both immunoglobulin E (IgE) and non-IgE mediated, can be defined as food allergy (FA) [1]. The prevalence of FA varies worldwide, but the rate of true FA in children and adults is consistently lower than self-reported rates [2-5]. A European systematic review found the overall point prevalence in Europe of self-reported FA to be 5.9% (95% CI: 5.7-6.1), compared with a food challenge confirmed FA rate of 0.9% (95% CI: 0.8-1.1) [6]. Reported FA persists over time in adults [7], and age-related changes can affect the immune system, increasing the potential for newly diagnosed food allergy in older adults [8].

This disparity between reported and actual levels of FA make a robust diagnosis imperative in order to avoid overt and/or unnecessary dietary restrictions leading to

either continuation of symptoms due to the wrong food being eliminated or nutritional deficiencies [9,10]. Guidelines [1,11-13] provide expert evidence-based support for the diagnosis of FA, which includes taking an allergy focussed history, performing appropriate tests and food reintroduction or controlled food challenge. Although international consensus agrees that the allergy-focused history is a key part of the diagnostic pathway [1,12], the lack of standardisation, and variable expertise of the history taker, may prevent the ascertainment of sufficient information, leading to poor interpretation of the facts.

However, when the right questions are posed and the answers are systematically linked to appropriate actions, an allergy history can be invaluable [11,14], and for some conditions, history has been demonstrated to be wholly diagnostic [15]. Included in the additional supporting information of recent European Academy of Allergy and Clinical Immunology (EAACI) FA guidelines was a useful list of key questions for the clinical history [1]. Therefore these guidelines not suggest an allergy-focused

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history is fundamental to the establishment of a diagnosis, they also provide a starting point for a structured approach to history-taking. Recognition of the importance of the diet and clinical history by EAACI led to the formation of an EAACI Task Force to develop paediatric and adult diet history tools for use by health care professionals.

## Methods

The Allergy-focussed Diet History Task Force, an international multi-professional group of food allergy experts, agreed that evidence-based tools would need to be developed. In order to gather evidence of links between reported symptoms, trigger foods and dietary intake, and the existence of any published diagnostic questionnaires, a search on Pub Med and Medline was undertaken. The search terms shown in Table 1 were used, and papers from 1990 to January 2012 were selected in the order given below:

- 1) Randomized controlled trials
- 2) Non-randomized controlled clinical trials
- 3) Before and after clinical trials
- 4) Prevalence studies employing oral food challenge
- 5) Systematic reviews and other meta analyses
- 6) Observational studies – cohort or case reports
- 7) Other subject reviews

The search resulted in 36 publications, but only one study measured the diagnostic efficacy of history against

**Table 1 Search terms for review**

Tree	food hypersensitivities
Tree	food hypersensitivity
Tree	hypersensitivity food
allergies food	questionnaire
allergy food	questionnaires
assessing	recognition
assessment	recognise
detect	recognising
detecting	screen
detection	screening
diagnose	signs
diagnoses	symptoms
diagnosing	work up
diagnosis	workup
diagnostic intent	
evaluating	
evaluation	
food allergies	
food allergy	

validated test methods [15]. Access to more rigorous methodology employed by the EAACI Food Allergy Guidelines Task Force [6], yielded no additional appropriate papers. In the absence of a lack of suitable published evidence, the Task Force undertook to develop age-specific diet history tools based on expert opinion, with the factors considered being supported by guidelines or underpinned by evidence from individual studies. A structure for the tools was established to enable rapid assimilation and interpretation of answers to standard questions on symptoms, atopic history, family history of atopy, co-factors, suspected or known food triggers, quantity of food involved, foods currently consumed and avoided, cross-reacting foods, growth and development and likelihood of nutritional risk. The pooled knowledge and expertise of the panel greatly contributed to the construction of the questions, especially where evidence was lacking.

## Results

Two allergy-focussed diet history tools for paediatric and adult patients were developed (see Additional files 1 and 2) based on a traffic light scheme similar to those used to inform consumers about healthy eating or energy efficiency:

**Red** - gather information through questioning the patient on their allergic history, foods habitually eaten, nutritional status, and relevant co-factors.

**Amber** – facilitates interpretation of the responses to the red-stage questions by linking the information gained to match potential diagnostic pathways, utilising the algorithms and tables provided to refine and formulate a potential diagnosis thus enabling the practitioner to prepare to move forward.

**Green** - the way ahead is clear to institute appropriate testing and/or onward referral to a specialist allergy centre, gastroenterologist or a specialist dietitian.

## Red stage - questioning and linking

### Symptoms and atopic history

#### Symptoms

The characterization of symptoms is an essential first step when taking a history from someone presenting with adverse reactions to foods.

#### Presenting symptoms: IgE-mediated FA

A number of guidelines have summarised these symptoms [1,11-13], primarily those seen in children, which can be skin related (pruritus, erythema, acute urticaria, acute angioedema), gastro-intestinal (anal pruritus, colicky abdominal pain, diarrhoea and vomiting) or upper and lower respiratory symptoms. In adults, skin reactions are also associated with IgE-mediated food allergy [16], but gastrointestinal symptoms are predominant especially dysaesthesia of the tongue, oro-

pharyngeal pruritus and vomiting [17-20]. Rhinitis, conjunctivitis and asthma are also presenting features of adult food allergy [5,15]. Tachycardia, hypotension, throat tightness, bronchospasm, laryngeal oedema, shortness of breath, syncope and anaphylaxis can affect all ages [21,22].

#### **Presenting symptoms: non-IgE mediated FA**

Proctocolitis [23] is characterised by blood and mucous in the stools and occasional diarrhoea, although the infant appears well. The symptoms of food protein-induced enterocolitis syndrome (FPIES) include late onset gastro-intestinal symptoms of profuse vomiting, diarrhoea, pallor and occasionally hypovolemic shock [24]. Children and infants with Eosinophilic Oesophagitis (EoE) often show signs of chronic vomiting, abdominal pain, poor appetite, food refusal, other feeding refusal behaviours as well as dysphagia [25]. Up to one third of young children with Atopic Dermatitis (AD) may present with a FA, particularly those with moderate to severe eczema unresponsive to topical treatment [26,27]. Adults have less well documented non-IgE-mediated food allergies, but dysphagia and oesophageal food impaction are particularly associated with adult-onset EoE [28].

