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Saffron and Other Spices as Potential Allergenic Sources

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ABSTRACT

Spices are used as food additives to confer flavour, odour and colour or as preservatives that kill microorganisms or prevent their growth. Many of these substances can also produce allergic reactions with symptoms that range from mild and local to severe systemic disorders. Spice allergies are a growing problem, with strict avoidance of allergens as the only effective treatment. Since spices are extensively consumed in homemade meals and also contained as hidden condiments in many pre-packaged foods, adverse reactions are often difficult to prevent. This article is an overview of the most important spices responsible for immediate hypersensitivity reactions mediated by IgE, with special attention given to saffron, the world's most expensive spice.

Keywords: advisory labelling, allergen, cross-reactivity, saffron, spice allergy
Abbreviations: Art v, *Artemisia vulgaris*; **Bet v 1**, PR10 birch pollen allergen; **Bet v 2**, birch pollen profilin; **Bra j 1**, brown mustard 2S albumin; **Bra o 3**, cabbage LTP; **Cro s 2**, saffron profilin; **Cuc m 4**, melon profilin; **rCro s 3.01**, recombinant saffron lipid transfer protein 1; **rCro s 3.02**, recombinant saffron lipid transfer protein 2; **LTP**, lipid transfer protein; **PRs**, pathogenesis-related proteins; **Pru p 3**, peach lipid transfer protein; **Sal k 1**, major *Salsola kali* pollen allergen; **Sin a 1**, yellow mustard 2S albumin; **Sin a 3**, *Sinapsis alba* lipid transfer protein; **Sin a 4**, *Sinapsis alba* profilin; **SLIT**, sublingual immunotherapy

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INTRODUCTION

Allergies are one of the illnesses that have increased on a worldwide scale over the past ten years, particularly in developed countries (Sampson 2005; Takeda and Gelfant 2009). It is expected that by 2020 over 50% of the population in westernized countries will suffer from allergies. An allergic reaction can be caused by any form of contact with allergens, i.e. by ingestion (eating or drinking), inhalation (pollen, house mites, etc.) or direct contact. In most cases, the agent responsible for this reaction is a protein that activates mast cells and basophils by means of an immunoglobulin known as IgE, resulting in an extreme inflammatory response. Hypersensitivity mediated by IgE is the result of mast cells and basophil mediators. Clinical symptoms are a result of cross-linking of IgE and aggregation of high-affinity receptors for IgE on mast cells and basophils. On activation, mast cells and basophils release both preformed mediators such as histamine and tryptase and newly formed ones such as leukotrienes and prostaglandins. These mechanisms allow recruitment of eosinophils, monocytes and lymphocytes in the area affected in the late phase response and release a variety of cytokines and inflammatory responses (Metcalfe and Peavy 2009; Simons 2009) (**Fig. 1**). This reaction is only produced in individuals with a predisposition for developing allergies. The typical symptoms of allergic reactions can occur on the skin in the form of hives, eczema, itching or swelling; in the gastrointestinal tract,

Received: 18 February, 2010. Accepted: 8 May, 2010.

