Sunflower (*Helianthus annuus*) Seed Allergy

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Abstract

Background: Sunflower seed is one of the most common edible seeds and its consumption is growing. Case reports of sunflower seed allergy have been described since the 1970s. However, there are few data on the prevalence and clinical manifestations of sunflower seed allergy.

Objective: To improve understanding of sunflower seed allergy.

Methods: We evaluated the clinical and immunological features of patients with sunflower seed allergy diagnosed in the Allergy Department of a tertiary hospital in Madrid over a 5-years period.

Results: Forty-seven patients reported adverse reactions after ingestion of sunflower seed and had specific sensitization to sunflower seed determined by skin prick test (median 8 mm) or specific IgE (median 1.10 kUA/L). Most had an adult-onset reaction to sunflower seed preceded by a history of atopy and other food allergies, predominantly to peach, peanut and nuts. Clinical presentation of sunflower seed allergy ranged from mild to severe, with a high proportion of patients suffering severe reactions, often undertreated. A variability in the severity of symptoms was seen on repeated exposures to sunflower seed on a same patient. Levels of sunflower seed IgE were strongly correlated with levels of IgE to non-specific lipid transfer proteins, while no significant differences were found in the severity of the reactions according to sensitization to those proteins.

Conclusion: Our findings reveal a variability of clinical presentations of sunflower seed allergy on repeated exposures and an underuse of epinephrine in anaphylaxis. We highlight the importance of strict avoidance of sunflower seed and accurate prescription and administration of epinephrine in allergic patients.

Key words: Sunflower seed. Food hypersensitivity. Severity. Skin prick test. Specific IgE.

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Resumen

Antecedentes: La semilla de girasol es una de las semillas comestibles más frecuentes y su consumo está creciendo. Desde los años 70 se han descrito casos de alergia a las semillas de girasol. Sin embargo, hay pocos datos sobre la prevalencia y las manifestaciones clínicas de la alergia a la semilla de girasol.

Objetivo: Mejorar la comprensión de la alergia a la semilla de girasol.

Métodos: Evaluamos las características clínicas e inmunológicas de pacientes con alergia a la semilla de girasol diagnosticados en el Servicio de Alergología de un hospital terciario de Madrid durante un periodo de 5 años.

Resultados: Cuarenta y siete pacientes reportaron reacciones adversas tras la ingesta de semillas de girasol y presentaron sensibilización a semilla de girasol determinada mediante prueba cutánea (mediana 8 mm) o IgE específica (mediana 1,10 kUA/L). La mayoría de las reacciones se iniciaron en la edad adulta precedidas de antecedentes de atopia y otras alergias alimentarias, predominantemente a melocotón, cacahuete y frutos secos. Las manifestaciones clínicas de la alergia a la semilla de girasol variaron de leves a graves, con una alta proporción de pacientes con reacciones alérgicas graves, a menudo infratratadas. Se observó una variabilidad en la gravedad de los síntomas en exposiciones repetidas a semillas de girasol en un mismo paciente. Se objetivó una fuerte correlación entre los niveles de IgE de semilla de girasol y los niveles de IgE a proteínas de transferencia de lípidos no específicas, sin encontrar asociación entre el grado de sensibilización a estas proteínas y la gravedad de las reacciones.

Conclusión: Los resultados revelan una variabilidad en las manifestaciones clínicas de la alergia a la semilla de girasol en exposiciones repetidas y un infrauso de adrenalina en la anafilaxia. Enfatizamos la importancia de la evitación estricta del consumo de semillas de girasol y la adecuada prescripción y administración de adrenalina en los pacientes alérgicos.

Palabras clave: Semilla de girasol. Hipersensibilidad a alimentos. Gravedad. Prick test. IgE específica.

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Summary box:

- **What do we know about this topic?** The consumption and prevalence of seeds allergy is subsequently increasing. However, there are few data on the overall prevalence and clinical manifestations of sunflower seed allergy.
- **How does this study impact our current understanding and/or clinical management of this topic?** Our findings reveal a variability of clinical presentations of sunflower seed allergy on repeated exposures and an underuse of epinephrine in anaphylaxis. We highlight the importance of strict avoidance of sunflower seed and accurate use of epinephrine in allergic patients.