#### **Presenting symptoms: other symptoms caused by food**

Sometimes symptoms of an allergic disorder can resemble those due to non-immune mediated adverse reactions to foods, thus complicating the diagnostic process. For example, dietary histamine has been reported to provoke rhino-conjunctivitis, flushing, pruritus, urticaria, asthma, hypotension and abdominal cramping [29]. The food additive sodium metabisulphite has been linked to symptoms of rhinitis, nasal blockage, wheeze and abdominal pain [30]. Abdominal symptoms are a notable feature of Irritable Bowel Syndrome (IBS), a diagnosis which is positively associated with pain in the lower abdomen, pain relieved by bowel movements, frequent pain and abdominal bloating [31].

#### **Onset of symptoms**

It is important to establish the temporal relationship between eating a food and the onset of symptoms. It is generally agreed that the sooner the symptoms occur after eating, the greater the likelihood that an IgE-mediated mechanism is provoking the reaction [1,12]. Non-IgE-mediated FA symptoms are usually delayed; children with FPIES typically experience severe vomiting 1–4 hours after ingestion of the suspect food [32]. However occasionally symptom speed of onset does not provide such clear signposting; co-factors such as exercise can mislead if the symptoms only occur during exercise, but the food involved could have been eaten up to 2 hours earlier [33]. Another example is delayed anaphylaxis to red meat occurs in relation to IgE antibodies

to the oligosaccharide galactose- $\alpha$ -1,3-galactose ( $\alpha$ -gal) generated by tick bites [34]. Conversely, some non-immune-mediated adverse food reactions may manifest soon after eating, i.e. gastrointestinal symptoms triggered by lactose intolerance [35], or wheeze provoked by the food additive sodium metabisulphite [30].

Other important factors relating to symptoms include ascertainment of the quantity of food required to trigger symptoms, frequency and reproducibility of the reactions and the interval since the last reaction [1,11-13]. The age of onset of symptoms, feeding/diet history, history of any food elimination and any previous therapeutic interventions are also significant.

#### **Atopic history**

For children, in addition to a physical examination focusing on growth and development, any allergy related co-morbidities and family history of atopy should be ascertained [1,11]. The likelihood of food allergy is enhanced in children with moderate to severe eczema [26]. For adults, a physical examination is also helpful, but it is also vital to establish atopic status due to the strong association between atopic disease and the manifestation of food allergy in adults [36]. Allergic rhinitis is increasingly prevalent in food allergic adults, reportedly affecting 41% of UK adults suffering from adverse reactions to foods in 2009, compared to 25% in 1991 [37,38]. Asthmatics with food allergy are also more likely to have severe and uncontrolled asthma [39] and suffer from fatal or near fatal food anaphylaxis [22].

#### **Known aeroallergen sensitisations**

There is an increasing level of aeroallergen sensitisation in all age groups, which persists into old age [40,41]. Thus establishing sensitisation to pollens, mites and animal dander is vital when interpreting the history, especially as geographical variation in aeroallergen sensitisation across Europe affects prevalence and foods involved [42]. Positive tests to pollen [15], latex [43] or house dust mite [44], can be linked to homologous reactions to fruits, vegetables, nuts or shellfish. More unusual cross-reactions include sensitisation to bird feathers inducing symptoms to the egg yolk allergen alpha livetin [45], individuals sensitised to galactose- $\alpha$ -1,3-galactose ( $\alpha$ -gal), reacting to beef and pork [34], and reactions to pork associated with sensitisation to cats [46]. The sensitising agent might be a food-related parasite such as cereal mites [47] or the *Anisakis* nematode worm [48].

#### **Extrinsic (co) factors and routes of exposure**

In adults, exercise, alcohol, stress, aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) can enhance or precipitate an allergic reaction to food [49]. Food-dependant exercise-induced anaphylaxis (FDEIA) is characterised by

a lack of reaction to the trigger food unless it is consumed in close proximity to taking exercise [50]. Wheat, crustaceans, tomatoes, celery, strawberries, cheese have all been implicated [33,49-53]. Alcohol can also act as a co-factor, but symptoms to alcohol could also be due to an allergy to grapes or barley [54,55], sensitivity to vasoactive amines or sodium metabisulphite [29,30], or caused by a congenital deficiency of the enzyme alcohol dehydrogenase [56]. Food additives have also been shown to act as co-factors in cases of anaphylaxis [53]. Co-factors may be less relevant in children [57], but exposure to allergens other than via the oral route require consideration in both children and adults; these include inhalation of aerosolized allergens, transfer of allergens during cooking or by skin contact [58-60].

### **Food & symptoms**

#### ***Food triggers – IgE-mediated FA***

Although there are a large range of foods which can be implicated in IgE-mediated FA, only a limited number regularly provoke reactions in most people. Most reactions in children are due to milk, egg and peanut [2], with milk and egg being the most common causes of anaphylaxis [61]. Milk, egg, wheat and soy allergy usually remit in late childhood [62-65], thus a primary allergy to these foods is rare in adults. Wheat allergy in adults is generally associated with FDEIA [52] and adults reporting reactions to soy usually develop symptoms due to homology between soy protein and birch pollen allergens [66]. Peanut and tree nut allergy in adults is usually a persistence of childhood allergy since only 8-10% of cases are newly diagnosed in adolescence or adulthood [67-69]. Up to 30% of adults with a peanut allergy have cross-reactivity to other legumes such as lupine [70]. Lupine allergy also presents in childhood, but is far less prevalent than peanut allergy in the general population, affecting less than 0.3% of patients with reported reactions to foods [71]. Seeds such as sesame and mustard are also a significant cause of IgE-mediated reactions in certain populations [72,73]. Those with a primary allergy to one type of nut, seed or legume can react to other similar foods due to co-sensitisation to homologous allergens [74-79].

Fish and shellfish trigger both paediatric and adult IgE-mediated FA [80,81], often provoking severe reactions [22,82]. Pan allergens in vertebrate fish account for strong interspecies cross-reactivity; thus sensitization to more than one species is common in fish-allergic individuals [80]. The same is true for crustaceans and molluscs, although sensitisation to minor seafood allergens can limit clinical reactivity solely to the seafood which provoked the index reaction [83]. The lack of homology between the pan allergens in vertebrate fish and shellfish usually means fish-allergic individuals can tolerate shellfish and vice versa [80].

