A practical focus on sesame allergy and a brief review of other seed allergies

Kelsey Kaman, M.D.[1] and Jeffrey M. Factor, M.D.[1,2]

ABSTRACT

Although seed allergies are relatively rare, sesame allergy has been increasing worldwide and is typically persistent in most patients. Because allergen labeling laws, until just recently, have not required the declaration of sesame as a major allergen, there is a clear need to better understand and potentially treat this food allergy. Although blood and skin prick testing for sesame have limited predictive value, this improves with the use of component diagnostics and skin-prick test with fresh sesame. A thorough history and oral food challenge should be used to diagnose sesame allergy. Treatment of sesame allergy with oral immunotherapy has been demonstrated to be safe and effective in case reports, and in at least one controlled study with a published sesame oral immunotherapy protocol. There is minimum literature with regard to management of other seed allergies.

(J Food Allergy 4:151–157, 2022; doi: 10.2500/jfa.2022.4.220008)

SESAME SEED ALLERGY OVERVIEW

The prevalence of sesame allergy varies throughout the world. In the United States, it is estimated to range from 0.1% to 0.24% of the population,[1] which makes it the ninth most common U.S. food allergen, and the most common seed allergy.[2] A sesame allergy typically appears early in life, on average between 6 months and 3.5 years of age[2–5]; however, in one study, >25.7% of the adults with sesame allergy reported an adult onset of sesame hypersensitivity.[2] Unfortunately, only ~20%–30% of individuals will outgrow their sesame allergy and the majority will remain allergic.[1] Among patients with multiple food allergies, 50% of them report their sesame allergy results in greater time burdens than their other food allergies.[5]

SESAME PROTEIN

Traditionally, sesame was used as a source of oil, but, more recently, it has been incorporated into Western diets given the increased awareness of nutritional benefits.[6,7] Sesame is often included in foods that contain other allergens, which may make it difficult to identify the responsible culprit. In addition, sesame protein has been identified as having electrostatic properties, which makes it problematic to avoid cross-contact during food production.[8] As of 2021, the United States joined the European Union, Canada, and Australia in mandating precautionary food labeling for sesame.[1] Common products that may contain sesame include foods such as breads and baked goods, cereal, candy, crackers, snacks, marinades, dressings, soups, oils, and many ethnic dishes. Sesame may also be found in cosmetics such as makeup products, soaps, and ointments as well as in pharmaceutical products such as progesterone, testosterone, and dronabinol. Typically, refined sesame oils are used in medications and are usually not allergenic, although delayed hypersensitivity has been described. Unrefined sesame oils are often used in baking, and these have been associated with episodes of anaphylaxis. Whole sesame seeds are often better tolerated among individuals with sesame allergy, and this is postulated to be due to food processing and heating, and the ability to travel through the gut undigested.[4,6,9] The key sesame seed allergens have been identified. They are known as Ses i 1 through 8. The protein type of each sesame seed allergen is described in Table 1.

CROSS-REACTIVITY

Among individuals with sesame allergy, 81.6% reported additional food allergies, with peanut being the most common, at 46.9%.[2] One study found that, among children with sesame allergy, 61.9% had more than five food allergies.[5] Between 58% and 99% of patients with sesame allergy may demonstrate sensitization to peanuts. Ses i 3 has been found to have 80% homology with the peanut allergen Ara h 1, which likely explains the cross-

From the [1]Connecticut Asthma and Allergy Center, West Hartford, Connecticut; and [2]New England Food Allergy Treatment Center, West Hartford, Connecticut
The authors have no conflicts of interest to disclose pertaining to this article
No external funding sources reported
Address correspondence to Kelsey Kaman, M.D., 836 Farmington Ave 207 West Hartford, CT, 06119
E-mail address: kkaman@ctallergy.net
*This manuscript is part of the **Journal of Food Allergy** collection of published works referred to as the "Oral Immunotherapy Manual." The contents of this work reflects the opinion(s) of the author(s) and is not intended to replace published guidelines or the clinician's medical advice in the doctor-patient relationship*

Table 1 **Sesame allergen components***

Ses i 1	2S albumin
Ses i 2	2S albumin
Ses i 3	7S vicilin-like globulin
Ses i 4	Oleosin
Ses i 5	Oleosin
Ses i 6	11 S globulin
Ses i 7	11 S globulin
Ses i 8	Profilin

From Ref. 4.