Invited Mini-Review

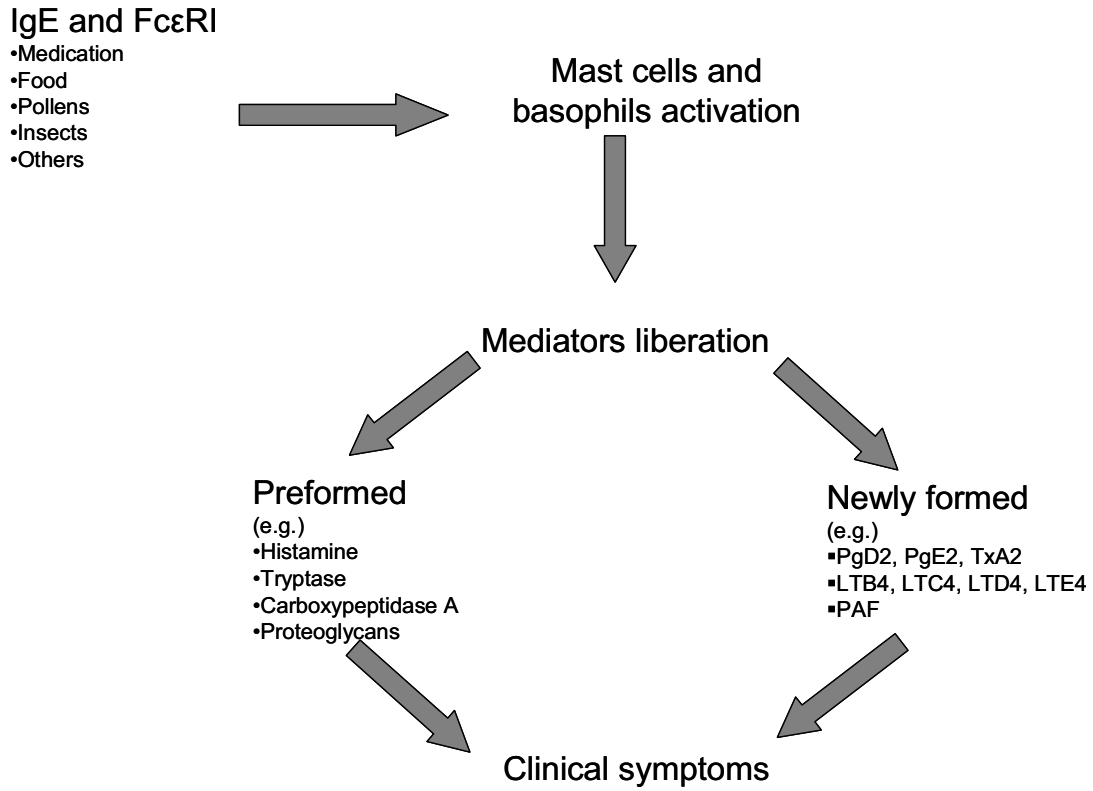

Fig. 1 Mechanisms of IgE-mediated allergenic reaction. Clinical symptoms are a result of cross-linking of IgE and aggregation of high-affinity receptors for IgE on mast cells and basophils. Common triggers include foods, medications, insect stings, and pollens. On activation, mast cells and basophils release both preformed and newly formed chemical mediators, only a few of which are listed.

producing nausea, vomiting or diarrhea; and in the respiratory tract as asthma, rhinitis, laryngeal edema or throat swelling. In highly sensitive individuals, ingestion of specific foods can produce anaphylactic shock which may even lead to death if appropriate treatment is not given (Tang *et al.* 2009).

FOOD ALLERGY

Food allergies affect 6% of young children and 3-4% of adults in westernized countries (Pieretti *et al.* 2009; Sicherer and Sampson 2009). There is no treatment for food allergies so specialists recommend strict avoidance of offending food allergens. However, numerous strategies for definitive treatment are being investigated, including sublingual/oral immunotherapy, injection of anti-IgE antibodies, etc. (Sicherer and Sampson 2009). For instance, Fernandez-Rivas *et al.* (2009) have recently shown the efficacy and safety of sublingual immunotherapy (SLIT) with a peach extract quantified in mass units for Pru p 3, the peach lipid transfer protein. They point out that the SLIT for peach allergy could be a promising therapeutic option that may modify patients' clinical reactivity to peach intake.

Food allergens are divided into two classes. Class 1 food allergens are also called true food allergens. This kind of allergen is almost always a protein which elicits both the sensitization and the effector phases of the IgE-mediated food allergy. Class 2 food allergens provoke allergic reactions in patients previously sensitized by inhalation or contact with homologous allergens from other allergenic sources such as pollen (Egger *et al.* 2006). Allergens belonging to Class 1 seem to share common characteristics, e.g. resistance to digestive enzymes and heat, thus indicating that sensitization occurs in the gastrointestinal tract. Class 2 food allergens are in general more sensitive to gastric digestion and heat and appear to cause a mild oral reaction.