Fruits and, to a lesser extent, vegetables, are often the most frequently reported foods to provoke new onset reactions in adults [5,17,18,37]. These reactions are usually due to oral allergy syndrome or pollen-food syndrome (PFS), a common manifestation of cross-reactive plant FA in adults, which can affect 50% or more pollen-sensitized individuals [15,17]. Such reactions are also reported in older children and teenagers, although only limited epidemiological data supports this [84]. Reactions, usually to more than one food, typically occur to tree nuts, apples, peaches and cherries, although peanuts and soy may also be involved [15,17,85-87]. If PFS is suspected then any history of seasonal hay fever should be linked to symptoms to fruits and vegetables, and symptoms may be worse or only occur during the pollen season [61]. Homologous allergens in natural rubber latex (*Hevea brasiliensis*) can also cross-react with foods, in particular kiwi fruit, avocado, chestnuts and bananas [44]. Lipid Transfer Protein (LTP) allergy should also be considered if plant foods provoke severe reactions [88]. LTP allergy, usually associated with sensitisation to the peach LTP allergen Pru p 3 [88,89], is highly prevalent in southern Europe [90], and associated with presence of co-factors such as alcohol and exercise [19,49].

Whilst children might have a reaction to a food soon after the first exposure, older children and adults may suddenly develop symptoms to foods consumed for many years. However, foods habitually eaten are mostly considered as safe and should not be tested. If the reactions are to multiple foods and PFS is not suspected, then a 'hidden' allergen used as an ingredient in composite dishes may be implicated, such as legumes, seeds, celery and natural food colourings.

#### ***Food triggers – non-IgE-mediated FA***

FPIES typically occurs in response to milk or soy proteins in infant formula, and more rarely to food proteins in breast milk [91,92]. The common provoking allergens in FPIES are milk, rice and soy, but reactions have also been reported to oats, fish, egg and some fruit and vegetables [93]. Although eosinophilic gastrointestinal disorders are seen in both adults and paediatrics, offending foods may differ [94]. In children, milk, egg, wheat and soy have been found to be most relevant, although in some cases fish, nuts and peanuts also play a role, although meat proteins (including, beef, lamb, chicken and pork), carrots, potato, maize and peanuts may also be causative foods [95]. Wheat and milk appear to be major allergens in adults although other triggers could include corn, rice and legumes [96].

#### ***Food triggers – non-immune mediated adverse food reactions***

Milk is most frequently linked to non-immune mediated conditions in adults including lactose intolerance [35],

IBS [97], and some respiratory conditions [98]. In the absence of IgE-sensitisation to wheat, coeliac disease [99] should be excluded prior to considering differential diagnoses such as non-coeliac gluten sensitivity [100] and IBS [101]. There is some evidence that foods containing high levels of oligo-, di-, mono-saccharides and polyols carbohydrates (FODMAPS™) could provoke some or all of the gut symptoms experienced by IBS sufferers [102]. Oligosaccharides (wheat, rye, onions, garlic, artichokes and legumes) probably affect the majority of IBS sufferers, 45% of whom will also be intolerant to foods high in fructose (honey, apples, pears, watermelon and mango) [103]. Reported reactions to multiple foods in the absence of IgE-sensitisation can indicate a non-immune mediated reaction. Potential mediators may include food additives (azo dyes, benzoates, sulphites and mono-sodium glutamate) [30,104], or naturally-occurring substances such as vasoactive or biogenic amines (fish, pork and fermented or aged meat products, strong cheeses, red wine, spinach, aubergine and yeast extract) [29], or salicylates (coffee, dried herbs and spices, cherries, strawberries and certain apple varieties) [105] although robust evidence on the prevalence of these types of reactions is lacking.

#### Foods eaten and avoided

All foods and food products which can be consumed without any symptoms should be noted, especially common allergenic foods, or 'hidden' food allergens such as mustard, celery, soy and lupine. Emerging evidence indicates that some children can tolerate foods containing

well-cooked milk and egg, but will still react to raw/less well cooked forms [106,107]. Similarly those with PFS may be able to tolerate cooked fruits but not raw [15,17]. Reported avoidance of the trigger food may not be complete exclusion [108] due to lack of awareness of foods that contain small amounts of allergen. A dietetic consultation will establish true abstinence and information from food labels, recipes or food diaries may also be useful [109].

#### Nutritional issues

Nutritional impairment is a tangible risk in those with suspected FA; the elimination of food allergens often entails the exclusion of foods that contribute essential nutrients for growth and development (Table 2) [9,10]. Poor growth and stunting has been shown to occur in children with IgE-mediated FA who are on exclusion diets [10,110], with the number of foods excluded linked to a low weight for age and height for age in such children [10]. Feeding difficulties may also occur as clinical features of non-IgE mediated FA. Adults generally are not in danger of developing major nutritional deficiencies although individuals avoiding multiple foods can be at risk [111]. Therefore both tools contain a nutritional assessment section which, on completion, should indicate whether onward referral for specialist nutritional intervention is required. A dietitian can ascertain and interpret the diet history to ensure normal growth and development, employing a variety of measures to determine dietary intake [112,113]. The nutritional analysis can signpost the requirement for nutritional supplements

**Table 2 Main food allergens and their nutrient content [118]**

Allergen	Nutrients involved
<b>Milk</b>	Protein, Carbohydrate, Fats, Vitamin A and vitamin D, riboflavin, pantothenic acid, vitamin B12, calcium, magnesium, phosphate
<b>Egg</b>	Protein, Riboflavin, biotin, vitamin A, vitamin B12, vitamin D, vitamin E, pantothenic acid, selenium, iodine, folate
<b>Fish</b>	All fish: Protein, iodine. Fish bones: calcium, phosphorus, fluoride. Fatty fish: Protein, fat, vitamins A and D, omega-3 fatty acids
<b>Shellfish</b>	Similar nutrients to white fish. Crab and mussels: Protein and good sources of omega 3 Selenium, zinc, iodine and copper
<b>Molluscs</b>	Varying amounts of protein (scallop), calcium (clam), zinc (oysters) and iron (clam)
<b>Wheat</b>	Carbohydrate, Protein Fibre, thiamine, riboflavin, niacin, calcium, iron, folate if fortified
<b>Peanut</b>	Protein, fats, Vitamin E, niacin, magnesium
<b>Soya</b>	Protein, Thiamine, riboflavin, pyridoxine, folate, calcium, phosphorus, magnesium, iron, zinc, fibre
<b>Lupin</b>	Negligible nutritional value when consumed as a condiment or taken in very small amounts
<b>Tree nuts</b>	Depends on type of nut, but similar to peanuts + omega 3 and 6 fatty acids
<b>Sesame seed</b>	Protein, fats, Vitamin E, calcium, potassium, phosphorus, vitamin B and iron and omega 6 fatty acids
<b>Mustard</b>	Negligible nutritional value when consumed as a condiment or taken in very small amounts
<b>Celery/celeriac</b>	Fibre

and give expert advice to ensure a healthy, regular and varied diet is taken as this has been shown to be of importance in the development of allergy [114,115] It is important that the user take into account the important role of under-nutrition in paediatric and adult food allergy since poor nutritional status may aggravate symptoms and a healthy diet could be important in the prevention of allergic disease [10,116,117].