Table 2 **Sesame protein content of foods**

Food	Protein Content, mg
Halvah (Brooklyn, NY), 5 g	500
Nature's Promise tahini (Ahold Delhaize Carlisle, PA), 1/2 tsp	500
Sesame flour (33.3% protein), 1.5 g	500

reactivity.[10] Clinically, ~40% of patients with sesame allergy will react to peanuts and 25% of patients with peanut allergy will react to sesame.[1,4] Alternatively, other studies found that sesame allergy does not increase the odds of having a peanut allergy or vice versa.[10]

Among individuals with sesame allergy, 40% demonstrated hypersensitivity to at least one tree nut.[1] Individuals with sesame allergy are nine times as likely to be allergic to pine nuts and 4.6 times as likely to be allergic to Brazil nut than those without sesame allergy. Alternatively, individuals with macadamia nut allergy were 8.8 times as likely to be allergic to sesame and those with hazelnut allergy were 3.6 times as likely to be allergic to sesame.[10] Serologic cross-sensitization has also been found to exist between sesame and black walnut, cashew, and pistachio[4]; however, these studies did not perform oral food challenges; therefore, it is unclear what percentage of these patients were only sensitized.

DIAGNOSIS

With regard to blood testing for sesame allergy, one study showed a 7 kU/L value that resulted in a 0.12 specificity and a 0.17 sensitivity.[6] Another study demonstrated an immunoglobulin E (IgE) value of 11.6 kU/L to provide a sensitivity of 0.93 and a specificity of 0.85.[1] A third study demonstrated that an IgE level of 50 kU/L would be required to obtain an 86% positive predictive value.[4] Among individuals with proven sesame allergy on oral challenge, the sesame IgE value was positive in only 2 of 16 participants.[9] With notable variability that has not been shown to correlate with oral food challenge outcomes, it has been suggested that the sesame IgE value should not be used independently to predict sesame allergy.[11,12]

With regard to skin-prick testing, a wheal ≥ 8 mm has been found to have a 95% positive predictive value, but this varies, depending on the source of the allergen used for testing.[4] A commercial extract for skin-prick testing has been shown to have 33.3%–54% sensitivity and 38%–86.4% specificity, which results in a positive predictive value of 77.8% and a negative predictive value of 18.2%.[6,9] The poor sensitivity with sesame commercial extracts is thought to be due to removal of the allergenic lipid derivatives (oleosins) during processing.[4,12] Alternatively, sesame paste for skin testing has proven to be more helpful as a screening tool. Sensitivity increases to 86.7%–100% and specificity decreases to 52.6%–82.1%.[4,9,12,13] Therefore, those who test negative to commercial sesame may test positive to a sesame paste.[4,9,12,13]

Sesame component-resolved diagnostics have begun to be used for a better understanding of sesame allergy. Although Ses i 4 and Ses i 5 have been associated with more severe systemic reactions,[9] Ses i 1 has been shown to be most useful for diagnostic utility. One study showed an elevated Ses i 1 level to 3.96 kU/L results in 86.1% specificity and 85.7% specificity,[1] whereas another study showed levels of 0.3 kU/L to have a sensitivity of 58.3% and specificity of 83.3%, which resulted in a significant area under the curve (AUC) of 0.715.[11] Unfortunately, component-resolved diagnostic testing for sesame is not widely available in commercial laboratories at this time. Given reports of patients with a negative sesame skin-prick test result and/or IgE value, if there is a reliable history of sesame reaction, in-office oral challenge should be considered.[4] Target doses for oral food challenge have ranged from 4.4 to 4.5 g.[1,9] Of note, sesame oral food challenges compared with those with tree nuts have higher severity symptoms scores, including more involvement of the lower respiratory tract, cardiovascular, or neurologic symptoms.[10]

