



# Proceedings of EAACI / GA<sup>2</sup>LEN Food Allergy Training Course

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REPORT BY DR A J MORRIS



THE UCB INSTITUTE OF ALLERGY

Join our forces against allergy

Expert panel of food allergists included:

Carsten Bindslev-Jensen, Philippe Eigenmann, Bodo Niggemann, Arne Host, Fabienne Rance, Jonathan Hourihane, Ronald van Ree, Antonella Muraro, Andre Knulst, Barbara Ballmer-Weber, Lars Poulsen, Susanne Halken, Torsten Zuberbier, Kristina Turjanmaa and staff of Allergy Center, Odense University Hospital.

**REPORT BY DR A J MORRIS**



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## What is True Food Allergy?

True Food Allergy is an immediate immune-mediated hypersensitivity reaction resulting in histamine release with tissue inflammation which occurs after exposure to common food proteins in the diet.

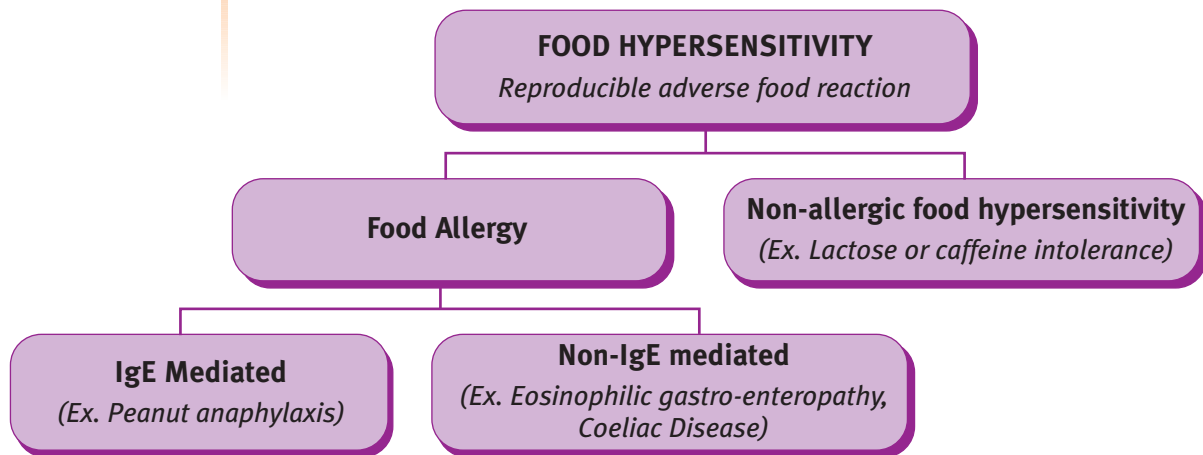


Figure 1: Current Nomenclature for adverse food reactions (WAO & EAACI)

## INCIDENCE OF FOOD ALLERGY

*Dr. Muraro*

Current data suggest that true food allergy affects 6-8% of infants, 3-5% of young children and 2-4% of adults.

In the last 20 years the incidence of food allergy has rapidly increased. Nut allergies, first documented in the early 1980's, are now very common, whilst the incidence of cow's milk allergy seems to be stabilising.

As our eating habits change we become exposed to more exotic foods; consequently the incidence and range of food allergy increases. For example we have seen a twenty fold increase in allergy to Kiwi fruit since this allergy was first recorded in 1981.

Food allergy is an important cause of anaphylaxis. In the European Union, half of all reported anaphylactic reactions are now due to food allergies.

Food colorant and additive induced reactions tend to be non-IgE mediated (via unknown mechanisms), and affect between 0.03 and 0.2% of general population (and 2% of atopic individuals).

## WHAT HAPPENS IN THE FOOD ALLERGIC REACTION?

*Dr Poulsen*

True food allergy develops in two steps:

First exposure to the allergenic food protein leads to the development of primary allergic sensitization. Production of food specific IgE antibodies then occurs with resultant immune memory.

On subsequent exposure, food allergens cross-link IgE antibodies bound on the tissue mast cells resulting in mast cell degranulation. Mast cells release inflammatory preformed mediators (such as histamine, tryptase, chymase, heparin) and newly synthesized mediators (leukotrienes). This "early phase" allergic reaction results in increased vasodilation and vascular leakage with erythema, oedema and excess mucus production. As the "inflammatory cascade" develops, eosinophils are attracted into the site of the allergic reaction where they release preformed granular mediators - Eosinophil Cationic Protein (ECP), Major Basic Protein (MBP), etc. – and also newly formed inflammatory mediators, leukotrienes and pro-inflammatory cytokines. This promotes persistent tissue inflammation called the "late phase" allergic reaction which develops 2 to 24 hours after allergen exposure.