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Introduction

Sunflower (Helianthus annuus) is a genus of plants in the Asteraceae family. The edible seeds of the sunflower plant are commonly consumed in snacks, breads or oils,[1] being the most popular food eaten during football matches in Spain and many other European countries.

With the inclusion of seeds in many foods, the consumption of edible seeds such as sunflower seed, sesame, mustard, poppy, pumpkin seed or flaxseed is growing worldwide and the prevalence of allergy to seeds is subsequently increasing.[1-7] Sesame seed seems to be the most common cause of seed allergy, with a worldwide prevalence of sesame allergy between 0.1% and 0.2%.[3] The prevalence of sensitization to sunflower seed in Europe has been investigated in the EuroPrevall project by averaging the prevalence over different centers of each country. The authors described a prevalence of sunflower seed sensitization ranging from 0% in countries such as Iceland to 4.8% in France, with a prevalence of 1.9% in Spain.[7] Cultural dietary habits might contribute to these differences. However, sunflower seed allergy overall prevalence and clinical manifestations have yet to be fully examined.

Despite frequent consumption, since the first sunflower seed allergy reported by Noyes et al.[8] in 1979, publications on sunflower seed allergy have been limited to case reports or case series with a small number of patients.[1-21]

This study sought to evaluate the clinical and immunological features of IgE-mediated hypersensitivity to sunflower seed and other food allergies present in sunflower seed allergic patients.

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Methods

Subjects

A retrospective case series study was conducted at the Allergy Department at the Hospital Universitario 12 de Octubre in Madrid over 5 years. Inclusion criteria were patients with clinical reactivity to sunflower seed, comprising patients who reported adverse reactions on exposure to sunflower seed confirmed by specific sensitization. Forty-seven patients with clinical reactivity to sunflower seed were evaluated. Informed consent of the patients was not required due to the retrospective nature of the study and the anonymization of clinical data. The study was approved by the Ethics Committee of the Hospital Universitario 12 de Octubre, Madrid, Spain (Approval No. 19/554).

Diagnostic procedures

First, we collected data regarding the history of atopy, food reactions, other comorbidities, number of reactions to sunflower seed, age at first reaction, symptoms, route of exposure, time between exposure and onset of the symptoms, treatment in the emergency department and medications used to treat the reactions.

Secondly, we recorded the results of the skin and in vitro diagnostic tests performed in our center.

Skin prick test (SPT) and prick-prick test (P-P) were performed according to the standard technique by trained technicians in our center. P-P were performed using fresh sunflower seeds, peanuts, nuts (walnut, almond, hazelnut, pistachio, cashew, pine nut and chestnut) and other foods reported as offenders of reactions in the patients' clinical history. SPT with commercially available panallergens extracts profilin (LETI®) and peach non-specific lipid transfer protein (ns-LTP) Pru p 3 (commercial extract from Alk-Abello®; concentration of Pru p 3 30 μg/ml) were performed. Positive results on P-

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P and SPT were defined as a wheal size at least 3 mm greater than the negative control. Histamine and saline (Alk-Abello®) were used as positive and negative controls respectively.

Total IgE and specific IgE against sunflower seed, peanuts, nuts, other foods and panallergens were measured using ImmunoCAP (Thermo Fisher Scientific®) following the manufacturer's instructions. Within the ns-LTPs group, specific IgE to rPru p 3, rAra h 9, rJug r 3 and rCor a 8 were tested. Specific IgE to other recombinant proteins, such as profilins (rPru p 4) and 2S albumins (rAra h 2 and rJug r1) were also evaluated. Specific IgE levels above 0.35 kUA/L were considered positive.