A common feature in patients allergic to a particular agent is their awareness of other allergenic sources. Cross reactions have been reported among similar agents in closely related species, such as birch and hazel pollen (Valenta *et al.* 1991) or fruits of the same botanical family (Pastorello *et al.* 1994; Ahrazem *et al.* 2005), and also between phylogenetically distant species, such as birch pollen and apple fruit or latex and some fruits (Salcedo *et al.* 2001; Hofmann and Burks 2008; Bartra *et al.* 2009). Cross-reactivity is caused by the presence of Ig epitopes within the proteins involved in allergies. For example, the distribution of members of the Bet v 1 family (the main allergen of birch pollen) in different pollens, fruits and vegetables is well documented (Vieths 1997; Breiteneder and Ebner 2000; Breiteneder and Radauer 2004); also profilins are cross-reactive plant allergens responsible for multiple pollen sensitization and pollen-associated food allergy (Rauder *et al.* 2006; Bonds *et al.* 2008). Many identified food allergens are also widely distributed throughout the plant kingdom and are involved in the cross-reactions between antigens from unrelated plant species, therefore being recognized as panallergens.

The majority of plant food allergens can be grouped into just 4 protein families: 1) the prolamin superfamily containing several allergenic (2S albumins, nonspecific lipid transfer proteins, cereal amylase and protease inhibitors) and prolamins member families; 2) the cupin superfamily comprising numerous functionally highly diverse protein families, although allergenicity within the cupins is observed mainly in the vicilin and legumin seed storage proteins; 3) the profilin family which contains ubiquitous eukaryotic proteins that are nonallergenic, with the exception of profilins from flowering plants; and 4) the Bet v 1 superfamily containing various families, with the pathogenesis-related protein 10 family as the only allergenic members (Radauer and Breiteneder 2007). When the amino acid sequences of all known food allergens are analyzed and compared, the majority of them can be included in some of the 17 described pathogenesis-related protein (PR) families (Breiteneder and Ebner 2000). Most PRs and related proteins are induced through the action of the signalling compounds such as salicylic acid, jasmonic acid, or ethylene, and possess antimicrobial or insecticidal activities (van Loon *et al.* 2006). Examples of allergens homologous to PRs are summarized in **Table 1**.

Table 1 Major pathogenesis-related proteins (PR) families involved in plant food allergy.

PR family/species/common name	Representative allergens	Protein classification
PR10		Bet v 1 homologous proteins
Malus domestica/Apple	Mal d 1	
Pyrus communis/Pear	Pyr c 1	
Prunus avium/Cherry	Pru av 1	
PR14		Lipid transfer protein
Prunus persica/Peach	Pru p 3	
Malus domestica/Apple	Mal d 3	
Coryllus aveana/Hazlnut	Cor a 8	
PR5		Thaumatin-like protein
Prunus avium/Cherry	Pru av 2	
Actinidia deliciosa/Kiwi	Act d 2	
Malus domestica/Apple	Mal d 2	
PR3		Class I chitinases
Persea americana/Avocado	Pers a 1	
Musa acuminata/Banana	Mus a 2	
Castanea sativa/Chesnut		
PR2		β-1,3-Glucanases
Musa acuminata/Banana	Mus a 5	

Spices are commonly used in cooking in order to add flavour, odour and visual appeal to food. They are usually made from seeds, bark, roots, fruits, or flowers of plants, and they are generally used as dried products with a brown, black or red colour and a pungent smell (Lai and Roy 2004). According to the United States Code of Federal Regulations, most spices are recognized as safe for human consumption, although spices considered toxic may provoke allergic reactions, ranging from mild and local to severe systemic. They can enter the body through inhalation or by ingestion even after prolonged cooking, thus suggesting the existence of resistant allergens. The following sections present data on allergies to the most important spices with specific reference to saffron.

ALLERGY TO SAFFRON

Saffron is a spice derived from the dry stigmas of the *Crocus sativus* flower. It is used in cooking as a seasoning and colouring agent and considered to be the most expensive spice in the world.

During the last ten years, numerous studies focusing on the biological and pharmaceutical properties of saffron have shown its implication in the reduction of cholesterol and triglyceride levels in the blood (Abdullaev *et al.* 1999), its capacity to combat neural disorders (Abe *et al.* 2000), as well as its role as an attenuator of the adverse effects of cisplatin used in chemotherapy (Premkumar *et al.* 2003). Saffron is traditionally used for medicinal purposes as a stimulant; aphrodisiac and antidepressant, while evidence of antitumoural effects in various cellular models is also present (Schmidt *et al.* 2007). Saffron has three main pharmacologically active metabolites: crocins, the water-soluble carotenoids that give saffron its colour; pirocrocin, responsible for the bitter taste; and safranal, the volatile compound that provides odour and aroma (Lai and Roy 2004; Moraga *et al.* 2009).