High IgE responders (those individuals who produce high levels of food specific IgE) seem to have more persistent food allergies, while low IgE responders (those with only slightly raised food specific IgE) tend to have transient food allergies that are often "outgrown" with age.



*Dr Ballmer-Weber*

Some investigators further divide Food Allergy into Class 1 and Class 2 food allergic responses.

Class 1 responses result primarily via gastro-intestinal food sensitisation, predominantly in infants with more robust initial allergic reactions. Many of these food sensitizations are out-grown in early childhood (cow's milk, egg, soy, wheat).

In contrast, Class 2 food allergic reactions are initiated by respiratory sensitisation to common inhaled pollen allergens which cross-react with food allergens. These reactions may be less intense and tend to develop in older children and young adults. Class 2 food allergic reactions due to respiratory pollen sensitisation or so-called Pollen-Food Syndrome (PFS) with resultant food cross-reactivity tend to persist and are not readily out-grown. A good example is the Oral Allergy Syndrome (OAS) with Birch Pollen sensitisation resulting in localised oral reactions to common vegetables, stone fruit and nuts.



The pattern of allergic sensitisation is very regional and certain food allergens tend to predominantly cause problems in specific geographic areas. For example, peanut is the predominant food allergen in the USA and UK; egg is the primary food allergen in France, seafood in Australia and Spain, celery in Switzerland, poppy seed in Poland while “birds nest” soup is the commonest food allergy in Thailand.

As new ingredients are farmed and introduced into everyday staple foods so patterns of food allergic sensitisation are changing. For example, Lupin flour is now mass produced and added to many baked products particularly in France. Lupin cross-reacts with peanut allergy, and has resulted in an increased incidence of reported Lupin flour anaphylaxis (16% of peanut allergic individuals are lupin flour allergic).

## CLINICAL MANIFESTATIONS OF FOOD ALLERGY

*Dr Muraro*

Food allergy may present with varying intensity, from mild oral itching to full blown anaphylaxis with swelling, respiratory obstruction and circulatory collapse, all within minutes of ingesting the offending food. At the mild end of the spectrum, we encounter the Oral Allergy Syndrome, seen mainly in pollen allergic individuals with intense oral itching, swelling of lips and mucosa and some palatal itch which resolve spontaneously and rarely lead to any more severe symptoms. In typical food allergy the following organ systems are affected to a lesser or greater extent depending on sensitivity:

- **Oro-pharynx:** Lip and oral itching, tongue and glottic oedema, laryngeal obstruction.
- **Skin:** Acute urticaria, angioedema or atopic dermatitis.
- **Respiratory:** Asthma. Rhino-conjunctivitis
- **Gastro-intestinal:** Vomiting, gastritis, diarrhoea, acute abdominal pain.
- **Circulatory:** Sudden hypotension.
- **Anaphylactic reaction:** Multi-system involvement with circulatory collapse and eventually death.

Some individuals are so exquisitely sensitive that they even react to inhaled or skin exposure from food allergen vapours such as fish being boiled, nuts being cooked or raw potatoes being peeled in the home or restaurant. Kiss Induced Allergy (KIA) is also a common but often overlooked source of allergen exposure. Food allergy is strongly related to atopy and usually co-exists with other inhalant allergies; also polysensitization to multiple food allergens is frequent - approximately 30% of food allergic individuals will have other additional food allergies.





## Compounding factors in food allergy

Certain physical factors may enhance or amplify a food allergic reaction. For example an individual may have no symptoms if he eats a specific food, but if he engages in certain activities or takes certain medication at the same time as eating the food then he develops an allergic reaction.

A typical example is food-dependent exercise-induced acute urticaria, asthma or anaphylaxis (FDEIA). These persons are symptom-free if they eat certain foods such as wheat, celery or shellfish or if they exercise without eating. But if they eat one of these foods and exercise shortly afterwards (within 4 hours) they will experience typical food allergic symptoms of urticaria, asthma or anaphylaxis. This may also occur as a non-specific phenomenon after ingesting any foodstuff that causes gastric dilatation before exercising.

Alcoholic drinks speed gastric emptying, cause vasodilatation and aid rapid absorption of food allergens which will make underlying food allergies more severe and of rapid onset.

Antacids and proton pump inhibitor medications such as omeprazole and lansoprazole which reduce gastric acidity result in food allergens passing intact to gut lymphoid tissue in a more allergenic form. This can result in unexplained episodic food allergic reactions not seen at other times of normal gastric pH.

Other factors that promote the occurrence of a food allergic reaction: concomitant viral illness, certain times of the menstrual cycle and pollen-related food hypersensitivity (which raises problems during peak tree or grass pollen season).

Patients with chronic persistent asthma, and concomitant food allergies, have a far greater risk for severe food-related asthma exacerbations or even asthma related death after specific food exposure compared with patients manifesting only food allergy. Heiners syndrome is a rare condition of acute pulmonary haemosiderosis, recurrent pneumonia and eosinophilia which is related to a delayed hypersensitivity to cow's milk proteins.

