



Food allergy management in adults

Key points

- Food allergy affects an estimated 1 to 3% of adults, and there is evidence that this percentage is increasing.
- Crustaceans, tree nuts, peanuts and fish are the most common food allergens in adults; along with wheat, soy, fruits, vegetables and seeds, they account for more than 90% of food allergy cases in adults.
- Diagnosis of food allergy requires a careful history and judicious selection of investigations, with careful consideration of differential diagnoses including food intolerances.
- The mainstay of therapy is education about allergen avoidance and risk minimisation, including optimised asthma management.
- The key treatment for food-induced anaphylaxis is prompt administration of adrenaline, usually intramuscularly; patients at risk should carry an anaphylaxis action plan and kit, including an adrenaline autoinjector, at all times.

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Food allergies cause a significant public and personal health burden. Accurate diagnosis and patient education on avoidance strategies and anaphylaxis treatment are the mainstays of management.

The prevalence of allergic disorders in Australia and New Zealand is among the highest in the developed world. An Access Economics report estimated that in 2007, 4.1 million Australians (almost 20% of the population) had at least one allergic disease, with the working age population having the highest prevalence (78% among those aged between 15 and 64 years).¹ Food allergy affects approximately 1 to 3% of adults, with growing evidence that its prevalence is increasing.²

The most common food allergens in adults are, in descending order of prevalence, crustaceans (e.g. shrimp, crab, lobster), tree nuts (e.g. walnut, brazil nut, cashew, hazelnut), peanuts and fish. Other common food allergens include wheat, soy, fruits (e.g. apple, pear, cherry, peach, plum, kiwifruit), vegetables (e.g. cucumber, melon, watermelon, zucchini, pumpkin) and

seeds (e.g. sesame). Although any food can potentially be an allergen, the aforementioned foods account for more than 90% of food allergies in adults.²⁻⁴

Food allergy causes a significant and increasing public and personal health burden. Although the costs of dealing with food allergy and anaphylaxis in Australia are currently not known, an American study in 2007 estimated that the direct medical costs of food allergy were \$225 million per year, with an additional \$115 million per year in indirect costs from medical consultations, hospital treatment and medication use.⁵ With regards to personal health burden, food allergy has a significant impact on health-related quality of life, associated with the need for dietary restriction, adrenaline self-administration and the psychological impacts related to the unpredictability of accidental exposure.^{6,7}

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1. FEATURES OF COMMON FOOD ALLERGIES⁹

Egg, milk

- Common in childhood
- Mostly outgrown during late childhood/teenage years
- Very uncommonly persists into adulthood
- Some patients can tolerate egg or milk in baked form (high-temperature cooking denatures the allergenic proteins)

Nuts (peanuts, tree nuts)

- Usually begins early in life
- Commonly persists into adulthood (approximately 80% of cases)
- Tree nut allergy is frequently associated with sensitisation to more than one variety
- Most common cause of life-threatening and fatal food-induced anaphylaxis

Seafood (crustaceans, molluscs, fish)

- Fish and crustacea allergies generally begin later in childhood or even in teen or adult years
- Clinical cross-reactivity is common within seafood species (fish, crustacea and molluscs) but uncommon between species
- Most common cause of food-induced anaphylaxis requiring emergency department attendance
- Most cases persist indefinitely

Wheat

- In adults, wheat allergy can present as wheat-dependent exercise-induced anaphylaxis (typically associated with IgE antibodies to omega-5-gliadin)
- Wheat allergy is distinct from coeliac disease, which is an autoimmune disease triggered by exposure to gliadin

RISK FACTORS AND CHARACTERISTICS

Multiple risk factors have been identified as influences on food allergy or sensitisation. Patients who are atopic or have a family history of food allergy or atopy are at an increased risk of developing food allergies.⁸ Other proposed risk factors for food allergy include vitamin D insufficiency, dietary fat (reduced consumption of omega-3 polyunsaturated fatty acids), reduced consumption of antioxidants, obesity, increased hygiene,

2. CLASSIFICATION OF ADVERSE REACTIONS TO FOODS*

Immune mediated (primarily food allergy)