IMMUNOTHERAPY

Although limited reports exist in the literature, sesame is the most common of the seeds described in oral immunotherapy cases. To our knowledge, there are no published data that compared different food sources of sesame for use in oral immunotherapy. Despite this, because black sesame seeds are unhulled, with a lower protein content compared with white sesame seeds, they are hypothesized to be less allergenic. In addition, given the variability in the absorption of seeds, the recommendations are either to grind seeds before use in oral immunotherapy or ensure that the patient thoroughly chews the seeds. Alternatively, practices may decide that formulations such as sesame flour, tahini,

Table 3 **Phase I of oral immunotherapy***

Phase 1	Dose, 5 doses/day
Week	
1	
Day	
1	0.1 μg#
2	0.1 μg
3	0.1 μg
4	0.1 μg
5	0.1 μg
6	0.1 μg
7	0.1 μg
2	0.2 μg
3	0.5 μg
4	1.0 μg
5	2.0 μg
6	5.0 μg
7	10 μg
8	20 μg
9	50 μg
10	100 μg
11	200 μg
12	500 μg
13	1.0 mg
14	2.0 mg
15	5.0 mg
16	10.0 mg

*From Ref. 16.
#The first day's doses were as follows: 0.1 μg (30 min); 0.1μg (2 hr); 0.1μg, 0.1μg, 0.1μg (over 4 hr).

Table 4 **Oral immunotherapy for sesame at the New England Food Allergy Treatment Center**

Visit (every 2–4 wk)	Dose, mg
1	0.1, 0.2, 0.4, 0.8, 1.5, 3
2	3
3	4.5
4	6
5	9
6	12
7	18
8	25
9	35
10	50
11	75
12	100
13	125
14	165
15	225
16	300
17	400
18	500 (protein)
19	600 (protein)

or halva have more consistent bioavailability. This is supported by the recent publication by Ocak *et al.*,[12] who found that, among patients who tolerated sesame seed oral challenge, 55% would go on to develop symptoms of IgE-mediated allergy with a tahini challenge. The protein content of commonly used sesame products can be seen in Table 2.

Given the cross-reactivity between peanuts and seed storage proteins, one study examined patients who were allergic to both peanuts and sesame yet who underwent only peanut oral immunotherapy and were followed up for 24 months.[14] They found that these patients had an increase in their sesame IgE levels from 0 to 12 months and then no significant change otherwise. Unfortunately, no further testing or oral challenges were subsequently performed.[14]

In 2014, Bégin *et al.*[15] described their approach to multiple-allergen oral immunotherapy. Among their cohort, they included six children with peanut allergy who were also allergic to sesame. The patients underwent three phases of initial escalation, then home dosing, followed by a maintenance phase. During the initial escalation day, the patients started at 0.1 mg, increasing to 5 mg.[15] For home dosing they advanced dosing every other week, increasing from 6.0 mg up to 4.0 g, depending on patient and safety parameters.[15] During the maintenance phase, they continued on 4.0 g daily.[15] Most patients had mild symptoms on the initial escalation day. There were no increased adverse rates compared with those only on peanut oral immunotherapy, but it was noted that it took much longer to reach target doses.[15]

The first case published with regard to solo sesame oral immunotherapy was published in 2019.[16] An adult woman with sesame allergy that resulted in seven episodes of anaphylaxis presented with negative IgE sesame test result but with a positive skin test result to tahini and sesame seed. The first phase of immunotherapy can be seen in Table 3. During the second phase, she was given three doses per day of 10 mg of white sesame seeds for 8 weeks, each week escalating to reach a dose of 150 mg. During the third phase, she was given tahini with two doses per day, starting at 200 mg/dose, increasing twofold each week up to 800 mg/dose. In the final phase, she was given one dose of tahini per day beginning with 1.5 g/dose the first week and 3.0 g/dose the second week, and then maintained for 4 weeks. During desensitization, she periodically experienced minor symptoms of itching and wheals, which were able to be controlled with antihistamines. She subsequently had negative skin-prick test results to sesame and was able to pass sesame oral food challenge.[16]

Table 5 **Sunflower seed oral immunotherapy dosing***

Visit (every 2– 4 wk)	Dose, mg
1	7
2	13
3	26
4	52
5	104
6	207
7	415
8	830
9	1660

*From Ref. 17.