## WHY DO WE DEVELOP FOOD ALLERGIES?

*Dr Poulsen*

Certain individuals have a familial or genetic predisposition to develop allergies which is termed "atopy". Initially it was hoped that genetic research in allergy will find the magical "allergy gene". However, after many years of research it becomes evident that this "genetic predisposition" to allergy is carried on multiple candidate genes, rather than being limited to a few specific chromosomal loci.

Breast feeding seems to be protective of allergy, and the incidence of cow's milk allergy has been steadily declining with increasing breast feeding practices (in Denmark cow's milk allergy has declined from 2.2% in 1985 to 1.0% in 1999). This is possibly due to protective secretory IgA antibodies and prebiotic oligosaccharides in breast milk which promote the growth of "protective" gut commensal bacteria. Although it is suggested that at risk mothers should avoid allergenic foods in late pregnancy and during breast feeding, no firm evidence for this practice has been forthcoming. Despite rigid avoidance of food allergens in the maternal diet, traces of food allergens are still found in breast milk and the pregnant mother's circulation.

However exclusive breastfeeding to at least 4 to 6 months is protective, as is avoidance of cigarette smoking and only introducing solid foods after 4 to 6 months of age.

During infancy, gastric acidity is not well maintained due to immature acid secretion. This lapse in gastric acidity and pepsin digestion may allow allergens to pass intact to the gut lymphoid tissue and promote food allergic sensitisation in infancy. Gastro-intestinal commensal flora including Lactobacilli (bifido-bacteria) seem to enhance gut immunity and studies show a reduced incidence of atopic dermatitis in infants who have "probiotic" lactobacilli supplements. But beware in cow's milk allergic infants that cow's milk proteins may contaminate the commercial probiotic supplements.

There is mounting evidence that "trace" exposure to food allergens in infancy promotes allergic sensitisation while high dose allergen exposure in infancy induces tolerance by switching T helper cells from Th2 to Th1 subtypes. Studies are underway to clarify whether we should in fact be promoting allergen "bombardment" in infancy and not avoidance! The Hygiene (or Microbial) Hypothesis is certainly highlighted in studies that indicate atopic children living on live stock farms and exposed early to animal bacterial endotoxins are less likely to develop inhalant allergies and food allergic sensitisation.

General trends in childhood dietary practices show that we are eating far more exotic and previously unknown foods than ever before. This is mirrored by more and more reports of allergic sensitization to these exotic foods such as kiwi fruit, Sharon fruit, lupin flour, nuts and shellfish.

## WHAT ARE THE PROBLEM FOODS?

*Dr Zuberbier and Dr van Ree*

In infancy, a finite number of foods are the principal culprits causing food allergic sensitisation. Over 90% of food allergies in infancy are due to cow's milk, hen's egg, wheat flour, soy milk, cod fish and peanut (Class 1 allergens). In older children and young adults the range of allergenic foods

is more extensive: we see allergy to tree nuts (Brazil nut, hazel nut, cashew, walnut and almond), sesame, shellfish (shrimp, mussel), stone fruits (apple, cherry, plum) and exotic vegetables and fruits (kiwi, avocado, Ethiopian eggplant).

FOOD ALLERGEN	CROSS-REACTING FOOD	CLINICAL SIGNIFICANCE
<b>Peanuts</b>	Other legumes (soybean, green peas, lupin flour etc.)	Generally mild reactions except lupin flour which can cause anaphylaxis
<b>Tree nuts</b>	Other tree nuts (Brazil nut, hazelnut, cashew, walnut, almond, coconut, pine nut)	Urticaria, angioedema, potential anaphylaxis, Oral Allergy Syndrome (OAS)
<b>Shrimps</b>	Other crab, lobster, mussel, clams etc	Urticaria, angioedema, potential anaphylaxis
<b>Wheat</b>	Barley, Rye	Urticaria, angioedema, potential anaphylaxis, FDEIA
<b>Salmon</b>	Swordfish, Sole	Urticaria, angioedema, potential anaphylaxis
<b>Cow's Milk</b>	Goats Milk, Beef meat	Urticaria, angioedema, potential anaphylaxis
<b>Latex products</b>	Banana, kiwi, avocado, chestnut, potato, pitted fruits	OAS, urticaria, angioedema, potential anaphylaxis
<b>Grass pollen</b>	Raw tomato, wheat	OAS
<b>Birch pollen</b>	Apple, hazelnut, celery, carrot, kiwi, raw potatoes. An increasing number of foods are associated with cross reactivity and include; avocado, banana, chestnut, tomato, bell pepper and chick pea.	OAS
<b>Ragweed pollen</b>	Watermelon, honeydew melon, cantaloupe, banana	OAS
<b>Mugwort pollen</b>	Celery, Mustard, Spice	OAS