- IgE mediated (e.g. food-induced anaphylaxis, food-dependent exercise-induced anaphylaxis, acute urticaria, oral allergy syndrome)
- Non-IgE mediated (e.g. food protein-induced enteropathy syndrome, coeliac disease)
- Mixed IgE and non-IgE mediated (e.g. eosinophilic oesophagitis, atopic dermatitis)
- Cell mediated (e.g. allergic contact dermatitis)

Non-immune mediated (primarily food intolerance)

- Metabolic (e.g. lactose intolerance)
- Pharmacological (e.g. caffeine)
- Toxic (e.g. scombroid poisoning)
- Idiopathic, undefined, other (e.g. sulfites)

* Adapted from: NIAID-Sponsored Expert Panel, et al. *J Allergy Clin Immunol* 2010.¹¹

and the timing and route of exposure to certain foods.²

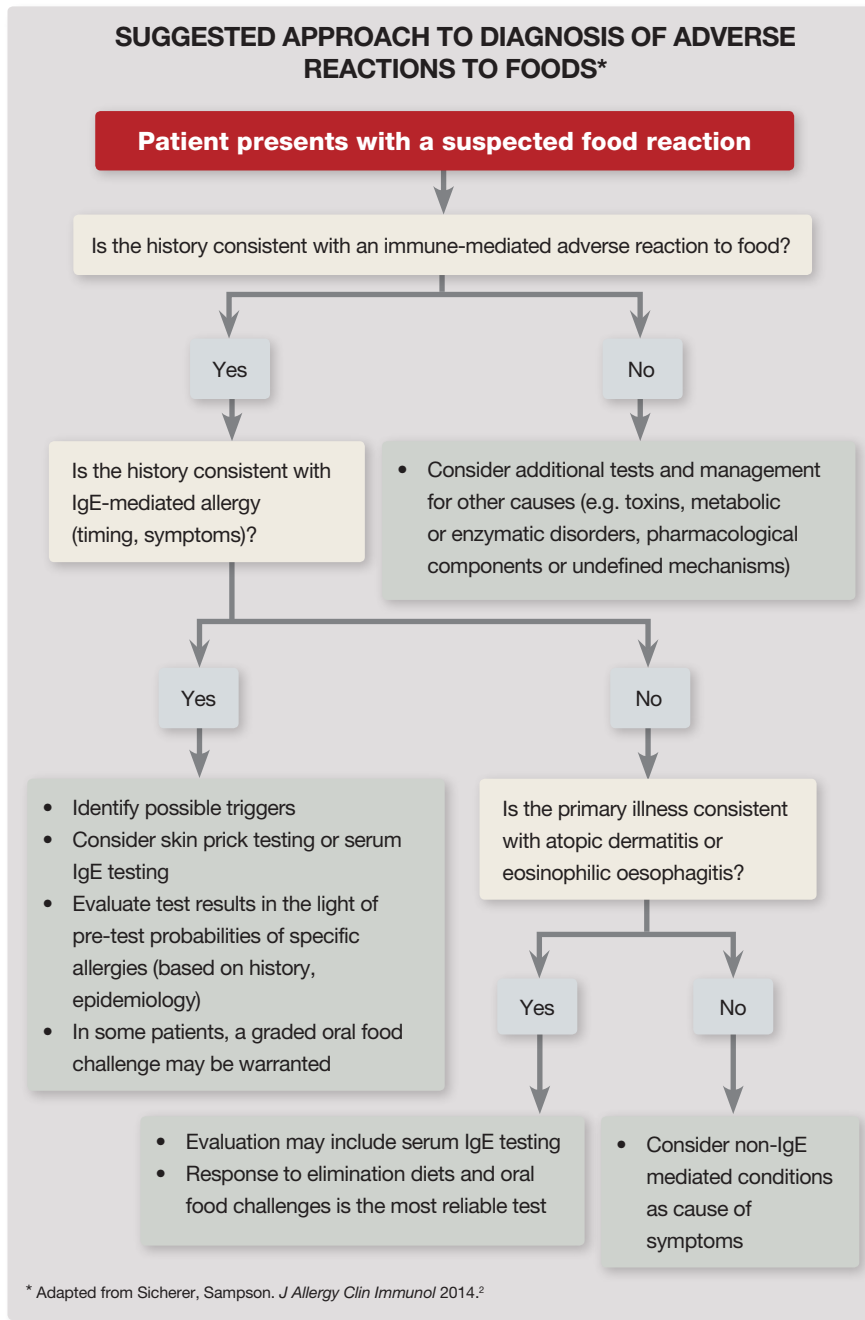
Box 1 describes the features of clinical allergy to different foods.⁹ Of the common food allergies, nut allergies are the most dangerous. More than 80% of deaths from food anaphylaxis occur in teenagers and young adults, particularly males, and most are due to peanut or tree nut reactions. Almost all fatalities are associated with poorly controlled asthma.