The planting of saffron corms is a difficult task with bulbs being planted one by one and by hand. Harvesting and removal of saffron stigmas is also done manually. The conditions of cultivation and handling of saffron facilitate the development of allergies by inhalation (pollinosis, asthma) and by contact.

Research on saffron allergies is scarce. One relevant article (Feo *et al.* 1997) showed the implication of saffron flowers in allergy development and its clinical significance as an occupational allergy. These authors analysed the IgE-binding fractions of saffron pollen and saffron stamens in 13 sera from sensitized patients. Their studies revealed the presence of a profilin with a molecular weight of 15.5 kDa showing a high IgE reactivity in all the sera.

Saffron allergy can even produce anaphylaxis. Wuthrich *et al.* (1997) reported the case of a German farmer who experienced a severe anaphylactic reaction after consuming a saffron rice dish. This reaction was due to high molecular weight proteins (40-90 kDa) present in the saffron extract.

Martínez *et al.* (2007) described the case of a grower with occupational airborne contact dermatitis caused by saffron bulbs. The grower showed eczematous and erythematous lesions on the back of his hands, forearms, neck, face and inframammary region, all associated with cleaning saffron corms previous to planting. These authors discarded the implication of the alpha-methyl-gamma-butyrolactone, which can cause similar symptoms in tulip bulb sensitivity, as the agent responsible for saffron allergy.

Varasteh *et al.* (2007) selected thirty-eight subjects with clinical manifestations of saffron pollen allergy in the nose (sneezing, blockage, and running), eyes (itching, redness, tears, and swelling), mouth and throat (itching and dryness), and chest (breathlessness, cough, wheezing, and tightness) to study saffron pollen allergenicity. In this study, 70% of the subjects showed an immediate reaction to saffron extract. These authors showed the involvement of saffron pollen as an aeroallergen by using skin prick tests with saffron extract in atopic subjects. Sankian *et al.* (2008) purified a profilin from saffron pollen named Cro s 2 involved in occupational allergy. In 2009, Varasteh *et al.* (2009) cloned the saffron profilin, expressed the recombinant rCro s 2 and showed its cross-reactivity with other plant profilins.

Saffron profilin Cro s 2 shared 73% of sequence identity with melon profilin Cuc m 2, but several non-conservative amino acid changes were detected in the regions corresponding to the weak and strong IgE epitopes defined for the melon allergen by López-Torrejón *et al.* (2007).

Recently, Gómez-Gómez *et al.* (2010) studied the involvement of lipid transfer proteins in saffron allergy. They selected six patients on the basis of clinical symptoms suggestive of a type I hypersensitivity reaction to saffron and with positive skin prick test responses and specific IgE to saffron extract. Immunodetection of saffron extract was performed with a rabbit polyclonal antiserum against the peach lipid transfer protein (Pru p 3), as well as with sera from saffron-allergic patients, revealing a band with an apparent molecular weight of approximately 9 kDa. Two LTPs, named rCro s 3.01 and rCro s 3.02 (Accession nº FJ997554, and nº FJ997555), have been isolated and expressed in *Pichia pastoris.* The authors pointed out that rCro s 3.01 and rCro s 3.02 were minor saffron allergens, with this report being the first on the involvement of LTPs in spice allergy. In addition, two allergenic members of the LTP family with a limited amino acid sequence identity (under 50%) have been found in a single plant source. Interestingly, ELISA tests showed cross-reactivity between Pru p 3 and the LTPs isolated from saffron.

Table 2 List of spices allergens included in the Official List of Allergens (International Union of Immunological Societies) updated in 27-01-2010. N.A.: Not available.