Table 1: Examples of cross-reacting food allergens that can cause food-induced allergic reactions

Cross-reactivity between pollens and foods occurs in older children who are pollen sensitised. These children develop primarily allergic rhino-conjunctivitis to tree and grass pollen and then later develop associated food allergies (table 1). A panallergen protein called Profilin found in both pollen and fruit causes the phenomenon of Oral Allergy Syndrome (OAS). Bet v 1, one of the Profilin panallergens found in Silver Birch pollen causes hay fever and can cross-react with nuts and legumes, while another profilin Bet v 2 cross-reacts with uncooked stone fruits and vegetables. This OAS which results in localised oral irritation but non-life threatening reactions occurs predominantly in northern European populations (Scandinavia). Curiously in southern Europe, where Silver Birch pollen allergy is uncommon, panallergen sensitisation to fruits is more related to the heat-stable Lipid Transfer Protein (LTP) fraction. In the southern European populations (Spain, Portugal, and Italy) LTP sensitisation leads to more severe oral allergic symptoms and even anaphylaxis to fruits, nuts and vegetables and there is usually no associated pollen-food allergy.

Interestingly, food farming and storage practices can induce the fruits to produce more of these “stress-induced” panallergens and render the food more allergenic.

Some panallergens (such as the Profilin Bet v 1) are heat labile. Bet v 1 has a conformational epitope (the IgE recognition

site results from folding of the protein chain which brings in close proximity molecules that are situated remote from each other when the chain is unfolded) (Fig. 3). This conformation or folding is damaged by heating. Consequently cooking, processing or canning of the fruits bearing Profilin Bet v 1 renders the allergen non-allergenic.

The LTP panallergen is a linear isotope with IgE recognising an unfolded site on the allergen protein (Fig. 3); hence heating does not alter allergenicity. LTP is therefore heat and acid stable and cooking does not reduce allergenicity.

It is interesting to note that heat may occasionally increase a foodstuff’s allergenicity as occurs with peanut. The “raw” peanut has relatively low allergenicity, but roasting the peanut at very high temperature rapidly increases its allergenicity by altering the protein conformation much more than boiling at lower temperatures.

It should be also borne in mind that “safe” foods can become contaminated with traces of allergen in food processing. Occasionally people may have an allergic reaction to a food that they have safely eaten before, because a known allergen has inadvertently entered the food chain. For example Lupin flour may be added to wheat flour, sesame residue may occur in noodles and peanut residue may enter ice cream.

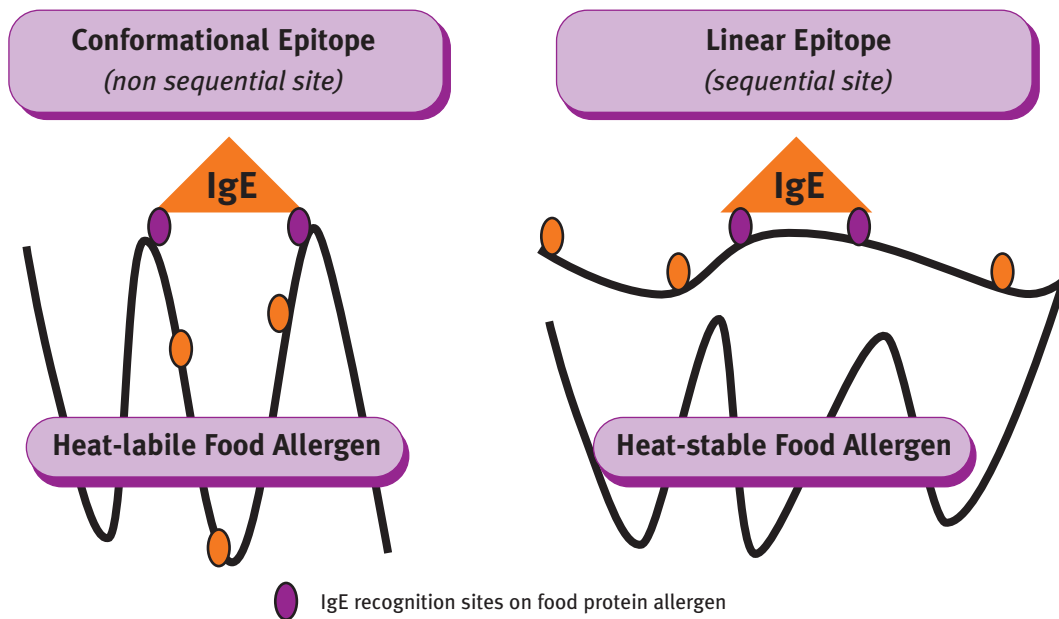


Figure 3: Conformational versus linear food allergen isotopes.