DIFFERENTIAL DIAGNOSES

Most patients who report an adverse reaction to food do not have true, or IgE-mediated, food allergy.¹⁰ Given these patients are often on self-imposed highly restrictive diets, it is important to carefully consider the differential diagnoses for their food-related symptoms, and provide reassurance and education about the diagnosis. Box 2 outlines the various types of adverse reactions to foods classified by immune and non-immune mediated aetiology.¹¹

Adverse reactions that are not true IgE-mediated food allergies include:

- host-specific metabolic disorders (e.g. lactose intolerance, galactosaemia, and alcohol intolerance)
- pharmacological responses (e.g. to caffeine, tyramine in aged cheeses which can trigger migraine or chemicals in spoiled dark-meat fish that can cause scombroid poisoning)
- toxin-related (e.g. food poisoning).¹²



Other food intolerances are to food chemicals, with sulfites (sulfur dioxide and sodium metabisulfite) and food colours (tartrazine) the most commonly implicated. The exact mechanism of these food intolerances is generally not clear, but most have no defined immunological mechanism.¹¹

There is no reliable skin or blood test

to diagnose food intolerance, but in some patients dietary elimination and challenge may assist diagnosis.¹³ Some patients with a diagnosis of irritable bowel syndrome may benefit from a diet focusing on low levels of fermentable oligosaccharides, disaccharides, monosaccharides and polyols (the low FODMAP diet).¹⁴

DIAGNOSIS OF FOOD ALLERGY

The diagnosis of food allergy requires a thorough history and physical examination to consider a broad range of aetiologies, as outlined in Box 2. The history may help to suggest the possible culprit foods and identify a likely pathophysiological mechanism, and specifically whether the food-related adverse reaction is likely to be IgE-mediated, which will guide further investigations.

A comprehensive food allergy history should identify:

- the possible food or foods implicated in the event
- the quantity and form ingested (e.g. baked, cooked or raw)
- the time course of the reaction
- any possible cofactors, such as exercise, medications or alcohol.

The effect of cooking varies according to the allergen. For fruits associated with the oral allergy syndrome, cooking generally destroys allergenicity; for nuts, cooking or roasting may enhance their allergenic potential.

A number of features in the history of the adverse reaction suggest a higher likelihood of an IgE-mediated food allergy. IgE-mediated reactions:

- generally occur within seconds to minutes of ingestion
- cause similar reactions on each exposure to the particular food or known cross-reacting foods
- are typically to commonly recognised food allergens
- occur in conjunction with a personal or family history of atopic disease.¹⁵

A general approach to the diagnosis of adverse reactions to foods is outlined in the flowchart and described in more detail below.

CLINICAL SPECTRUM OF IGE-MEDIATED FOOD ALLERGIES
IgE antibody-dependent food allergies

Food-induced anaphylaxis

Food-induced anaphylaxis is a serious allergic reaction that is rapid in onset and

may cause death. It is a systemic reaction mediated by sensitised mast cells and basophils. It is defined as including any of the following signs:

- shortness of breath or stridor
- tongue and/or laryngeal angioedema
- throat tightness or swelling
- a hoarse voice
- wheeze or persistent cough
- persistent dizziness or collapse.¹³

It can also manifest as gastrointestinal symptoms such as nausea, vomiting and diarrhoea.

Food-dependent exercise-induced anaphylaxis

This is a variant of food-induced anaphylaxis where the reaction depends on the temporal association between the consumption of the trigger food and exercise (usually within two hours). Wheat allergy is the most common cause of food-dependent exercise-induced anaphylaxis but any food allergen has this potential.

Acute urticaria and angioedema

Cutaneous reactions to food are some of the most common presentations of food allergy. IgE-mediated cutaneous reactions include acute urticaria, angioedema, flushing and pruritus. Skin lesions often develop rapidly after ingesting the trigger food.