#### **Amber stage - interpretation of history**

This stage involves linking the answers to the questions from the 'red' section and includes interpretation of symptoms, medical history, trigger foods and nutritional issues. Typical symptoms and foods associated with IgE mediated FA are listed in the left hand column of this section, and adapted to differences in age-related onset. The right-hand column lists foods and symptoms most associated with adverse food reactions that are not immune-mediated. In general, paediatric patients are far more likely to suffer from non-IgE mediated conditions than non-immune mediated conditions [24]. Thus the paediatric tool also contains a middle column listing foods and symptoms most associated with paediatric non-IgE mediated FA. However, this middle column is not present on the adult tool since these conditions are often most prevalent in childhood [24], the exception being EoE, which does exist in adults, with the adult form usually being diagnosed between the ages of 30–50 years [119].

This section also contains a guide to the foods most likely to be causative of FA at different ages and to support the interpretation of reported food triggers. There is information on cross-reactivity, a PFS diagnostic algorithm [15] and lists of potential allergens in composite meals. Collectively, the information in the amber section enables the user to decide whether the patient has an IgE- or a non IgE-mediated FA, a non-immune mediated adverse food reaction or a differential diagnosis not related to allergy, or combined symptoms.

#### **Green stage - tests and diagnosis**

This section contains a test algorithm and care plan to summarise the outcome. Once the green stage is completed, the user should have sufficient information to undertake further tests, or make an onward referral to specialist services as indicated.

#### **Conclusion**

The EAACI Food Allergy and Anaphylaxis Guidelines [1] emphasise the importance of taking a careful allergy-focussed history, and asking structured questions. These age-appropriate diet history tools, developed by a multi-professional international group of experts, bring together relevant factors, important in the aetiology of FA.

The authors acknowledge that all of the evidence submitted in support of the tools has not been systematically graded, however, this is the first time that all of the relevant questions in an allergy focussed diet history have been agreed, documented, referenced and linked in a systematic way to provide standardised practical tools. The paediatric and adult diet history tools can be adapted to country specific dietary norms, whilst ensuring a standardised approach and screening for nutritional status. The information gained is vital in the formulation of a provisional diagnosis.

These tools can be used by anyone who needs guidance in taking a diet and clinical history, being particularly useful for those who not working in a specialist allergy setting. An accurate history determines the need for allergy testing. Once a diagnosis is made, patients can be managed appropriately or be referred onwards to an allergy specialist physician and/or dietitian as required. Newly diagnosed food allergy is a growing burden for all ages, against a backdrop of complex dietary intake patterns, multiple co-factors, social complications and diverse presenting features. These tools provide a scaffold of questions on which the diagnosis of FA can be built.

#### **Additional files**

**Additional file 1: Allergy-Focussed Diet History Paediatric version.**  
**Additional file 2: Allergy-Focussed Diet History Adult version.**

#### **Competing interests**

The authors declare that they have no competing interests.

#### **Authors' contributions**

IS chaired the Task Force, wrote the initial tools and drafted the manuscript. CV participated in the Task Force, provided input into the tools, and helped to draft the manuscript. RM participated in Task Force and contributed to the final version of the paediatric tool. NdJ participated in Task Force and contributed to the final version of the adult tool. AF participated in the Task Force and contributed to the paediatric tool. MG participated in the Task Force and contributed to the paediatric tool. JOE participated in Task Force. AS participated in the Task Force. LD participated in the Task Force. BVB conceived the idea for the Task Force and greatly participated in the development of the tools. All authors read and approved the final manuscript.