## FOOD ALLERGY DIAGNOSTIC TESTS:

### *The expert panel*

Food allergy testing involves screening for food specific IgE antibodies to common and exotic foods.

A full and extensive food allergy history is pivotal in guiding the physician to which foods require specific food allergy testing.

The Double Blind Placebo Controlled Food Challenge (DBPCFC) test remains the most accurate way of confirming a specific food allergy. However, food challenge tests are time consuming, need to be conducted in a hospital environment with full resuscitation equipment available and an experienced dietician is essential to “mask” the placebo and active food

ingredient for the patient. As a result, particularly in children, we tend to perform more “open challenges” with suspected foods starting with milligram amounts and doubling the test dosage every 20 minutes until a reaction occurs or the patient tolerates the suspected food.

Outside the hospital environment, more emphasis is placed on the food allergy history and the results of specific allergy testing using skin prick tests with the fresh native food allergens or specific IgE utilising the ImmuncAP RAST testing system. Atopy Patch Testing (APT) may be used to determine delayed food hypersensitivities. APT used together with SPT or RAST may give increased Positive Predictive Value (PPV) to the food allergy diagnosis.

## DOUBLE BLIND PLACEBO CONTROLLED FOOD CHALLENGE TESTING (DBPCFC):

*Dr Bindselev-Jensen*

This accurate food allergy testing process utilises the raw allergen, concealed in a “cake” of “broth” so that the actual food’s taste and texture cannot be identified by the subject being tested. The physician is also unaware whether the active ingredient or placebo is administered to the patient until the code is broken (double blinded). This is a very accurate way of determining a true food allergy without any patient or doctor bias complicating the process. The patient is given increasing amounts of the placebo and then on a separate occasion, the active ingredient until a reaction occurs or they tolerate a substantial amount of the food allergen. Obviously the patient is carefully monitored by nursing staff and doctors in hospital with full resuscitation equipment including adrenaline and oxygen available. The patient is brought into hospital for the day and monitored. Some researches suggest the patient should be monitored in hospital for 2 days and then daily thereafter to document any delayed food hypersensitivity reactions. This is a labour intensive process and only about 4 food challenges can be done per day even in the most active allergy unit. As a consequence, more “open” challenges are performed in hospital when both patient and doctor know what the active ingredient is, and the patient is monitored for objective signs of food allergy. If there

is a reaction to a challenge test food, the patient should avoid that food for a further six to twelve months before considering a re-challenge. There is some debate whether challenge testing actually “re-sensitises” the person to the test allergen. It is therefore recommended that once an individual successfully tolerates a food challenge that they should continue to include that particular food in their diet on a regular basis to maintain their tolerance.

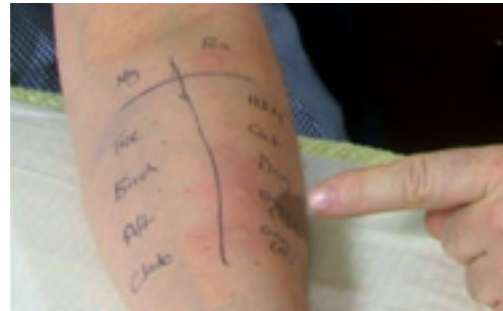
## FOOD ALLERGEN SKIN PRICK TESTING

*Dr Turjanmaa*

Skin Prick Testing (SPT) is the most common and cheapest diagnostic procedure used to confirm a food allergy. It is best to use the fresh food extract such as cow’s milk, whole hen’s egg, wheat paste, soy milk, codfish, and peanut applied to the skin using the Prick-Prick method. Any foodstuff can be tested in this manner using the native fresh food. The standardised test lancet is dipped into the test food solution and the patient’s skin is pricked with this allergen impregnated lancet. A new lancet is used for each test. The skin test site is then observed for 15 to 20 minutes and any wheal reaction measured. A positive test is any wheal 3mm or greater than the negative saline control test. In addition, we always have a positive histamine or codeine control which is used to gauge skin reactivity. This test is simple, safe, cheap and easy to perform and the results are immediately available.



Skin Prick Testing using standardised lancet



Skin Prick Testing wheal and flare reaction

Variants of skin prick testing include the Scratch Patch test (the skin is scratched and allergen then applied under an occlusive patch) and Skin Application Food test (in which food is applied to the skin without pricking but examined every 10 minutes for a reaction). Neither of these tests is commonly used as they offer no benefit over routine Skin Prick Testing (SPT) with allergen. Intradermal testing for food allergies is not used either.