The concept of allergy was used according to the definition of the European Academy of Allergy and Clinical Immunology's (EAACI) Food Allergy and Anaphylaxis Guidelines.[22] IgE-mediated symptoms on exposure to sunflower seed and specific sensitization were required to diagnose sunflower seed allergy (Figure 1). Reactions were classified according to the World Allergy Organization (WAO) Systemic Allergic Reaction Grading System[23] in 5 grades based on the organs or systems involved and the severity of the reaction.

Specific sensitization to sunflower seed was demonstrated in all patients through skin tests and/or specific IgE. Clinical reactivity was confirmed by food challenge in 3 patients, including 2 double-blind placebo-controlled oral food challenges and 1 open food challenge.

Statistical analysis

Descriptive statistics of the quantitative variables, including number of subjects (n), median (P50) and interquartile range (IQR: P25-P75) were used. Qualitative variables were described using frequency distributions (absolute and relative).

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Inferences were estimated depending on the nature of the variables using chi-square test or Fisher's exact test for categorical variables and non-parametric Mann-Whitney or Krustal-Wallis U test for continuous variables. The correlation between severity of reactions to sunflower seed and IgE levels to sunflower seed and other allergens was measured through Spearman's Rank Correlation Coefficient. All analyses were done using Stata Intercooled for Windows version 16 StataCorp. 2019. Stata Statistical Software: Release 16. College Station, TX: StataCorp LLC) and a significance level of 5%.

Results

Fourty seven patients (median age 36 years; male/female ratio 0.88) with sunflower seed allergy were evaluated. Table 1 shows demographic features, symptoms, skin tests and specific IgE to sunflower seed and food proteins and other previous food allergies in 47 patients with sunflower seed allergy.

Clinical features

The onset of the symptoms occurred mostly in adulthood. Twelve patients (25%) reported an onset of symptoms under 18 years. Age of onset before 18 years was found in 43% of patients who reported sunflower seed allergy as their first food reaction, compared to 16% of patients with other previous food allergies (p 0.013).

Clinical manifestations appeared within 30 minutes after sunflower seed ingestion in all patients. Two patients described additional routes of reactions besides ingestion, including symptoms on cutaneous contact (patient number 8) and symptoms on inhalation (patient number 7).

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Figure 2 shows the classification of the reactions according to the WAO Systemic Allergic Reaction Grading System. Twenty-three out of 47 patients (49%) reported severe or life-threatening reactions (grade 3 or higher), including bronchospasm, laryngeal edema, gastrointestinal symptoms, collapse or hypotension. Emergency medical services were requested in 18 patients (38%) and 4 patients (8%) received epinephrine.

Sixteen patients reported more than one reaction to sunflower seed before the diagnosis. Among them, 10 patients (62%) reported different clinical profiles of reactions on each exposure; the variability of symptoms in the reactions of those patients is represented in Table 2.

Other allergies or comorbidities

A history of rhinitis, asthma or atopic dermatitis was documented in 41 patients (87%). Forty-three patients (91%) had a history of other food reactions. No significant differences were found in the history of atopy or other food reactions according to the age of onset of sunflower seed allergy (younger or older than 18 years).

Sunflower seed was the first food allergy reported in 16 patients. Most patients with sunflower seed allergy had a history of other food allergies preceding the first reaction to sunflower seed (31 patients; 66%). Most frequent food allergies preceding sunflower seed allergy diagnosis were caused by peach (34%), followed by peanut (30%) and nuts (mainly by walnut 25%, hazelnut 19%, almond 17% and pistachio 11%). Other fruits and nuts were involved in <10% of patients.

A reaction to a different kind of seed was reported in one patient (patient number 28) who suffered facial swelling after mustard seed ingestion before the sunflower seed allergy diagnosis.

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Skin Tests and specific IgE measurements

All patients had a positive result on sunflower seed SPT (median diameter 8 mm) or specific IgE (median IgE 1.1 kUA/L). Sunflower seed specific IgE values in patients with a history of atopy (rhinitis, asthma or atopic dermatitis) significantly exceeded those found in patients without atopic history (1.50, 0.56-2.88 kUA/L for atopic; 0.33, 0.11-0.50 kUA/L for non-atopic; p 0.017).