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## References

- Muraro A, Werfel T, Hoffmann-Sommergruber K, Roberts G, Beyer K, Bindslev-Jensen C, et al. EAACI food allergy and anaphylaxis guidelines: diagnosis and management of food allergy. *Allergy*. 2014;69:1008–25.
- Venter C, Pereira B, Voigt K, Grundy J, Clayton CB, Higgins B, et al. Prevalence and cumulative incidence of food hypersensitivity in the first 3 years of life. *Allergy*. 2008;63:354–9.
- Eller E, Kjaer HF, Host A, Andersen KE, Bindslev-Jensen C. Food allergy and food sensitization in early childhood: results from the DARC cohort. *Allergy*. 2009;64:1023–9.
- Vierk KA, Koehler KM, Fein SB, Street DA. Prevalence of self-reported food allergy in American adults and use of food labels. *J Allergy Clin Immunol*. 2007;119:1504–10.
- Zuberbier T, Edenharter G, Worm M, Ehlers I, Reimann S, Hantke T, et al. Prevalence of adverse reactions to food in Germany – a population study. *Allergy*. 2004;59:338–45.
- Nwaru BI, Hickstein L, Panesar SS, Muraro A, Werfel T, Cardona V, et al. The epidemiology of food allergy in Europe: a systematic review and meta-analysis. *Allergy*. 2014;69:62–75.
- Patelis A, Gunnbjörnsdóttir M, Borres MP, Burney P, Gislason T, Torén K, et al. Natural history of perceived food hypersensitivity and IgE sensitisation to food allergens in a cohort of adults. *PLoS One*. 2014;10(9):e85333.
- Diesner SC, Untersmayr E, Pietschmann P, Jensen-Jarolim E. Food allergy: only a pediatric disease? *Gerontology*. 2011;57:28–32.
- Christie L, Hine RJ, Parker JG, Burks W. Food allergies in children affect nutrient intake and growth. *J Am Diet Assoc*. 2002;102:1648–51.
- Meyer R, De Koker C, Dziubak R, Venter C, Dominguez-Ortega G, Cutts R, et al. Malnutrition in children with food allergies in the UK. *J Hum Nutr Diet*. 2014;27:227–35.
- NICE clinical guideline 116 – Food allergy in children and young people. National Institute for Health and Clinical Excellence. Diagnosis and Assessment of Food Allergy in Children and Young People in Primary Care and Community Settings. London: National Institute for Health and Clinical Excellence; 2011. [www.nice.org.uk/guidance/CG116](http://www.nice.org.uk/guidance/CG116) guidelines.
- Boyce JA, Assa'ad A, Burks AW, Jones SM, Sampson HA, Wood RA, et al. NIAID-sponsored expert panel. Guidelines for the Diagnosis and Management of Food Allergy in the United States: Summary of the NIAID-Sponsored Expert Panel Report. *J Allergy Clin Immunol*. 2010;126:1105–18.
- Burks AW, Tang M, Sicherer S, Muraro A, Eigenmann PA, Ebisawa M, et al. ICON: food allergy. *J Allergy Clin Immunol*. 2012;129:906–20.
- Venter C, Arshad SH. Guideline fever: an overview of DRACMA, US NIAID and UK NICE guidelines. *Curr Opin Allergy Clin Immunol*. 2012;12:302–15.
- Skypala IJ, Calderon MA, Leeds AR, Emery P, Till SJ, Durham SR. Development and validation of a structured questionnaire for the diagnosis of Oral Allergy Syndrome in subjects with seasonal allergic rhinitis during the UK birch pollen season. *Clin Exp Allergy*. 2011;41:1001–11.
- Soares-Weiser K, Takwoingi Y, Panesar SS, Muraro A, Werfel T, Hoffmann-Sommergruber K, et al. The diagnosis of food allergy: a systematic review and meta-analysis. *Allergy*. 2014;69:76–86.
- Eriksson NE, Forman H, Svenonius E. Food hypersensitivity in patients with pollen allergy. *Allergy*. 1982;37:437–43.
- Osterballe M, Hansen TK, Mortz CG, Høst A, Bindslev-Jensen C. The prevalence of food hypersensitivity in an unselected population of children and adults. *Pediatr Allergy Immunol*. 2005;16:567–73.
- Pascal M, Muñoz-Cano R, Reina Z, Palacín A, Vilella R, Picado C, et al. Lipid transfer protein syndrome: clinical pattern, cofactor effect and profile of molecular sensitization to plant-foods and pollens. *Clin Exp Allergy*. 2012;42:1529–39.
- Hebling A, McCants ML, Musmand JJ, Schwartz HJ, Lehrer SB. Immunopathogenesis of fish allergy: identification of fish-allergic adults by skin test and radioallergen sorbent test. *Ann Allergy Asthma Immunol*. 1996;77:48–54.
- Ross MP, Ferguson M, Street D, Klontz K, Schroeder T, Luccioli S. Analysis of food-allergic and anaphylactic events in the National electronic injury surveillance system. *J Allergy Clin Immunol*. 2008;121:166–71.
- Pumphrey RS, Gowland MH. Further fatal allergic reactions to food in the United Kingdom, 1999–2006. *J Allergy Clin Immunol*. 2007;119:1018–9.
- Meyer R, Schwarz C, Shah N. A review on the diagnosis and management of food-induced gastrointestinal allergies. *Curr Allergy Clin Immunol*. 2012;25:1–8.
- Katz Y, Goldberg MR, Rajuan N, Cohen A, Leshno M. The prevalence and natural course of food protein-induced enterocolitis syndrome to cow's milk: a large-scale, prospective population-based study. *J Allergy Clin Immunol*. 2011;127:647–53.
- Spergel JM, Brown-Whitehorn TF, Beausoleil JL, Franciosi J, Shuker M, Verma R, et al. 14 years of eosinophilic esophagitis: clinical features and prognosis. *J Pediatr Gastroenterol Nutr*. 2009;48:30–6.
- National Institute for Health and Clinical Excellence. Atopic Eczema in Children (CG57). [www.nice.org.uk/guidance/CG57](http://www.nice.org.uk/guidance/CG57). 2007.
- Werfel T, Ballmer-Weber B, Eigenmann PA, Niggemann B, Rancé F, Turjanmaa K, et al. Eczematous reactions to food in atopic eczema: position paper of the EAACI and GA2LEN. *Allergy*. 2007;62:723–8.
- Straumann A, Aceves SS, Blanchard C, Collins MH, Furuta GT, Hirano I, et al. Pediatric and adult eosinophilic esophagitis: similarities and differences. *Allergy*. 2012;67:477–90.
- Maintz L, Novak N. Histamine and histamine intolerance. *Am J Clin Nutr*. 2007;85:1185–96.
- Vally H, Misso NL. Adverse reactions to the sulphite additives. *Gastroenterol Hepatol Bed Bench*. 2012;5:16–23.
- Neri M, Laterza F, Howell S, Di Gioacchino M, Festi D, Ballone E, et al. Symptoms discriminate irritable bowel syndrome from organic gastrointestinal diseases and food allergy. *Eur J Gastroenterol Hepatol*. 2000;12:981–8.
- Feuille E, Nowak-Węgrzyn A. Definition, etiology, and diagnosis of food protein-induced enterocolitis syndrome. *Curr Opin Allergy Clin Immunol*. 2014;14:222–8.
- Romano A, Scala E, Rumi G, Gaeta F, Caruso C, Alonzi C. Lipid transfer proteins: the most frequent sensitizer in Italian subjects with food-dependent exercise-induced anaphylaxis. *Clin Exp Allergy*. 2012;42:1643–53.
- Commins SP, James HR, Stevens W, Pochan SL, Land MH, King C, et al. Delayed clinical and ex vivo response to mammalian meat in patients with IgE to galactose-alpha-1,3-galactose. *J Allergy Clin Immunol*. 2014;134:108–15.
- Raithel M, Weidenhiller M, Hagel AF, Hetterich U, Neurath MF, Konturek PC. The malabsorption of commonly occurring mono and disaccharides. *Dtsch Arztebl Int*. 2013;110:775–82.
- Schäfer T, Böhrer E, Ruhdorfer S, Weigl L, Wessner D, Heinrich J, et al. Epidemiology of food allergy/food intolerance in adults: associations with other manifestations of atopy. *Allergy*. 2001;56:1172–9.
- Skypala IJ, Bull S, Deegan K, Gruffydd-Jones K, Holmes S, Small I, et al. The prevalence of pollen-food syndrome (PFS) and prevalence and characteristics of reported food allergy; a survey of UK adults aged 18–75 incorporating a validated PFS diagnostic questionnaire. *Clin Exp Allergy*. 2013;43:928–40.
- Young E, Stoneham MD, Petuckevitch A, Barton J, Rana R. A population study of food intolerance. *Lancet*. 1994;343:1127–30.
- Berns SH, Halm EA, Sampson HA, Sicherer SH, Busse PJ, Wisnivesky JP. Food allergy as a risk factor for asthma morbidity in adults. *J Asthma*. 2007;44:377–81.
- Linneberg A, Gislum M, Johansen N, Husemoen LL, Jørgensen. Temporal trends of aeroallergen sensitization over twenty-five years. *Clin Exp Allergy*. 2007;37:1137–42.
- Wüthrich B, Schmid-Grendelmeier P, Schindler C, Imboden M, Bircher A, Zemp E, et al. Prevalence of atopy and respiratory allergic diseases in the elderly SAPALDIA population. *Int Arch Allergy Immunol*. 2013;162:143–8.
- Newson RB, van Ree R, Forsberg B, Janson C, Lötvall J, Dahlén SE, et al. Geographical variation in the prevalence of sensitization to common aeroallergens in adults: the GA(2) LEN survey. *Allergy*. 2014;69:643–51.
- Wagner S, Breiteneder H. The latex-fruit syndrome. *Biochem Soc Tans*. 2002;30:935–40.
- Boquete M, Iraola V, Morales M, Pinto H, Francisco C, Carballás C, et al. Seafood hypersensitivity in mite sensitized individuals: is tropomyosin the only responsible allergen? *Ann Allergy Asthma Immunol*. 2011;106:223–9.