Species	Common name	Allergen	Biochemical name	Molecular weight (SDS-PAGE) (kDa)
Apium graveolens	Celery	Api g 1	Pathogenesis-related protein, PR-10	9
		Api g 2	Non specific lipid-transfer protein, type 1	15
		Api g 3	Chlorophyll *a-b* binding protein, chloroplast	N.A.
		Api g 4	Profilin	14
		Api g 5	FAD-containing oxidase	52
Brassica juncea	Oriental mustard	Bra j 1	2S seed storage albumin	N.A.
Sinapsis alba	Yellow mustard	Sin a 1	2S albumin	14
		Sin a 2	11S albumin	51
		Sin a 3	Non specific lipid-transfer protein	12
		Sin a 4	Profilin	14
Capsicum annum	Bell pepper	Cap a 1w	Thaumatin-like protein	23
		Cap a 2	Profilin	14
Sesamum indicum	Sesame	Ses i 1	2S Albumin	9
		Ses i 2	2S Albumin	7
		Ses i 3	7S Vicilin-like globulin	47
		Ses i 4	Oleosin	17
		Ses i 5	Oleosin	15
		Ses i 6	11S Globulin	52
		Ses i 7	11S Globulin	57

ALLERGY TO OTHER IMPORTANT SPICES

Mustard

Mustard, which belongs to the *Brassicaceae* family, includes three types of seeds: yellow (*Sinapsis alba*), brown (*Brassica juncea*) and black (*Brassica nigra*). Black seeds constitute one of the most frequent causes of allergy although they are not widely consumed due to difficulties in harvesting (Uhl 2000). Several reactions to this spice in the form of an immediate skin reaction, angioedema, and anaphylactic shocks have been reported in patients after ingestion of mustard seed flour or manufactured foods (Panconesi *et al.* 1980; Widstrom and Johansson 1986; Jorro *et al.* 1995; Kanny *et al.* 1995; Palomares *et al.* 2007; Sirvent *et al.* 2009). Major allergens of yellow and brown mustard seeds, Sin a 1 and Bra j 1, respectively (**Table 2**), have been characterized as seed storage proteins, belonging to the 2S albumin family, with an approximate molecular weight of 14 to 16 kDa (Menéndez-Arias *et al.* 1988; Gonzalez de la Peña *et al.* 1991). In 2007, an 11S globulin storage protein of 51 kDa was isolated and identified as a novel major allergen of yellow mustard seeds (Palomares *et al.* 2007). More recently, two allergens, Sin a 3 and Sin a 4, corresponding to a non-specific lipid transfer protein and a profiling, respectively, have been isolated. These proteins were IgE-reactive in ELISA and immunoblotting and showed IgE cross-reactivity with fruits such as peach (Pru p 3) and melon (Cuc m 4) (Sirvent *et al.* 2009). Furthermore, Figueroa *et al.* (2005) have proved mustard allergy by double-blind placebo-controlled food challenges. Mustard has been recently included in the list of 12 potential allergenic foods to be labelled in a European Union directive on the identification of foods (Pieretti *et al.* 2009).

Apiaceae spices: anise, fennel, coriander and cumin

Jensen-Jarolim *et al.* (1997) investigated allergens originating from anise (*Pimpinella anisum*), fennel (*Foeniculum vulgare*), coriander (*Coriandrum sativum*) and cumin (*Cuminum cyminum*) spices in 15 patients who showed adverse reaction to spiced food. These patients showed the presence of Bet v 1 homologous in all the spices and the presence of profilin in anise and cumin extracts. High molecular weight proteins binding IgE were also detected. ELISA assays confirmed the role of profilin and Bet v 1. The authors pointed out that only patients with birch and/or mugwort pollinosis and/or celeriac allergy showed positive reactions to the tested spices. These data reinforced the frequent coincidence of the mugwort-celery-syndrome with food allergy to spices (Wuthrich *et al.* 1990; Jensen-Jarolim *et al.* 1997; Egger *et al.* 2006).

Pepper and paprika

Pepper (*Piper nigrum*) and paprika (*Capsicum annum*), belonging to the *Piperaceae* and *Solanaceae* families, respectively, are widely used as spices in international cuisine. Wultrich (1993) studied 402 patients from Central Europe with food allergies, 5.7% of which had allergies to paprika and 1% to pepper. Leitner *et al.* (1998) characterized allergens from these spices in 22 patients suffering from celery-mugwort-spice syndrome. In immunoblotting, 73% and 95% of patients displayed IgE to pepper and paprika proteins, respectively. Two major allergens (of 14 and 28 kDa) were found in pepper and one in paprika (23 kDa). The partial sequence of the 14 kDa pepper allergen did not permit the identification of similarities with others contained in the database banks, while the 28 kDa was a germin-like protein and the 24 kDa paprika allergen was a thaumatin-like protein.