Patients clinically reactive to sunflower seed underwent skin tests and specific IgE with panallergens ns-LTP (rPru p 3, rAra h 9, rJug r 3, rCor a 8), profilin (rPru p 4) and 2S albumins (rAra h 2, rJug r 1) and other foods according to their history of reactions. There was no significant difference in sensitization to panallergens between different ages of onset of sunflower seed allergy (younger or older than 18 years). Sensitization to ns-LTPs was observed in 26 out of 35 (74%) patients who underwent ns-LTPs specific IgE (rPru p 3, rAra h 9, rJug r 3, rCor a 8) and Pru p 3 skin tests as shown in Table 3. Levels of sunflower seed IgE were strongly correlated with IgE levels to lipid transfer proteins (Spearman's Rank Correlation Coefficient 0.61-0.83; p <0.001). No significant correlation was found between the severity of reactions to sunflower seed and the grade of sensitization to sunflower seed or any food proteins.

Discussion

In the present study, we included 47 consecutive adult patients with sunflower seed allergy from a tertiary hospital in Spain to better characterize and improve our understanding of this allergy in adults.

Most patients had an adult-onset reaction to sunflower seed preceded by a history of atopy (including atopic dermatitis, allergic rhinitis and asthma) and other food allergies. An earlier onset of symptoms was found in patients who reported sunflower

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seed allergy as their first food reaction. The difference in the age of onset could be explained by the sensitization mechanism, leading most frequently in adults by cross-reacting allergens shared by foods and between inhalants and foods.[2, 24] However, in our sample, no significant differences were found in the atopic history, food allergies history or sensitization to panallergens between different groups of ages.

Almost half of the patients (49%) suffered severe or potentially life-threatening reactions after sunflower seed ingestion. Few patients underwent sunflower seed oral food challenges to confirm the diagnosis due to the severity of the reactions confirmed by specific sensitization and the patients' preference to avoid sunflower seed ingestion. Other case reports of anaphylactic reactions to sunflower seed have been previously published.[1, 8-21] Garcia Ortiz et al.[12] reported 15 reactions to sunflower seed ranging from OAS (oral allergy syndrome) to anaphylaxis in 11 patients from a series of 84 patients monosensitized to Artemisia pollen. Compared to our series, they found a lower proportion of severe reactions, with only 2 anaphylaxis after sunflower seed ingestion. Our results support that sunflower seed allergy should be considered as a potential cause of life-threatening reactions.

The high prevalence of systemic reactions in other common food triggers of severe reactions, like peanuts or nuts, is usually explained by sensitization to stable proteins, such as 2S albumins or ns-LTPs.[2] In our sample, no significant correlation was found between the severity of reactions to sunflower seed and the sensitization to other foods ns-LTPs or 2S albumins. Sunflower seed specific component-resolved diagnostics could help us predict the severity of specific sensitization patterns.

Among 16 patients with more than one reaction to sunflower seed before the diagnosis, we found that 10 patients (62%) with a history of OAS experienced other reactions to sunflower seed of higher severity. There are several reports of food induced-

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systemic reactions in patients with OAS. A nationwide study of pollen food allergy syndrome in Korea[25] reported a prevalence of anaphylaxis of 8.9% in patients with OAS. Skypala[26] associated the variability in clinical presentations of food allergy to atmospheric conditions, with increasing pollen counts and pollution leading to more severe reactions to foods in patients with OAS. Considering our results, strict avoidance of sunflower seed ingestion seems recommendable in sunflower seed allergic patients regardless the severity of the initial reaction.

Despite a high number of severe reactions in our series, those were often undertreated, with only 17% of these reactions treated with epinephrine. Our results are consistent with other studies routinely showing the underutilization of epinephrine, even though it is the first-line treatment recommended for anaphylaxis.[27-29] A national registry of Anaphylaxis in Portugal, including seeds as elicitors of 3% of the reactions, showed only 43% of patients admitted to the emergency department received epinephrine[28]. Another prospective study of anaphylaxis in an emergency care setting in Denmark estimated an administration of epinephrine in only 25% of the cases of anaphylaxis[29]. These findings emphasize the need for both patients and medical education concerning treatment of anaphylaxis.