## BLOOD TESTING FOR SPECIFIC IgE

*Dr Poulsen*

Tests for identifying specific IgE antibodies have been improved upon, since the original Radio-absorbent tests (RAST) were introduced. Now the ImmunoCAP specific IgE test can measure a large number of individual allergens (over 150 food tests available); also test panels for screening nut, cereal, fish and paediatric food allergens are available. This test measures serum specific IgE to recognised food allergens in a particular food (Graded 0 to 6 or measured in ku/l of specific IgE). In vitro determination of specific IgE, although convenient in food allergy testing, is slightly less accurate than prick-prick skin test using the raw food material. The ImmunoCAP

manufacturers identify relevant food allergens using the “western blotting” method and then add these purified recombinant allergens to the test “CAP” depending on the prevalence of that specific allergy in a particular population group (such as Ara h1 and Ara h2 the principal peanut allergens). False positive results of these in vitro tests for food allergy can often confuse the diagnosis. It seems that non specific anti Cross-reactive Carbohydrate Determinants (CCD) IgE or anti bromelain IgE antibodies are responsible for these false positive responses (CCD are glycan structures xylose and fucose found in foods which convey allergenicity). Newer ImmunoCAP tests can now measure CCD.

Serum specific IgG testing is a good measure of food allergen exposure and levels persist for many years but offer no allergy diagnostic value.



ImmunoCAP® multi-channel specific IgE analyser

## ATOPY PATCH TEST (APT)

*Prof Niggemann*

The Atopy Patch Test (APT) is a relatively new application of the original Allergen Patch Test, procedure previously used only to diagnose contact dermatitis due to delayed reactions to topical chemicals and contact preservatives. In this test, one drop (50µl) of each raw food is applied to the skin for 48 hours in a series of 12mm Finn chambers. The patch is then removed and the skin examined for erythema or blistering; another re-examination of the skin is performed after further 24 hours (72 hours in total). Non specific skin irritation may initially result from the contact of the skin with the patch, but only a delayed hypersensitivity reaction should be evident after a further 24 hours.

This test is useful in children with Atopic Dermatitis (AD) for determining a delayed hypersensitivity to foods such as cow's milk, hen's egg, wheat and soy which can be involved in AD.

APT is highly specific for food allergy but lacks sensitivity. Positive APT and SPT in combination, increase the likelihood of a food allergy being present in atopic dermatitis.