Fenugreek

Fenugreek (*Trigonella foenum-graecum*) is a legume plant with maple-like flavour traditionally used as an ingredient of curry spice mainly added as an aromatic condiment to different manufactured foods. Incidents of IgE mediated allergy to fenugreek have been reported and a cross-reaction with allergy to peanut has been suggested. In 2010, Fæste *et al.* characterized allergens from fenugreek using mass spectroscopy based proteomic strategy. A reactive protein at 50 kDa has been described as the most consistent allergen in all the patients included in this work. The major allergen was characterized as a 7S vicilin-like protein named Tri f 1. Other allergens as pathogenesis-related proteins 10 (Tri f4), 2S albumin (Tri f 2) and 11S legumin (Tri f 3) have been also characterized. These allergens revealed high identities with Ara h 1, Ara h2, Ara h 3, Ara h 4, Ara h 6, Ara h 7 and Ara h 8 isolated from peanut. The authors concluded that significant homologies to major peanut allergens provide an explanation to the proliferation of secondary fenugreek allergy by cross-reaction in peanut allergic patients.

Sesame

Sesame (*Sesamum indicum*) seeds are used for the preparation of a range of traditional dishes and as flavouring agents in the preparation of specialty breads, cakes, and delicacies.

Other common foods containing sesame seeds include vegetarian burgers, spice mixtures, salad dressings and a wide range of diet foods. Sesame oil is also used in pharmaceutical products and the cosmetic industry (Orruño and Morgan 2006). Severe reactions to sesame, including anaphylactic shock, have been reported in the literature (Asero *et al.* 1999; Pajno *et al.* 2000; Dalal *et al.* 2003). Sesame hypersensitivity has been reported both in adult and pediatric populations (Sporik and Hill 1996; Rance *et al.* 1997; Levi and Danon 2001; Dalal *et al.* 2002). Both seeds and oil have been linked to allergic reactions (Chiu and Haydik 1991; Kanny *et al.* 1996; Morisset *et al.* 2003). Hypersensitivity reactions to products containing sesame oil have been described, constituting a risk for atopic patients (Pecquet *et al.* 1998). In 2001, Pastorello *et al.* identified a major sesame seed allergen named ses i 1 and was a 2S albumin with a molecular weight at 9 kDa. Later, Beyer *et al.* (2002) using sera from 20 patients with sesame seed allergy identified 4 sesame seed allergens a protein at 45 kDa, which was recognized by 75% of the patients, was found to be a 7S vicilin-type globulin, a seed storage protein of sesame and named Ses i 3. The protein at 7 kDa was found to be a 2S albumin, another seed storage protein of sesame and named Ses i 2. In addition, the proteins at 78 and 34 kDa were found to be homologous to the embryonic abundant protein and the seed maturation protein of soybeans, respectively. More recently, Leduc *et al.* (2006) showed the implication of two Oleosins named Ses i 4 (17 kDa) and Ses i 5 (15 kDa) in sesame seed allergy.

POLLEN-SPICE ASSOCIATIONS

In general, spice allergy results from cross-reactivity with pollen allergens such as mugwort (*Artemisia vulgaris*, Compositae) and birch (*Betula verrucosa*, Fagales) pollinosis. The resulting cross-allergies are summarized in the two syndromes listed below (Scholl and Jensen-Jarolim 2004; Egger *et al.* 2006).

Celery-mugwort-spice syndrome

This syndrome has been established to describe cross-reactivity between *Artemisia* and *Apiaceae* food (Egger *et al.* 2006). Ten different food allergies are associated to the Celery-mugwort-spice syndrome including *Apiaceae* (e.g. fennel (*Foeniculum vulgare*) and coriander (*Coriandrum sativum*)) and *Liliaceae* (e.g. garlic (*Allium sativum*) and onion (*Allium cepa*)). There is an evident association between birch, mugwort pollinosis and celery hypersensitivity, since these syndromes share common allergens. Patients with allergy to spices are young adults sensitized to mugwort and birch pollen, sharing cross-sensitization with various plants.