Among 47 patients, only a patient reacted to other seeds, suffering facial swelling after mustard seed ingestion confirmed by specific sensitization. In vitro experiments have demonstrated serological cross-reactivity between sunflower seed and mustard 2S albumins[30] while there are few data in the literature concerning clinically relevant cross-sensitization among different seeds. According to our results, a diagnosis of seed allergy does not seem to predict other seed allergies.

Most patients with sunflower seed allergy had a history of other food allergies preceding the first reaction to sunflower seed, mostly to peach, peanut and nuts (walnut,

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hazelnut, almond and pistachio). The majority of patients were sensitized to ns-LTPs from peach, peanut or nuts, while few patients were sensitized to 2S albumins. Levels of sunflower seed IgE were strongly correlated with levels of IgE to those ns-LTPs. Nevertheless, no significant differences were found in the severity of the reactions according to sensitization to ns-LTPs. In the last decades, ns-LTPS have been identified as sunflower seed allergens, along with other proteins such as profilins or 2S albumins[31-32]. Considering the high prevalence of ns-LTPs sensitization in our area, it remains to be seen whether sensitization to those ns-LTPs develops independently or whether cosensitization occurs due to cross-reactivity with allergenic sunflower seed proteins. Data on the literature on cross-reactivity between sunflower seeds and other foods is scarce and is mostly based on nuts. Cross-reactivity between sunflower seed and pistachio has been demonstrated by inhibition studies[33]. Kort AA et al. [34] also showed a 34% identity between sunflower seed and Brazil nut 2S protein.

With sunflower seed as a potential severe allergen, further research is needed to investigate the allergenic content of sunflower seed, determine the clinical cross-reactivity among different seeds and other foods, and to develop component-resolved diagnostics for both diagnosis and prediction of severity associated with specific sensitization patterns.

In summary, most of the reactions to sunflower seed had an adult-onset and were preceded by a history of atopy and other food reactions, more frequently to peach, peanut and nuts. Sunflower seed reactions are often undertreated and its severity in the same patient could change on repeated exposures, ranging from OAS to anaphylaxis; which warrants the strict avoidance of this food in allergic patients and the epinephrine auto-inyector prescription when indicated.

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Authorship requirements

All authours made substantial contributions to the design of the work or the analysis of data for the work, revised intellectual content and approved the version to be published. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Conflict of interest

The authors declare no conflict of interest in relation to this work.

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FIGURES

Figure 1. Clinical classification of 47 patients labelled as sunflower seed-allergic.

[a]A positive double-blind placebo-controlled oral food challenge and a positive open food challenge with sunflower seed confirmed the diagnosis in 2 patients.

[b]OAS, Oral allergy syndrome.

[c]A positive double-blind placebo-controlled oral food challenge confirmed the diagnosis in 1 patient.