Atopy Patch Test applied to back

## PRACTICAL TESTING WITH CROSS-REACTING FOOD ITEMS

Allergy Center, Odense University Hospital, Denmark

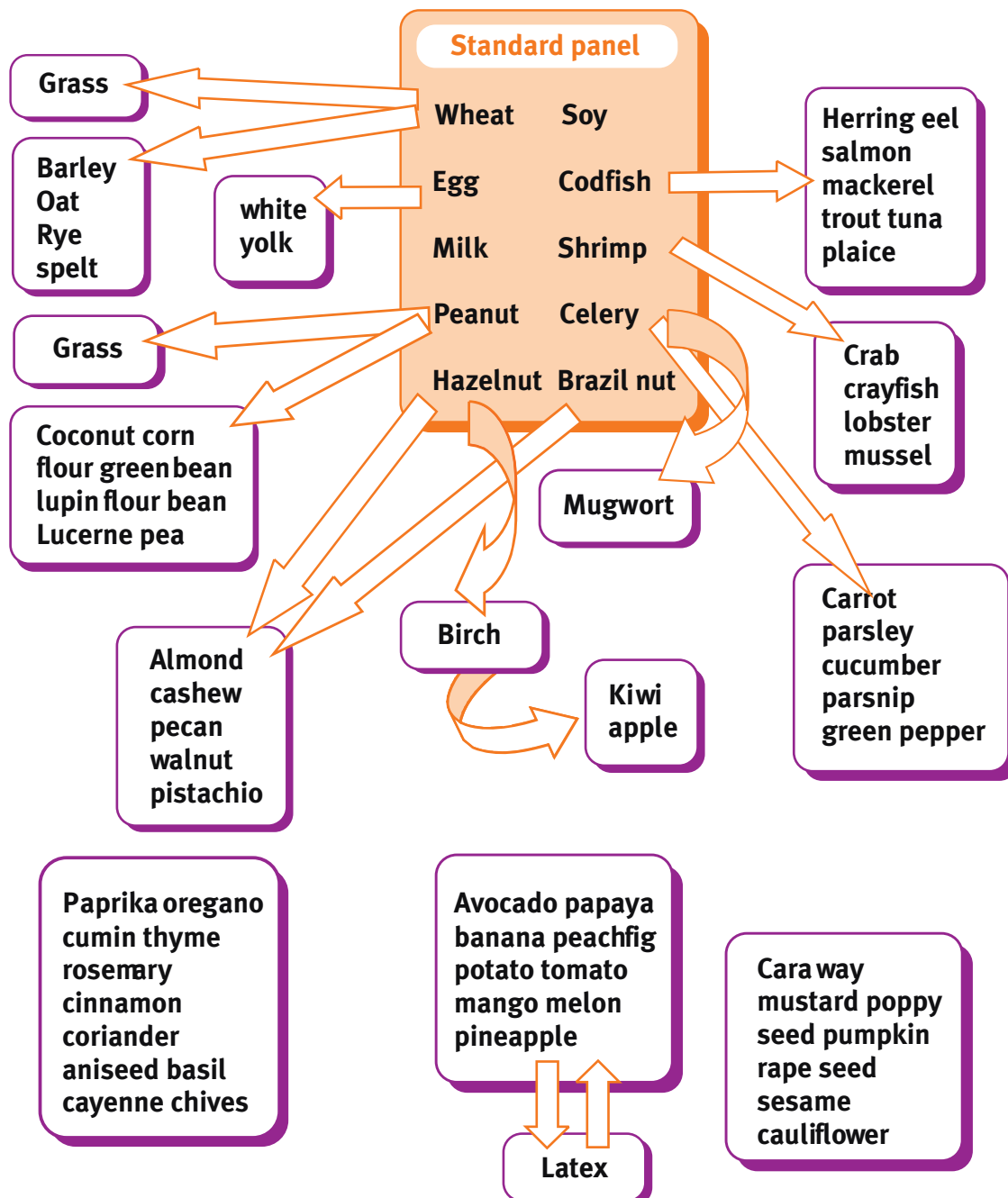


Figure 4: Practical testing with cross-reacting food items

## RELIABILITY OF PREDICTING FOOD ALLERGIES AND DIFFICULTIES ENCOUNTERED

*Prof Niggemann & The Expert Panel*

It is extremely difficult to predict with any degree of accuracy whether an allergen identified by a positive skin test or raised serum specific IgE result is responsible for a food allergic reaction. The patient's allergy history is therefore extremely important in determining the correct offending allergen that needs to be excluded from the diet.

Sporik & Sampson in the USA have produced predictive value cut-off points (up to the 90% confidence intervals) for Skin Prick Test and Specific IgE results

above which a food allergy is likely to be present. In individuals with results above these cut-off values, a specific allergy to that food is highly likely to be present and they postulate that allergen challenge testing is therefore unnecessary. However the US cut-off values determined by Sporik & Sampson were different from the European experience of Eigenmann et al. The European cut-off levels for predicting cow's milk and egg allergy were higher, with no accurate or reliable cut-off value being found for wheat and soy. They therefore suggest that these predictability cut-off levels are population specific and should be developed in each local population before using them as predictors for food allergy diagnosis.

### Predictive value of specific blood IgE levels (KU/L)

<b>EGG</b>	<b>7</b>
Infants ≤ 2yrs	2
<b>MILK</b>	<b>15</b>
Infants ≤ 2yrs	5
<b>PEANUT</b>	<b>15</b>
<b>FISH</b>	<b>20</b>
<b>TREE NUTS</b>	<b>15</b>

### Predictive value of Skin Prick Tests (Wheal Diameter)

<b>MILK</b>	<b>8mm</b>
Infants ≤ 2 yrs	6mm
<b>EGG</b>	<b>7mm</b>
Infants ≤ 2yrs	5mm
<b>PEANUT</b>	<b>8mm</b>
Infants ≤ 2yrs	4mm

Table 5: Food Allergen-Specific IgE Levels and Food Allergen Skin Prick Tests with a Positive Predictive Values ≥ 95%. (adapted from Sporik *et al.*, 2000<sup>2</sup> and Eigenman *et al.*, 1998<sup>3</sup>)

The other problem with using the predictive values is that at least one in ten patients will fall outside their ranges. For example it is possible to have a positive serum specific IgE level of over 100ku/l to the peanut allergen but to tolerate peanuts in the diet with no adverse reaction! It is equally possible to have a serum specific IgE level to cow's milk of under 0,35k/l and have anaphylaxis on milk exposure. There are therefore limitations to the predictability and accuracy of entrenched cut-off values for skin prick tests and specific IgE testing and the DBPCFC still remains the allergy diagnostic "gold standard" for the foreseeable future.

Occasionally non-IgE delayed food hypersensitivity may present as Allergic Eosinophilic Eosophagitis with problematic

gastro-esophageal reflux (GER) resistant to medication. This condition responds to a food elimination diet (milk, egg or wheat, depending on the culprit). Diagnostic tests for delayed hypersensitivity to common infant foods are not available, but typically an oesophageal biopsy will show predominantly eosinophilic inflammation. This delayed cow's milk hypersensitivity reaction occurs in early infancy and usually resolves spontaneously by the end of the second year of life. Other manifestations of delayed food hypersensitivity include allergic eosinophilic gastritis and eosinophilic enterocolitis manifesting with vomiting, colicky abdominal pain and bloody diarrhoea or constipation which may be confused with Coeliac Disease.