Oral allergy syndrome

Oral allergy syndrome is also known as pollen-associated food allergy syndrome. It is a form of localised IgE-mediated allergy, usually to raw fruits or vegetables, with symptoms confined to the lips, mouth, throat and gastrointestinal tract. Box 3 shows some of the recognised potential cross-reactions between airborne allergens and foods.¹⁵

IgE antibody-associated and cell-mediated food allergies

IgE antibody-associated, cell-mediated food allergies generally have a delayed onset and a longer-term effect.

Atopic dermatitis

Atopic dermatitis is associated with complex interactions between skin barrier dysfunction and environmental factors such as irritants, microbes and allergens. Although a link between atopic dermatitis and food allergy is well established in children, the role of food allergy in the pathogenesis and severity of this condition in adults is controversial.

Eosinophilic oesophagitis

Although the pathogenesis of eosinophilic oesophagitis is not yet understood, both IgE and non-IgE mediated mechanisms are thought to be involved, leading to localised eosinophilic inflammation of the oesophagus. In adults and adolescents, eosinophilic oesophagitis most often presents with dysphagia and oesophageal food impactions. Symptoms may include episodic 'sticking' of food; patients may need to drink water to help swallowing.

Eosinophilic oesophagitis is diagnosed based on the finding of more than 15 to 20 eosinophils per high power field in an oesophageal biopsy specimen. A minority of patients have a coexisting sensitisation to food allergens. Evidence that eosinophilic oesophagitis is food induced includes resolution of both symptoms and oesophageal eosinophilia with dietary elimination and recurrence with reintroduction of the suspected food culprit.¹⁶ Eosinophilic infiltration of other areas of the gastrointestinal tract can cause a range of symptoms, depending on the area involved.

Although some patients who opt to use an elimination or elemental diet show normalisation of histopathology, these diets are typically difficult to maintain. Combination therapy with topical corticosteroids such as swallowed fluticasone and acid suppression provides effective therapy in most patients and is more accessible. Chewable proton pump inhibitors seem particularly effective and are worth trying.

3. POTENTIAL CROSS-REACTIONS BETWEEN AIRBORNE ALLERGENS AND FOODS*

Birch pollen: carrots, celery, fresh fruit, hazelnuts, parsnips, potatoes

Grass pollen: kiwifruit, tomatoes

Ragweed pollen: bananas, melons

* Adapted from Kurowski K, Boxer R. *Am Fam Physician* 2008;¹⁵

INVESTIGATIONS

After a comprehensive medical history has been taken, if an allergic reaction (IgE or non-IgE mediated) is suspected then specific tests can be selected to determine the culprit food.

Skin prick testing

Epicutaneous skin prick testing (SPT) can help identify foods that may be responsible for IgE-mediated food-induced allergic reactions. However, SPT alone is not considered diagnostic of food allergy, which is a clinical diagnosis.¹² SPT can only indicate sensitisation, and because many patients are sensitised to certain foods these tests have low positive predictive value in unselected populations. The negative predictive value of SPT to nuts or seafood is higher than the positive predictive value. The benefits of SPT are the immediate results and relatively low cost. In SPT, use of fresh food in a 'prick to prick' test (where the food is pricked and then the skin) increases the diagnostic yield.¹⁷

Allergen-specific serum IgE testing

Allergen-specific serum IgE (sIgE) tests, also known as RAST (radioallergosorbent) tests, are useful for identifying foods that provoke IgE-mediated food-induced allergic reactions, based on specified parameters. A number of similar methodologies are used, each using antibody specific for human IgE to detect the presence of serum IgE bound to a fixed allergen substrate. They are useful when SPTs cannot be done

TABLE 1. CROSS-REACTIVITY BETWEEN RELATED FOODS AND SUBSTANCES*

Allergen	Related food or substance	Approximate clinical reaction rate
Peanut	Other legumes (e.g. peas, lentils, beans)	5%
One type of tree nut	Other tree nuts (e.g. walnut, brazil nut, cashew, hazelnut)	35% (higher for walnut/pecan, almond/hazelnut, cashew/pistachio)
One type of fish	Other fish	50%
One type of shellfish	Other shellfish	75%
One type of grain	Other grains	20%
Cow's milk	Goat and sheep's milk	>90%
	Mare's milk	5%
	Beef	10%
Melon	Other fruits	92%
Latex	Fruits (kiwifruit, banana, avocado)	35%
Fruits (kiwifruit, banana, avocado)	Latex	11%