Table 6 **Poppy seed oral immunotherapy dosing***

Visit (every 2–4 wk)	Dose, mg
1	4
2	8
3	16
4	31
5	62
6	125
7	250
8	500
9	1000

*From Ref. 17.

Shortly after, also in 2019, Nachshon *et al.*[7] described the first cohort of patients to successfully undergo oral immunotherapy to sesame. Throughout oral immunotherapy, a liquid form of a high-protein sesame extract was used for doses < 240 mg, otherwise raw tahini was used for higher doses.[7] During the first phase, the patients underwent oral challenge to identify the single highest tolerated dose. Those able to tolerate 4800 mg of sesame protein were determined to be nonallergic.[7] The patients who were allergic were instructed to consume the single highest tolerated dose once daily at home for 24 days. Subsequently, the patients underwent an updosing cycle over 4 days to achieve a fourfold dose increase. This was then followed by 24 days of home consumption of this dose.[7] The third phase included 3 days of increasing to a threefold goal dose.[7] The fourth and the fifth cycle consisted of 2 days each with a twofold dose increase.[7] The patients then arrived for a 1-day cycle with a goal of 50% dose increase until reaching a goal of 4000 mg.[7] Once fully desensitized, they were maintained on 1200 mg daily.[7] This was meant to mirror peanut oral immunotherapy studies in which a low-maintenance dose can maintain full desensitization but also improve adherence.

Over 6.5 months, 53 of 60 patients were able to achieve full desensitization.[7] The remaining seven patients were able to become partially desensitized to > 240 mg of sesame protein. There were no significant identifiers among those patients able to become partially or fully desensitized. Predictors included a low starting dose and a high rate of reactions during the buildup hospital visits.

During oral immunotherapy, 4.7% of induction doses and 2% of home doses were associated with adverse reactions. Epinephrine was required in 16.7% of the patients during the hospital buildup phase and 8.3% during home treatment. Three patients developed gastrointestinal symptoms and eosinophilia that resolved after a month of lower dosing and they subsequently were able to undergo complete desensitization. Given the limited publications with regard to sesame oral immunotherapy, it is unclear if this reaction rate is secondary to sesame itself or attributed to the unique escalation protocol. Testing of the patients demonstrated no significant change in IgE levels, but a significant decrease in wheal size, decrease in basophil activation, and an increase in the IgG4 level to sesame and to Ses I 1.[7]

At our practice, the New England Food Allergy Treatment Center in West Hartford, Connecticut, we have successfully desensitized 28 patients to sesame by using the protocol in Table 4. Doses of ≤400 mg use sesame flour, whereas larger doses use tahini, Joyva Marble Halvah, or Joyva Marble Halvah Bar (Joyva; Brooklyn, NY). Visits with patients to updose occur every 2 weeks as tolerated.

OTHER SEEDS

Poppy, flax, pumpkin, mustard, and sunflower seeds have all been reported to cause immediate hypersensitivity, yet this remains rare. Therefore, there is limited literature available to provide guidance with regard to management. There are no longitudinal studies that describe the average duration or persistence of these seed allergies.[4] One published study used sunflower seed (n = 4), poppy seed (n = 1), flaxseed (n = 1), and pumpkin seed (n = 1) in multiallergen oral immunotherapy.[17] Protocol doses can be found below. Although there are no specifics with regard to the outcomes among these patients, none of those receiving seed oral immunotherapy required an emergency department visit during their treatment.[17]

Sunflower Seed

Sunflower seeds are incorporated into diets for their nutritional benefits, either raw or roasted, or used in baked goods. Sunflower oil is also used in the diet, and, although, it tends to be tolerated by those with sunflower seed allergy, this is not a uniform finding. Three

Table 7 **Flaxseed oral immunotherapy dosing***

Visit (every 2–4 wk)	Dose, mg
1	3
2	7
3	13
4	27
5	53
6	107
7	213
8	427
9	854

*From Ref. 17.