Mugwort-mustard allergy syndrome

Caballero *et al.* (2002) researched patients allergic to mustard who suffered from associated pollinosis or allergy to other plant-derived foods. Upon ingestion of mustard, patients showed diverse symptoms ranging from oral allergy syndrome to systemic anaphylaxis. More than 97% of the patients studied were allergic to mugwort, and 100% were sensitized to *Brassicaceae* plants. Using these data, Figueroa *et al.* (2005) suggested the term mugwort-mustard allergy syndrome to describe the association of mustard allergy with mugwort pollinosis and other plant-derived food allergies. Three possible allergens were suggested by Egger *et al.* (2006) that can be involved in this syndrome: Art v 60 kDa, profilin, and the mugwort nonspecific lipid transfer protein (Art v 3). In 2007, Palacin *et al.* searched for potential cross-reacting allergens widely distributed throughout the *Brassicaceae* family, using cabbage leaves (*Brassica oleracea* var *capitata*) as the target food. This approach enabled the authors to detect an LTP, named Bra o 3, as responsible for cross-reactions among *Brassicaceae* foods including mustard. They suggest that the LTP may play a crucial role in the mugwort pollen–mustard allergy syndrome.

OTHER POSSIBLE ASSOCIATIONS

Russian thistle-saffron association

Russian thistle (*Salsola kali*), which belongs to the *Chenopodiaceae* family, is considered the main cause of pollen allergy in arid and semiarid countries. In a recent study describing the sensitization profiles in complex pollen areas in Spain (Barber *et al.* 2008), *Salsola* was found to be the third most frequent cause of pollinosis in the southern part of the country. Sal k 1, a major allergen of *Salsola* (Barderas *et al.* 2007), is practically absent in other *Chenopodiaceae* species. The increase in *Salsola* allergy prevalence seems to be linked to global warming. In semiarid south-eastern areas of Spain, *Salsola* was the most frequent cause of seasonal allergies. An association between *Salsola* and saffron was suggested in a study on occupational allergies in Spanish saffron workers from Ciudad Real (Central Spain), where the climate is semiarid (Feo *et al.* 1997). However, the clinical significance of this finding remains to be elucidated.

FOOD ALLERGY LABELLING AND CONSUMER PROTECTION

Consumers with food allergy occasionally experience allergic reactions from the ingestion of food products containing undeclared ingredients derived from allergenic sources. Hefle *et al.* (2007) researched food products (n = 625 in 2003 and n = 645 in 2006) with advisory labels that listed peanut allergens and whether consumers with allergies ignored these advisory labels or not. The conclusions of this study stated that peanut allergens were detected in 10% of total products and that the percentage of consumers paying attention to advisory labels decreased to 75% in 2006, down from 85% in 2003. Pieretti *et al.* (2009) determined the frequency and language used in 20,241 manufactured food products, and identified labelling ambiguities affecting consumers with food allergy. This study showed that nonspecific terms, such as “spices”, were found on 65% of products and they were not labelled as specific ingredients in 83% of the cases. Allergists should advise their patients with spice allergy to avoid ingestion of packaged food products with ingredients listed as spices or natural flavour. Consumers with food allergy should pay attention to allergy advisory labelling and avoid consumption if they have any doubt about the risk of suffering an adverse reaction. It is also strongly recommended to food manufacturers to make the advisory labels clear and truthful, and not misleading.

CONCLUSIONS

The food industry, especially fast foods and exotic cuisines, have contributed to the spread of spice allergens that can be described as emerging. Anaphylactic recurrences are often associated with the presence of hidden ingredients such as spices and, much more rarely, additives. Among the frequently masked spice allergens, the most common are celery, coriander, anise, cumin, sesame, mustard and saffron. Despite their low incidence, spice allergies can provoke serious problems. Food labels should therefore alert individuals as to hidden ingredients, and a standardized list of allergens would be helpful to many consumers. Patients with food allergies should read the ingredient labels of packaged foods carefully, and allergists must advise their patients of this problem.

ACKNOWLEDGEMENTS

We are grateful for support from the “Junta de Comunidades de Castilla-La Mancha, Fundación de Investigación Sanitaria en C-LM” (FISCAM, PI-2007/61). We wish to thank KA Walsh (Escu-

ela Técnica Superior de Ingenieros Agrónomos, Universidad de Castilla-La Mancha, Albacete, Spain) for language revision. O Ahrazem received funding from the Spanish MICINN through the Ramon y Cajal Programme.

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