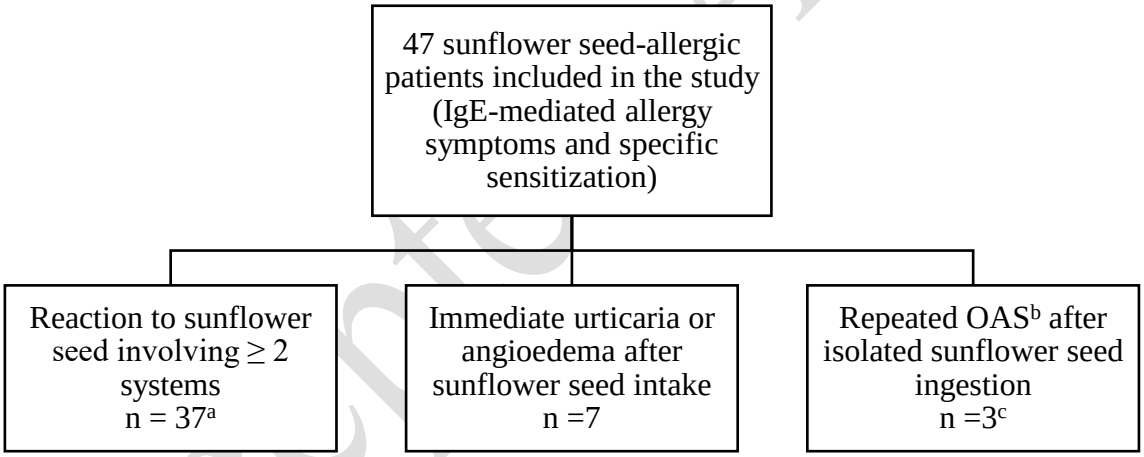

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Figure 2. Severity of symptoms according to the WAO Systemic Allergic Reaction Grading System in 47 patients with sunflower seed allergy.

[a]WAO Systemic Allergic Reaction Grading System: Grade 1: Symptom(s)/sign(s) from 1 organ or system (cutaneous, lips, upper respiratory, throat-clearing, cough not related to bronchospasm, conjunctival, nausea); Grade 2: Previous symptom(s)/sign(s) from ≥ 2 organ; Grade 3: Lower airways (mild bronchospasm) and/or gastrointestinal and/or uterine cramps; Grade 4: Lower airways (severe bronchospasm) and/or upper airway (laryngeal edema with stridor); Grade 5: Respiratory failure and/or collapse/hypotension and/or loss of consciousness.

In patients with more than one reaction to sunflower seed, the most severe reaction was considered.

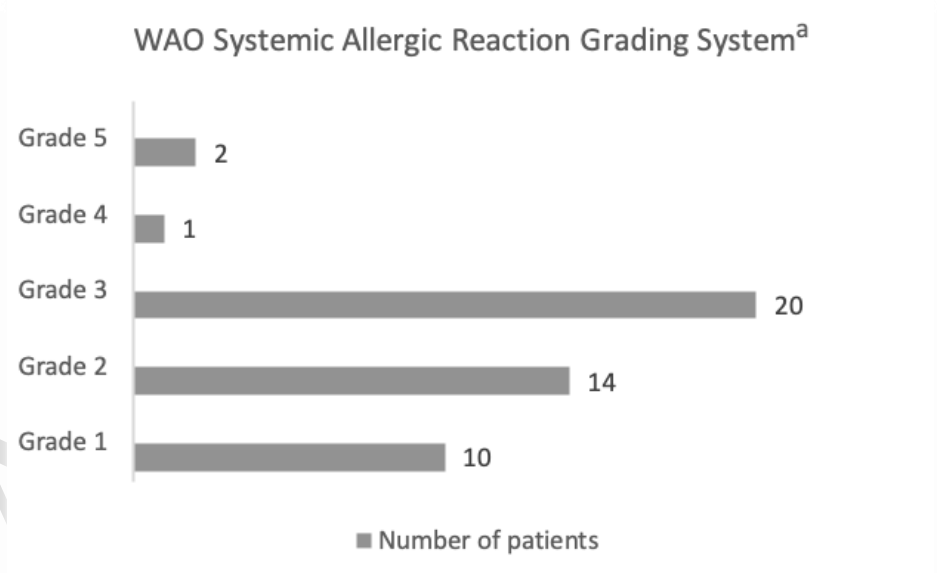

Table 1. Demographic features, symptoms after sunflower seed ingestion, skin tests and specific IgE, and other previous food allergies in 47 patients with sunflower seed allergy