Table 8 **Pumpkin seed oral immunotherapy dosing***

Visit (every 2–4 wk)	Dose, mg
1	6
2	11
3	23
4	46
5	92
6	183
7	367
8	733
9	1466

*From Ref. 17.

major allergens have been identified in sunflower seed. Hel a 1 is an inhalatory allergen, cross-reactive with sunflower pollen. Hel a 2 is a profilin and is also an inhalatory allergen. Hel a 3 is a lipid transfer protein. Hel a 2S albumin and Hel a 3 have been postulated as the major food allergens in sunflower seed. These allergens are also highly heat resistant.[4,18] Sunflower seed allergens have been found to cross-react with other allergens in the Asteraceae family. Hel a 2 cross-reacts with ragweed, mugwort, and *Mercurialis perennis.* Hel a 3 has demonstrated homology with hazelnut and peanut allergens, although the clinical implications of this are unclear.

Clinically, patients with sunflower allergy have been reported to also react to Brazil nut, pistachio, mustard, and mugwort, with laboratory demonstration of protein homology.[4,18] Sunflower seed allergy has been reported to result in IgE-mediated symptoms with ingestion, occupational allergy, contact urticaria, angioedema, and contact dermatitis.[4,19] Because sunflower seeds are also often found in bird feed, this may account for sensitization in some individuals. One study showed that, among bird breeders, 79% tested positive to sunflower skin testing, yet none had allergic symptoms with exposure.[18] Sunflower butter was used when possible, or parents were instructed to finely chop the seeds to a flour consistency.[17] Dosing steps can be seen in Table 5.[17]

Poppy Seed

Poppy seed is derived from the weed Papaveraceous. It is often used for garnishing or making breads and cakes. Although there are limited studies that examined the specific allergenic proteins, one study identified a 40- and 45-kDa glycoprotein with IgE-binding moieties.[20] These allergens are notably resistant to high temperatures. Among the cases of poppy seed allergy that have been published, ~72% had coexisting tree nut allergy and 10% had coexisting sesame seed allergy.[19] Poppy seed has been found to be homologous with Bet v 1 and Bet v 2, which may explain the co-occurrence of symptoms among those with pollen or tree nut hypersensitivity.

Table 9 **Integrating seeds into the diet**

Mix in pudding, yogurt, or applesauce
Add to smoothies
Homemade hummus
Baked into brownies or cookies

Serologic and *in vitro* tests also demonstrate cross-reactivity to rye, kiwi, and buckwheat.[4,19] Poppy seed allergy has been reported to result in IgE-mediated symptoms with ingestion, hypersensitivity by inhalation, and food-dependent exercise-induced hypersensitivity.[4] Poppy seed was reported in one study of multiallergen oral immunotherapy.[17] Dosing steps are included in Table 6.[17]

Flaxseed

Flaxseed is commonly incorporated into baked goods and cereals. The major allergen identified thus far is a 56-kDA dimer malate dehydrogenase. Serologic studies demonstrate cross-sensitization with lupine, peanut, soybean, rapeseed, rape pollen, and wheat, but the clinical implications of this were not analyzed. Flaxseed allergy has been reported to result in IgE-mediated symptoms with ingestion and occupational allergy. Of note, among 77 patients who tested positive to flaxseed on skin testing, only two had clinical reactions.[4] Flaxseed was reported in one study of multiallergen oral immunotherapy, provided in the form of ground flaxseed. Dosing steps are included in Table 7.[17]