45. Szépfalusi Z, Ebner C, Pandjaitan R, Orlicek F, Scheiner O, Boltz-Nitulescu G, et al. Egg yolk  $\alpha$ -livet in (chicken serum albumin) is a cross-reactive allergen in the bird-egg syndrome. *J Allergy Clin Immunol*. 1994;93:932–42.
46. Hilger C, Kohnen M, Grigioni F, Lehnert C, Hentges F. Allergic cross-reactions between cat and pig serum albumin. Study at the protein and DNA levels. *Allergy*. 1997;52:179–88.
47. Sánchez-Borges M, Suárez-Chacón R, Capriles-Hulett A, Caballero-Fonseca F, Fernández-Caldas E. Anaphylaxis from ingestion of mites: pancake anaphylaxis. *J Allergy Clin Immunol*. 2013;131:31–5.
48. Nieuwenhuizen N, Lopata AL, Jeebhay MF, Herbert De'B R, Robins TC, Brombacher F. Exposure to the fish parasite *Anisakis* causes allergic airway hyper reactivity and dermatitis. *J Allergy Clin Immunol*. 2006;117:1098–105.
49. Cardona V, Luengo O, Garriga T, Labrador-Horrillo M, Sala-Cunill A, Izquierdo A, et al. Co-factor-enhanced food allergy. *Allergy*. 2012;67:1316–8.
50. Shadick NA, Laing MH, Partridge AJ, Bingham C, Wright E, Fossel AH, et al. The natural history of food-dependant exercise-induced anaphylaxis: survey results from 10-year follow-up study. *J Allergy Clin Immunol*. 1999;104:123–7.
51. Romano A, Di Fonso M, Guiffreda F, Papa G, Artesani MC, Viola M. Food-dependent exercise-induced anaphylaxis: clinical and laboratory findings in 54 subjects. *Int Arch Allergy Immunol*. 2001;25:264–72.
52. Matsuo H, Dahlström J, Tanaka A, Kohno K, Takahashi H, Furumura M, et al. Sensitivity and specificity of recombinant x-5 gliadin-specific IgE measurement for the diagnosis of wheat-dependent exercise-induced anaphylaxis. *Allergy*. 2008;63:233–6.
53. Hompes S, Dölle S, Grünhagen J, Grabenhenrich L, Worm M. Elicitors and co-factors in food-induced anaphylaxis in adults. *Clin Trans Allergy*. 2013;3:38.
54. Asero R, Mistrello G, Roncarolo D, Amato S, van-Ree R. A case of allergy to beer showing cross-reactivity between lipid transfer proteins. *Ann Allergy Asthma Immunol*. 2001;87:65–7.
55. Kalogeromitros DC, Makris MP, Gregoriou SG, Mousatou VG, Lyras NG, Tarassi KE, et al. Grape anaphylaxis: a study of 11 adult onset cases. *Allergy Asthma Proc*. 2005;26:53–8.
56. Agarwal DP, Harada S, Goedde HW. Racial differences in biological sensitivity to ethanol: the role of alcohol dehydrogenase and aldehyde dehydrogenase isozymes. *Alcohol Clin Exp Res*. 1981;5:12–6.
57. du Toit G. Food-dependent exercise-induced anaphylaxis in childhood. *Pediatr Allergy Immunol*. 2007;18:455–63.
58. James JM, Crespo JF. Allergic reactions to foods by inhalation. *Curr Allergy Asthma Rep*. 2007;7:167–74.
59. Lehrer SB, Kim L, Rice T, Saidu J, Bell J, Martin R. Transfer of shrimp allergens to other foods through cooking oil. *J Allergy Clin Immunol*. 2007;119:S112.
60. Eriksson NE, Moller C, Werner S, Magnusson J, Bengtsson U. The hazards of kissing when you are food allergic. *J Invest Allergol Clin Immunol*. 2003;13:149–54.
61. Vetander M, Helander D, Flodström C, Ostblom E, Alfvén T, Ly DH, et al. Anaphylaxis and reactions to foods in children—a population-based case study of emergency department visits. *Clin Exp Allergy*. 2012;42:568–77.
62. Wood RA, Sicherer SH, Vickery BP, Jones SM, Liu AH, Fleischer DM, et al. The natural history of milk allergy in an observational cohort. *J Allergy Clin Immunol*. 2013;131:805–12.
63. Sicherer SH, Wood RA, Vickery BP, Jones SM, Liu AH, Fleischer DM, et al. The natural history of egg allergy in an observational cohort. *J Allergy Clin Immunol*. 2014;133:492–9.
64. Kotaniemi-Syrjänen A, Palosuo K, Jartti T, Kuitunen M, Pelkonen AS, Makela MJ. The prognosis of wheat hypersensitivity in children. *Pediatr Allergy Immunol*. 2010;21:e421–6.
65. Savage JH, Kaeding AJ, Matsui EC, Wood RA. The natural history of soy allergy. *J Allergy Clin Immunol*. 2010;125:683–6.
66. Mittag D, Veiths S, Vogel L, Becker W-M, Rhis H-P, Hebling A, et al. Soybean allergy in patients allergic to birch pollen: clinical investigation and molecular characterisation of allergens. *J Allergy Clin Immunol*. 2004;113:148–54.
67. Skolnick HS, Conover-Walker MK, Koerner CB, Sampson HA, Wesley Burks W, Wood RA. The natural history of peanut allergy. *J Allergy Clin Immunol*. 2001;107:367–74.
68. Fleischer D, Conover-Walker M, Matsui E, Wood R. The natural history of tree nut allergy. *J Allergy Clin Immunol*. 2005;116:1087–93.
69. Mullins RJ, Dear KB, Tang ML. Characteristics of childhood peanut allergy in the Australian capital territory, 1995 to 2007. *J Allergy Clin Immunol*. 2009;123:689–93.