N	Sex	Age	Symptoms (WAO)[a]	P-P to SS (mm)	Specific IgE to SS and food proteins (kUA/L)								Food reactions previous to SS	
					SS	Ara h2	Ara h9	Jug r1	Jug r3	Cor a8	Pru p3	Pru p4	Peanut/Nuts	Other foods
1	F	41	1		1,9						42,6			Pc, Pl, Av, K, Ba
2	M	40	3	+ 20	3,0		2,1							Cru
3	F	42	3	+ 5	40,3	0,1	88,4	0,0	53,6	16,4	57,3			K
4	M	26	3	+ 11	2,9	0,0	7,0	0,0	3,9	2,7	8,8		Al	K
5	F	57	3	+ 4	12,7	0,0	54,3		19,9	5,4	59,4	0,1	Pn	Pc
6	F	27	3	+	8,8	0,0	15,0	0,0	9,3	5,2	13,4	0,0		Pc
7	M	21	3	+ 22	0,4	0,0	0,4	0,0	0,6	0,3	0,7			Pc
8	M	45	3	+ 11	0,6									
9	F	26	3	+ 11	3,0		3,2			1,3	5,3	1,2	Pn, Hn, Cn, Al	
10	M	25	3	+ 5	0,7	0,0	1,8	0,0	1,2		2,2	0,0		
11	F	63	4	+ 11	2,8	0,0	7,8	0,0	6,9	4,5	11,8			
12	M	42	2	+ 8	1,2	0,0	1,2	0,0	1,4				Pn, Hn	
13	M	26	3	+ 11	1,7	0,0	4,6	0,0	3,6	3,3				
14	F	48	3	+ 9	0,7	0,0	0,0			0,0				
15	M	28	3	+ 4	9,4	0,1	28,7				26,9	2,3		
16	M	68	3	+ 9	0,2	0,0	0,0	0,0	0,0	0,0				
17	F	37	2	+ 5	0,8	0,0	1,8	0,0	1,6	1,2	2,1	0,0	Ps	Pc
18	M	40	1	+ 7	2,3			0,0	1,4	0,0				
19	M	42	2		1,2									
20	M	41	5		1,0								Pn, Wn, Hn	
21	M	24	3	+ 12	2,3						11,4	0,0	Wn, Pi	Pc, Apr, St, K, To
22	M	33	1	-	0,5						10,3	0,5	Pn	Pc
23	M	31	1	+ 9	3,1	0,0	4,4	0,0	4,9	2,1	9,2		Pn	Pc
24	F	21	2	+ 5	2,9		7,2	0,0	8,9	6,0	12,0		Pn, Wn	Pc
25	F	23	3		4,1	0,0	9,5	0,0	8,4	3,7	10,9	0,1	Pn, Hn, Wn	
26	M	20	2		2,1						5,2	0,0	Cn	Pc, To
27	M	41	2	+ 8	0,4					0,0			Ps	Ma, Cu
28	F	27	1	-	2,0	0,0	3,8	0,0	3,6	1,5				Pc, Mu-S
29	F	37	3	+ 15	24,3	0,0	0,0	0,0	0,0	0,0	0,0		Hn	
30	M	16	2	+ 7	2,2	0,1	0,1	0,2	0,1	0,1				

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31	F	49	2	+	5	1,0	0,0	2,3	0,8	0,7	0,2				
32	F	44	2	-		0,9	0,0	0,0	0,0	0,1	0,1	0,1		Pn, Ps, Hn, Al	
33	F	36	1			0,4	0,0	1,7	0,0	1,2	0,4			Wn, Al	
34	F	17	2	+	9	0,2								Pn, Wn, Hn, Al	To, Gb, Wb
35	M	42	1	-		8,2								Hn	App
36	F	30	2	+	6	1,1	0,0	0,0	0,0	0,0	0,0	0,0		Pn, Wn, Al, Hn, Ps, Pi, Cn	Pc, App, Pl, Ch, St, Av, On, Ga, Ba, Me, Wm, O, Le, To, Lt, Sp
37	M	24	1	+	10	1,5									
38	F	46	2	+	20	0,8								Pn, Al, Wn	Pc
39	F	51	3	+	6	0,5	0,0	4,4	1,4	2,0	0,8	3,4	0,8		
40	F	25	1	+	5	0,0			0,0	0,0		0,0			
41	F	65	3	+	8	0,0			0,0	1,1	0,4	1,3			Pc
42	F	45	3	+		0,1									
43	F	18	1	+	6	0,1	0,0	0,3	0,0	3,4	0,0	8,0	0,0	Pn, Wn, Al	Pc, App, Ch, Pr, Le
44	F	41	3	+	3	0,1		0,4	0,0	0,2		0,8		Wn	Pc, Me
45	F	27	2	+	5	0,1	0,0	0,0						Pn, Wn, Pi, Ps	Pl, Ch
46	M	32	5	+		0,1									
47	M	16	2	+	6	0,2	0,0	0,5	0,0	1,3		3,3	0,0	Wn	