Pumpkin Seed

Although pumpkin seed allergens have not been well defined, a profilin homolog of 14 kDA and a heat-stable lipid transfer protein of 12 kDA have been identified as potential culprits. Pumpkin seed allergy has been reported to result in IgE-mediated symptoms with ingestion. Some patients with pumpkin seed

allergy have been reported to tolerate pumpkin pulp, whereas other patients may react. Clinical co-reactivity has been reported to peach, apple, pear, cucumber, and melon. Cross-sensitization has been reported between pumpkin pulp and peach.[4] Pumpkin seed was reported in one study of multiallergen oral immunotherapy, with dosing steps included in Table 8.[17]

Mustard Seed

Mustard seed comes in two forms: white and brown. White mustard seed, *Sinapis alba* L., is commonly known as yellow mustard. It is typically used as a condiment or a spice. Brown mustard seed, *Brassica juncea* L., is commonly known as oriental mustard. The major allergen of white mustard, Sin a 1, a 2S albumin storage protein, is resistant to high heat and gastric digestion. Sin a 2 is an 11S globulin. Sin a 4 is a nonspecific lipid transfer protein. Sin a 4 is a profilin. Sin a 2, Sin a 3, and Sin a 4 are presumably responsible for cross-reactivity. Mustard seed allergens have demonstrated cross-reactivity to peach, melon, *Artemisia vulgaris,* sesame, walnut, hazelnut, peanut, pistachio, Brazil nut, cashew, and pecan.[21] Mustard seed allergy has been reported to result in IgE-mediated symptoms with ingestion as well as contact dermatitis.[4,21] Skin testing to raw extracts of mustard is superior to using commercial extracts or IgE testing.[4]

Chia Seed

Chia seed has been incorporated in the diet as a medication historically and more recently for its health benefits. Although there are anecdotal reports of chia seed hypersensitivity, there do not seem to be any published reports. Despite this, chia seed allergenicity *in vitro* has been investigated. The main chia seed proteins are cupin superfamily storage proteins. There has not yet been an investigation into which proteins are responsible for IgE binding. *In vitro* studies demonstrate cross-reactivity between chia storage proteins and other seed allergens (particularly sesame) as well as to hazelnut.[22]

CONCLUSION

With the increasing incidence of sesame seed allergy, as well as the significant impact on quality of life, utilization in a variety of foods, and the risk for cross-contamination, there is a driving need to provide accurate clinical information and the option of treatment for these patients. Despite an extensive literature search, as far as we are aware, there is only one case-study protocol and one case series protocol published for sesame oral immunotherapy.[7,16] Reassuringly among these published cases, 54 patients were able to be safely and fully desensitized and the remaining 7 patients were able to be partially desensitized. There is minimum literature available with regard to other seed oral immunotherapy, and often this is used in multifood regimens. Tips to integrate seeds into the diet can be seen in Table 9.

CLINICAL PEARLS

- Given poor diagnostic modalities and higher rates of severity symptom scores, sesame oral challenge and desensitization should be approached with caution and supportive treatment readily available.[4,10]
- Despite the need for precautions, oral immunotherapy to sesame (including multifood desensitization) has been shown to be safe. Given the frequency of permanence[1] and significant impact sesame allergy has on quality of life, it is likely that providing oral immunotherapy can decrease burdens faced.[5,7,16,17]
- Despite cross-reactivity between sesame and peanuts, it is unknown yet if peanut oral immunotherapy decreases clinical reactivity to sesame.[1,4,16]