70. Peeters KA, Koppelman SJ, Penninks AH, Lebens A, Bruijnzeel-Koomen CA, Hefle SL, et al. Clinical relevance of sensitization to lupine in peanut-sensitized adults. *Allergy*. 2009;64:549–55.
71. de Jong NW, van Maaren MS, Vlieg-Boersta BJ, Dubois AEJ, de Groot H, Gerth van Wijk R. Sensitization to lupine flour: is it clinically relevant? *Clin Exp Allergy*. 2010;40:1571–7.
72. Dalal I, Goldberg M, Katz Y. Sesame seed food allergy. *Curr Allergy Asthma Rep*. 2012;12:339–45.
73. Morisset M, Moneret-Vautrin D-A, Maadi F, Fremont S, Gunard L, Croizier A, et al. Prospective study of mustard allergy: first study with double-blind placebo-controlled food challenge trials (24 cases). *Allergy*. 2003;58:295–9.
74. Gaspolo IN, de Leon MP, Prickett SR, O'Hehir RE, Rolland JM. Clinical allergy to hazelnut and peanut: identification of T cell cross-reactive allergens. *Int Arch Allergy Immunol*. 2011;155:345–54.
75. Maleki SJ, Teuber SS, Cheng H, Chen D, Comstock SS, Ruan S, et al. Computationally predicted IgE epitopes of walnut allergens contribute to cross-reactivity with peanuts. *Allergy*. 2011;66:1522–9.
76. Stutius LM, Sheehan WJ, Rangsitienchai P, Bharmanee A, Scott JE, Young MC, et al. Characterizing the relationship between sesame, coconut, and nut allergy in children. *Pediatr Allergy Immunol*. 2010;21:1114–8.
77. Barre A, Sordet C, Culerrier R, Rancé F, Didier A, Rougé P. Vicilin allergens of peanut and tree nuts (walnut, hazelnut and cashew nut) share structurally related IgE-binding epitopes. *Mol Immunol*. 2008;45:1231–40.
78. de Leon MP, Drew AC, Gaspolo IN, Suphioglu C, O'Hehir RE, Rolland JM. IgE cross-reactivity between the major peanut allergen Ara h 2 and tree nut allergens. *Mol Immunol*. 2007;44:463–71.
79. Wallowitz ML, Chen RJ, Tzen JT, Teuber SS. Ses i 6, the sesame 11S globulin, can activate basophils and shows cross-reactivity with walnut in vitro. *Clin Exp Allergy*. 2007;37:929–38.
80. Turner P, Ng I, Kemp A, Campbell D. Seafood allergy in children: a descriptive study. *Ann Allergy Asthma Immunol*. 2011;106:494–501.
81. Sicherer SH, Munoz-Furlong A, Sampson HA. Prevalence of seafood allergy in the United States determined by a random telephone survey. *J Allergy Clin Immunol*. 2004;114:159–65.
82. Thong BY, Cheng YK, Leong KP, Tang CY, Chng HH. Anaphylaxis in adults referred to a clinical immunology/allergy centre in Singapore. *Singapore Med J*. 2005;46:529–34.
83. Sahabudin S, Misnan R, Yazir ZH, Mohamad J, Abdullah N, Bakhtiar F, et al. Identification of major and minor allergens of black tiger prawn (*Penaeus monodon*) and King Prawn (*Penaeus latisulcatus*). *Malays J Med Sci*. 2011;18:27–32.
84. du Toit G, Santos A, Roberts G, Fox AT, Smith P, Lack G. The diagnosis of IgE-mediated food allergy in childhood. *Pediatr Allergy Immunol*. 2009;20:309–19.
85. Ortolani C, Ispano M, Pastorello E, Bigi A, Ansaloni R. The oral allergy syndrome. *Ann Allergy*. 1988;61:47–52.
86. Mortz CG, Andersen KE, Bindslev-Jensen C. Prevalence of peanut sensitization and the association to pollen sensitization in a cohort of unselected adolescents – The Odense Adolescence Cohort Study on Atopic Diseases and Dermatitis (TOACS). *Pediatr Allergy Immunol*. 2005;16:501–6.
87. De Swert LF, Gadisseur R, Sjölander S, Raes M, Leus J, Van Hoeyveld E. Secondary soy allergy in children with birch pollen allergy may cause both chronic and acute symptoms. *Pediatr Allergy Immunol*. 2012;23:117–23.
88. Fernández-Rivas M, González-Mancebo E, Rodríguez-Pérez R, Benito C, Sánchez-Monge R, Salcedo G, et al. Clinically relevant peach allergy is related to peach lipid transfer protein, Pru p 3, in the Spanish population. *J Allergy Clin Immunol*. 2003;112:789–95.
89. Javaloyes G, Goikoetxea MJ, García Nuñez I, Aranda A, Sanz ML, Blanca M, et al. Pru p 3 acts as a strong sensitizer for peanut allergy in Spain. *J Allergy Clin Immunol*. 2012;130:1432–4.
90. Salcedo G, Sanchez-Monge R, Diaz-Perales A, Garcia-Casado G, Barber D. Plant non-specific lipid transfer proteins as food and pollen allergens. *Clin Exp Allergy*. 2004;34:1336–41.
91. Meyer R, Fleming C, Dominguez-Ortega G, Lindley K, Michaelis L, Thapar N, et al. Manifestation of gastrointestinal food allergies presenting to a single tertiary paediatric gastroenterology unit. *World Allergy Organ J*. 2013;6:13.
92. Heine RG. Pathophysiology, diagnosis and treatment of food protein-induced gastrointestinal diseases. *Curr Opin Allergy Clin Immunol*. 2004;4:221–9.