* Adapted from Sicherer SH, Sampson HA. *J Allergy Clin Immunol* 2014.²

(e.g. because of dermatitis or dermatographism) or are unavailable. Infrequently, SPTs and sIgE assays have discordant results, because of differences in available conformational epitopes in liquid versus solid states. If patients have negative results on initial testing but clinical suspicion for allergy remains high then the alternative test should be performed. It is also important to select the specific allergens to be tested on the basis of the clinical history, rather than using standard mixes of allergens.

Graded oral food challenges

A lack of response to SPT and sIgE tests does not exclude IgE-mediated food allergy. In intermediate-probability cases, further evaluation may be necessary before food allergy can be confirmed or excluded. The double-blind placebo-controlled food challenge is the gold standard for diagnosing food allergy.¹² However, single-blind

or open-food challenge may be considered diagnostic under certain circumstances. Given that food challenges are time consuming, expensive, and subject the sensitised patient to a risk of anaphylaxis, patients should be reviewed by a specialist beforehand to ensure appropriate patient selection. Oral food challenges are less commonly performed in adults because comorbidities increase the risk of anaphylaxis. A strong clinical history with confirmatory allergen-specific IgE detection is sufficient for diagnosis.

Unproven methods of allergy testing

Between 50 and 70% of adults and children with allergic disease consult alternative practitioners for diagnosis and treatment. Hence, some patients will present with previous diagnoses of food allergies from other practitioners. Some will have undergone unproven diagnostic 'allergy tests' or

treatments. Examples of unproven diagnostic methods include cytotoxic food testing, kinesiology, Vega testing, electrodermal testing, pulse testing, reflexology, IgG food antibody testing and hair analysis. These tests have not been scientifically validated and may lead to dangerous major dietary restrictions. Furthermore, access to more effective diagnostic techniques and treatments may be delayed, with lost productivity from inadequately controlled disease.¹⁸ Further information about these tests is available at the website of the Australasian Society of Clinical Immunology and Allergy (ASCI; www.allergy.org.au/health-professionals/hp-information/asthma-and-allergy/unorthodox-testing-and-treatment).

MANAGEMENT

Education: dietary avoidance and risk minimisation

Dietary avoidance is the mainstay of the management of IgE-mediated food allergy. Education about avoidance should encourage careful reading of food labels and care in obtaining foods from restaurants. Furthermore, patients should be counselled about being cautious with foods that may be cross-reactive with known allergens, taking into account skin prick or serum testing of related foods and cross contact among foods in preparation (see Table 1).^{2,11,12} For patients who require particularly restrictive diets because of multiple food allergies, it is important to involve a dietitian to ensure the diet is nutritionally adequate.

Patients should be encouraged to obtain medical alert bracelets identifying their allergy and be taught to recognise symptoms that herald an allergic episode (Box 4).¹⁹ They should be given a written anaphylaxis plan and receive training in use of an autoinjector to self-administer intramuscular adrenaline. It is also important to discuss strategies to ensure that patients carry the autoinjector at all times when away from home. Ambulance insurance should be recommended. Further resources and downloadable action and

4. COMMON FEATURES OF ANAPHYLAXIS¹⁹

Respiratory effects

- Stridor/wheeze
- Difficulty swallowing
- Persistent cough
- Dyspnoea
- Hoarse voice
- Throat/chest tightness

Cardiovascular effects

- Tachycardia/bradycardia
- Hypotension
- Collapse/loss of consciousness

Skin effects

- Urticaria (hives, wheals)
- Angioedema
- Flushing
- Generalised itch

Gastrointestinal effects

- Nausea, vomiting
- Abdominal pain
- Diarrhoea

travel plans are available at the ASCIA website (www.allergy.org.au).