REFERENCES

1. Sokol K, Rasooly M, Dempsey C, et al. Prevalence and diagnosis of sesame allergy in children with IgE-mediated food allergy. Pediatr Allergy Immunol. 2020; 31:214–218.
2. Warren CM, Chadha AS, Sicherer SH, et al. Prevalence and severity of sesame allergy in the United States. JAMA Netw Open. 2019; 2:e199144.
3. Garkaby J, Epov L, Musallam N, et al. The sesame-peanut conundrum in Israel: reevaluation of food allergy prevalence in young children. J Allergy Clin Immunol Pract. 2021; 9:200–205.
4. Patel A, Bahna SL. Hypersensitivities to sesame and other common edible seeds. Allergy. 2016; 71:1405–1413.
5. Lemoine-Courcelles C, Abrams EM, Kim H, et al. Sesame allergy a time burden for families of children with multiple food allergies. J Allergy Clin Immunol Pract. 2021; 9:1753–1755.e1.
6. Ziegler JB, Aalberse RC. Sesame: an increasingly popular word and common food allergen. J Allergy Clin Immunol Pract. 2020; 8:1689–1691.
7. Nachshon L, Goldberg MR, Levy MB, et al. Efficacy and safety of sesame oral immunotherapy–a real-world, single-center study. J Allergy Clin Immunol Pract. 2019; 7:2775–2781.
8. Dano D, Remington BC, Astier C, et al. Sesame allergy threshold dose distribution. Food Chem Toxicol. 2015; 83:48–53.
9. Salari F, Bemanian MH, Fallahpour M, et al. Comparison of diagnostic tests with oral food challenge in a clinical trial for adult patients with sesame anaphylaxis. Iran J Allergy Asthma Immunol. 2020; 19:27–34.
10. Brough HA, Caubet J-C, Mazon A, et al. Defining challenge-proven coexistent nut and sesame seed allergy: a prospective multicenter European study. J Allergy Clin Immunol. 2020; 145:1231–1239.
11. Saf S, Sifers TM, Baker MG, et al. Diagnosis of sesame allergy: analysis of current practice and exploration of sesame component Ses i 1. J Allergy Clin Immunol Pract. 2020; 8:1681–1688.e3.
12. Ocak M, Murat Sahiner U, Soyer O, et al. The role of diagnostic tests and oral food challenge results to predict sesame allergy. Ann Allergy Asthma Immunol. 2022; 128:46–52.e1.
13. Epov L, Garkaby J, Almog M, et al. Using skin prick test to sesame paste in the diagnosis of sesame seed allergy. J Allergy Clin Immunol Pract. 2020; 8:1456–1458.

14. Bird JA, Kulis M, Burk CM, et al. Tree nut- and sesame-specific IgE do not decrease from baseline with peanut oral immunotherapy (OIT). Ann Allergy Asthma Immunol. 2012; 109:470–471.
15. Bégin P, Winterroth LC, Dominguez T, et al. Safety and feasibility of oral immunotherapy to multiple allergens for food allergy. Allergy Asthma Clin Immunol. 2014; 10:1. Erratum in: Allergy Asthma Clin Immunol. 2016;12:28.
16. Costa Paschoalini M, Bernardes AF, Buzolin M, et al. Successful oral desensitization in sesame allergy in an adult woman. J Investig Allergol Clin Immunol. 2019; 29:463–465.
17. Eapen AA, Lavery WJ, Siddiqui JS, et al.. Oral immunotherapy for multiple foods in a pediatric allergy clinic setting. Ann Allergy Asthma Immunol. 2019; 123:573–581.
18. Ukleja-Sokołowska N, Gawrońska-Ukleja E, Żbikowska-Gotz M, et al. Sunflower seed allergy. Int J Immunopathol Pharmacol. 2016; 29:498–503.
19. Keskin O, Sekerel BE. Poppy seed allergy: a case report and review of the literature. Allergy Asthma Proc. 2006; 27:396–398.
20. Jensen-Jarolim E, Gerstmayer G, Kraft D, et al. Serological characterization of allergens in poppy seeds. Clin Exp Allergy. 1999; 29:1075–1079.
21. Pałgan K, Żbikowska-Gotz M, Bartuzi Z. Dangerous anaphylactic reaction to mustard. Arch Med Sci. 2018; 14:477–479.
22. Albunni BA, Wessels H, Paschke-Kratzin A, et al. Antibody cross-reactivity between proteins of chia seed (*Salvia hispanica* L.) and other food allergens. J Agric Food Chem. 2019; 67:7475–7484. □