93. Leonard SA, Nowak-Węgrzyn A. Food protein-induced enterocolitis syndrome: an update on natural history and review of management. *Ann Allergy Asthma Immunol.* 2011;107:95–101.
94. Bohm M, Richter JE. Treatment of eosinophilic esophagitis: overview, current limitations, and future direction. *Am J Gastroenterol.* 2008;103:2635–44.
95. Spergel JM, Brown-Whitehorn TF, Cianferoni A, Shuker M, Wang ML, Verma R, et al. Identification of causative foods in children with eosinophilic esophagitis treated with an elimination diet. *J Allergy Clin Immunol.* 2012;130:461–7.
96. Lucendo AJ, Arias Á, González-Cervera J, Yagüe-Compadre JL, Guagnozzi D, Angueira T, et al. Empiric 6-food elimination diet induced and maintained prolonged remission in patients with adult eosinophilic esophagitis: a prospective study on the food cause of the disease. *J Allergy Clin Immunol.* 2013;131:797–804.
97. Monsbakken KW, Vandvik PO, Farup PG. Perceived food intolerance in subjects with irritable bowel syndrome – aetiology, prevalence and consequences. *Eur J Clin Nutr.* 2006;60:667–72.
98. Wuthrich B, Schmid A, Walther B, Seiber R. Milk consumption does not lead to mucus production or occurrence of asthma. *J Am Coll Nutr.* 2005;24:547S–55.
99. Hin H, Bird G, Fisher P, Mahy N, Jewell D. Coeliac disease in primary care: case finding study. *BMJ.* 1999;318:164–7.
100. Catassi C, Bai JC, Bonaz B, Bouma G, Calabrò A, Carroccio A, et al. Non-Celiac Gluten sensitivity: the new frontier of gluten related disorders. *Nutrients.* 2013;5:3839–53.
101. NICE Clinical Guideline. Irritable Bowel Syndrome in Adults: Diagnosis and Management Of Irritable Bowel Syndrome In Primary Care. 2008.
102. Gibson PR, Shepherd SJ. Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J Gastroenterol Hepatol.* 2010;25:252–8.
103. Barrett JS, Irving PM, Shepherd SJ, Muir JG, Gibson PR. Comparison of the prevalence of fructose and lactose malabsorption across chronic intestinal disorders. *Aliment Pharmacol Ther.* 2009;30:165–74.
104. Di Lorenzo G, Pacor ML, Mansueto P, Martinelli N, Esposito-Pellitteri M, Lo Bianco C, et al. Food additive-induced urticaria: a survey of 838 patients with recurrent chronic idiopathic urticaria. *Int Arch Allergy Immunol.* 2005;138:235–42.
105. Mitchell J, Skypala I. Aspirin and salicylate in respiratory disease. *Rhinology.* 2013;51:195–205.
106. Nowak-Węgrzyn A, Bloom KA, Sicherer SH, Shreffler WG, Noone S, Wanich N, et al. Tolerance to extensively heated milk in children with cow's milk allergy. *J Allergy Clin Immunol.* 2008;122:342–7.
107. Lemon-Mule H, Sampson HA, Sicherer SH, Shreffler WG, Noone S, Nowak-Węgrzyn A. Immunologic changes in children with egg allergy ingesting extensively heated egg. *J Allergy Clin Immunol.* 2008;122:977–83.
108. Vlieg-Boerstra BJ, van der Heide S, Bijleveld CM, Kukler J, Duiverman EJ, Wolt-Plompen SA, et al. Dietary assessment in children adhering to a food allergen avoidance diet for allergy prevention. *Eur J Clin Nutr.* 2006;60:1384–90.
109. Groetch M, Nowak-Węgrzyn A. Practical approach to nutrition and dietary intervention in pediatric food allergy. *Pediatr Allergy Immunol.* 2013;24:212–21.
110. Flammarión S, Santos C, Guimber D, Jouannic L, Thumerelle C, Gottrand F, et al. Diet and nutritional status of children with food allergies. *Pediatr Allergy Immunol.* 2011;22:161–5.
111. Skypala I. Adverse food reactions—an emerging issue for adults. *J Am Diet Assoc.* 2011;111:1877–91.
112. Mofidi S. Nutritional management of pediatric food hypersensitivity. *Pediatrics.* 2003;111:1645–53.
113. Venter C, Laitinen K, Vlieg-Boerstra B. Nutritional aspects in diagnosis and management of food hypersensitivity—the dietitians role. *J Allergy (Cairo).* 2012;2012:269376.
114. Grimshaw KE, Maskell J, Oliver EM, Morris RC, Foote KD, Mills EN, et al. Diet and food allergy development during infancy: birth cohort study findings using prospective food diary data. *J Allergy Clin Immunol.* 2014;133:511–9.
115. Roduit C, Frei R, Depner M, Schaub B, Loss G, Genuneit J, et al. Increased food diversity in the first year of life is inversely associated with allergic diseases. *J Allergy Clin Immunol.* 2014;133:1056–64.
116. Meyer R, Venter C, Fox AT, Shah N. Practical dietary management of protein energy malnutrition in young children with cow's milk protein allergy. *Pediatr Allergy Immunol.* 2012;23:307–14.
117. Skypala I, Vlieg-Boerstra B. Food intolerance and allergy: increased incidence or contemporary inadequate diets? *Curr Opin Clin Nutr Metab Care.* 2014;17:442–7.
118. Venter C, Meyer R. Session 1: allergic disease: the challenges of managing food hypersensitivity. *Proc Nutr Soc.* 2010;69:11–24.
119. Hruz P. Epidemiology of eosinophilic esophagitis. *Dig Dis.* 2014;32:40–7.

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