[a]Symptoms were graded according to the WAO systemic allergic reaction grading system. In patients with multiple reactions to sunflower seed the most severe reaction was considered.

Abbreviations: SS, Sunflower seed; IgE, immunoglobulin E, P-P, prick-prick test; Pn, Peanut; Wn, Walnut; Al, Almond; Hn, Hazelnut; Ps, Pistachio; Pi, Pine nut; Cn, Chestnut; Mu-S, Mustard seed; Pc, Peach; Pl, Plum; Apr, Apricot; St, Strawberry; App, Apple; Pr, Pear; Ch, Cherry; Av, Avocado; K, Kiwi; Ba, Banana; Cru, Crustaceans; To, Tomato; Ma, Mango; Cu, Curry; Lt, Lettuce; Gb, Green bean; Wb, White bean; Me, Melon; Co, Coconut; On, Onion; Ga, Garlic; Wm, Watermelon; O, Orange; Le, Lemon; Sp, Spinach.

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Table 2. Variability of symptoms after sunflower seed exposure in 10 patients allergic to sunflower seed with multiple reactions.

Patient	Grade of symptoms	
n	Mildest reaction	Most severe reaction
4	OAS	OAS + urticaria + respiratory + gastrointestinal
6	OAS	OAS + hands swelling + respiratory
7	OAS	OAS + urticaria + gastrointestinal + nasal + conjuntival
10	Urticaria	Urticaria + facial swelling + respiratory
20	OAS + urticaria + swelling	OAS + urticaria + facial swelling + respiratory + collapse
21	Gastrointestinal	Gastrointestinal + urticaria
27	OAS	OAS + urticaria
29	OAS	OAS + respiratory
30	OAS	OAS + respiratory
38	OAS	OAS + foot and hands swelling

Abbreviations: OAS, Oral allergy syndrome.

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Table 3. Food proteins sensitization and correlation between sunflower seed IgE, food proteins specific IgE and severity of reactions to sunflower seed in 47 patients with sunflower seed allergy.

			Spearman's Rank Correlation Coefficient	
	N and % of patients sensitized	Median IgE (kUA/L)	Correlation between SS specific IgE and other proteins specific IgE	Correlation between severity of SS reactions (WAO) and specific IgE (SS and other proteins)
Sunflower seed	47	1,10		
LTPs				
Pru p 3 (peach)	22/26 (85%)	6,7	0.6095 (p <0.001)	0.1003 (NS)
Ara h 9 (peanut)	22/30 (73%)	1,9	0.8162 (p <0.001)	0.2702 (NS)
Jug r 3 (walnut)	21/28 (75%)	1,4	0.8319 (p <0.001)	0.2508 (NS)
Cor a 8 (hazelnut)	15/26 (58%)	0,6	0.7619 (p <0.001)	0.3255 (NS)
Any LTP (IgE/SPT)	26/35 (74%)			
Other proteins				
Pru p 4 (peach)	4/13 (31%)	0	0.2901 (NS)	0.2665 (NS)
Ara h 2 (peanut)	0/26 (0%)	0	0.5007 (p 0.009)	0.1207 (NS)
Jug r 1 (walnut)	2/27 (7%)	0	-0.1039 (NS)	0.1117 (NS)

Abbreviations: SS, sunflower seed; NS, non significant; LTP, lipid transfer proteins.

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