## TREATING FOOD ALLERGY

*Dr Knulst*

**Allergen avoidance** is the only effective treatment in food allergy and anaphylaxis. Depending on sensitivity, some people will react to microgram traces of allergens in food and even cooking vapours, while others will tolerate small amounts and only react to milligram amounts of ingested allergen. Hypoallergenic extensively hydrolysed casein and whey cow's milk formulas still contain minimal cow's milk protein, and exquisitely sensitive individuals may need to go onto amino acid formulas such as Neocate.

Cross-reaction hypersensitivities between food families (legumes and stone fruits) and unrelated foods with similar panallergens (apple and carrot) should be always considered.

### **Pharmacologic treatment**

One has to adapt a pragmatic approach and try to preserve the quality of life.

The treatment of severe food allergy manifested as anaphylaxis involves an individualised emergency action plan which should include provision of emergency epinephrine by auto-injector (Epipen®, Anapen®, Twinject®) especially in those food and nut allergic individuals who have concomitant asthma.

Milder allergic reactions involving the skin and mucosa may be adequately treated using oral antihistamines but an

individualised treatment plan should always consider epinephrine, antihistamines and include oral steroids to prevent late reactions. All allergic events should be discussed with a physician or allergist and each allergic reaction requiring treatment should be followed up with emergency room assessment by an experienced physician.

### **Prophylaxis**

Oral sodium cromoglicate prophylaxis for food allergy as used in the past seems ineffective and expensive and is no longer widely used, similarly the use of low dose oral steroids is not recommended as prophylaxis. Long-term prophylaxis with long-acting oral antihistamine medication is discouraged as these antihistamines may actually mask the early stages of an allergic reaction. This could lead the food allergy sufferer to misinterpret their usual early symptoms and not take adequate precautions to treat an allergic reaction in the proper timeframe.

In Pollen-Food Allergic (PFS) syndromes such as Birch pollen Oral Allergy Syndrome (OAS), Birch pollen desensitisation immunotherapy should be considered as it may also result in diminished reactions to the cross reacting foods including apple, cherry and hazelnut.



## FUTURE TREATMENT OPTIONS FOR FOOD ALLERGY

*Dr Host*

A review of the history of food allergy treatment options over the last 30 years was presented.

Food allergy vaccines are in the process of development, but there is concern that once desensitised to one (peanut) allergen, a period of temporary food allergen tolerance may ensue; but the individual may then subsequently develop allergies to other (peanut) allergens and the treatment will then ultimately be ineffective. Genetically Modified (GM) foods devoided of the offending allergen have been explored, and again the fear is that individuals will then go on to develop sensitisation to other candidate allergens.

Heat-killed *Listeria* bacteria used in early animal trials seem to increase the dose of peanut allergen tolerated before a reaction occurs, and this may represent an effective future therapeutic option.



Anti-IgE monoclonal antibodies seem to protect peanut allergic individuals by binding peanut specific IgE, but this treatment is expensive, has to be given by fortnightly injection and the treatment has to be continued indefinitely. Specific vaccines and mutant vaccines are also being explored, but at this point in time the only effective treatment is specific food allergen avoidance.

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## THE UCB INSTITUTE OF ALLERGY

Division of UCB Pharma S.A., The UCB Institute of Allergy (IOA) is an independent, European and not-for-profit organisation, created in 1987 to combat allergy. In response to the international epidemic of this disease, the Institute's objective is to implement all the resources necessary to raise awareness of allergy as a major health issue amongst the general public, patients, Healthcare professionals and public authorities.

Under the supervision of a Scientific Advisory Board made up of eminent European specialists in the field of allergy, IOA has initiated many actions. These aim to inform and educate about allergy, to improve prevention, to promote research, to analyse the current situation and to define key actions to be taken over the coming years. Moreover IOA favours cooperation between various allergy related organisations. The Institute is present all around Europe with 20 national sections and in South Africa.

The Institute's web site (<http://www.theucbinstituteofallergy.com>) and central membership library provide members with current relevant information and publications about allergy. For the general public, schools and children, IOA has produced videos (e.g. "Who's sleeping in your pillow?", "Allergic: to be or not to be?...Rhinitis"), educational games and other information material. IOA also organises and holds meetings, symposia, conferences, panel discussions.

As a result of these activities, The UCB Institute of Allergy hopes to forestall the sobering prediction of certain epidemiologists: In 30 years' time, everyone may be allergic... Unless we act now!

# FOOD

Expert panel of food allergists included:

Carsten Bindslev-Jensen, Philippe Eigenmann, Bodo Niggemann, Arne Host, Fabienne Rance, Jonathan Hourihane, Ronald van Ree, Antonella Muraro, Andre Knulst, Barbara Ballmer-Weber, Lars Poulsen, Susanne Halcken, Torsten Zuberbier, Kristina Turjanmaa and staff of Allergy Center, Odense University Hospital.

**REPORT BY DR A J MORRIS**

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