It is also essential for patients to be educated about other risk-minimisation strategies, especially when eating away from home. These strategies include:

- optimising their asthma management (as asthma is the main risk factor for death due to anaphylaxis)
- informing friends or alerting restaurants in advance about their particular allergies (to reduce risks from inadvertent exposure)
- carrying a written anaphylaxis emergency plan and kit, including an adrenaline autoinjector, a non-sedating antihistamine (for cutaneous symptoms) and oral corticosteroids (to minimise rebound or biphasic reactions while seeking appropriate emergency-department follow up). It is important to note that antihistamines and corticosteroids are considered second-line treatment

and should not be used as an alternative or before adrenaline to treat anaphylaxis¹⁹

- in circumstances where food has been prepared by other people but is believed to be safe, pausing before eating and cautiously touch-testing a trace of food on the outside of the lip as an extra precaution. Warning signs of a chilli-like reaction, or tingling, burning or swelling, will alert them not to proceed²⁰
- checking what partners have eaten during the past few hours before engaging in intimate kissing (as patients can react to foods eaten by a partner with whom they have intimate contact).

Role of the GP

GPs have a key role to play in the identification and management of food allergies and food-induced anaphylaxis. Besides providing emergency treatment in the event of an acute reaction or anaphylaxis and referring patients for specialist allergy assessment, GPs can contribute to the management of patients with food allergies by:

- ensuring that patients have two current adrenaline autoinjectors and renewing the prescriptions for these when they are used or approximately every two years when they expire
- ensuring that patients have a written anaphylaxis action plan
- ensuring that asthma (if present) is well controlled
- reinforcing the dietary avoidance and risk minimisation strategies outlined above at each visit.

Emergency treatment

The key treatment for food-induced anaphylaxis is prompt administration of adrenaline, usually intramuscularly. Details of the acute management of anaphylaxis, adrenaline dosage and ancillary treatment are available from the ASCIA website (www.allergy.org.au)²¹ and in a recent review.¹⁹

When to refer

All patients with a life-threatening allergy attributed to food exposure should be referred for specialist allergy assessment. Referral allows the careful evaluation of possible triggers and the appropriate provision of a management plan, including a written anaphylaxis plan, and initial education, including recommended dietary avoidance. In particular, patients with concurrent asthma, patients with nut allergy and adolescent patients should be referred. Many specialist allergy clinics have access to dietitians skilled in food allergy who can be integral in helping patients to avoid their food triggers, especially those that are often hidden, such as peanuts, eggs, dairy, soy or wheat. Specialist allergy nurses can also play a supportive and educational role for patients.

FUTURE THERAPIES

Immunotherapy for food allergy remains an active frontier for research in allergic disease. Past clinical trials have proven disappointing, with a high rate of adverse reactions, particularly with extracts from intact food preparations that retain IgE-binding.²² Long-term effective tolerance has not been achieved in these studies and there have been frequent side effects. Anaphylaxis has been observed even in patients who had previously mild symptoms or had already tolerated prior doses.

Oral immunotherapy has been the most extensively trialled with some evidence of desensitisation (clinical tolerance), but no reliable evidence of long-term tolerance or memory. However, side effects are numerous and ongoing daily ingestion of the particular food is required to maintain benefit, an almost impossible requirement. A recent single-centre phase 2 study demonstrated successful desensitisation in approximately half of a group of children undergoing oral peanut immunotherapy; these findings need to be replicated in a larger study before the approach can be widely advocated.²³

Combination approaches with allergen oral immunotherapy and anti-IgE therapy have also shown some promise of increased

safety.^{2,24,25} Recently, encouraging phase IIb results have been obtained in children receiving peanut patch desensitisation in a multicentre trial (VIPES trial). Current research by one of the authors sets the scene for the trial of non-IgE-binding T cell epitope-based peptides of peanut allergens to induce long-term immunological tolerance.²⁶ A similar approach for cat, grass and house dust mite allergy has been very successful in early-phase clinical trials.²⁷

CONCLUSION

Food allergies are common, and their prevalence is likely to continue to increase. Food allergy significantly impacts on quality of life, with dietary restriction and frequent hospitalisations after reactions, and can also cause fatal anaphylaxis. Numerous genetic and environmental risk factors have been identified as possibly contributory to the development of food allergy; however, studies of prevention have been inconclusive. Although recent studies have offered some encouragement, a widely available cure is still to be developed. Patient education in food avoidance strategies and the management of anaphylaxis remain the mainstays of therapy for food allergy. **MT**

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