Allergy caused by ingestion of persimmon *(Diospyros kaki)*: Detection of specific IgE and cross-reactivity to profilin and carbohydrate determinants

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Background: Allergy to persimmon *(Diospyros kaki)* is very rare and not yet confirmed by means of double-blind, placebo-controlled, food-challenge (DBPCFC). Thus far, specific IgE to this fruit and cross-reactivity to pollen and other foods has not been determined.

Objective: The objective was to confirm allergy to persimmon in 3 patients with an according personal history and to characterize allergens and cross-reactivity of specific IgE antibodies to pollen and food allergens. One patient reacted with pruritus, penis edema, urticaria, and asthma; the second reacted with nausea and vomitus; and the third reacted with rhinoconjunctivitis, asthma, and stomachache after ingestion of persimmon.

Methods: Patients underwent skin prick testing with routine allergens, latex, persimmon, and other foods. Allergy to persimmon was confirmed by means of a DBPCFC. Specific serum IgE levels were measured with CAP-FEIA and the enzyme allergosorbent test (EAST) method. EAST and immunoblot inhibitions were carried out with persimmon; birch, grass, and ragweed pollen; latex; and N-glycans as inhibitors.

Results: All patients had positive skin test responses, DBPCFC and specific IgE assays to persimmon. Blot and EAST inhibition assays revealed IgE to cross-reactive profilin in one patient and IgE to cross-reacting carbohydrate determinants in all patients.

Conclusions: This is the first report on 3 cases of allergy to persimmon verified by means of DBPCFC and detection of specific IgE. The sensitization is due to cross-reactive profilin and carbohydrate determinants. (J Allergy Clin Immunol 2001;107:718-23.)

Key words: *Carbohydrate determinants; cross-reactivity; double-blind, placebo-controlled, food-challenge; allergy;* Diospyros kaki*; persimmon; profilin; IgE*

Abbreviations used

CCD: Cross-reacting carbohydrate determinant
DBPCFC: Double-blind, placebo-controlled, food-challenge
EAST: Enzyme allergosorbent test
PPT: Prick-to-prick test
SPT: Skin prick test

Inevitably, the increasing variety of foods being consumed and the increasingly atopic and allergic population is leading to previously unknown allergies to food. These usually occur as an immediate IgE-mediated reaction[1] either caused by cross-reactive allergies with a primary sensitization, for instance, to pollen[2] or latex[3] or as a primary sensitization to a defined food. Often, so-called panallergens, such as profilin[4,5] or carbohydrates,[6-8] are involved in cross-reactivity.

Persimmon *(Diospyros kaki)* is a sweet fruit growing in a moderate-to-warm climate and is commonly becoming a more popular food during its season in late autumn. We report on the first 3 cases of allergy to ingestion of persimmon, including double-blind, placebo-controlled, food challenge (DBPCFC); detection of specific IgE to persimmon; and identification of allergens that belong to known panallergen families.

METHODS

Skin testing

The patients underwent skin prick tests (SPTs) with routine allergens (*Dermatophagoides pteronyssinus*; *Dermatophagoides farinae*; goose feathers; dog, cat, and horse dander; *Alternaria* species; *Aspergillus* species; *Cladosporium* species; *Penicillium* species; *Candida albicans*; grass, mugwort, birch, alder, ash, hazel, and rye pollen; latex; and cow's milk [ALK, Hørsolm, Denmark]). Testing and interpretation were performed according to the position paper of the European Academy of Allergology and Clinical Immunology.[9] SPTs were also performed for pollen-related foods (Stallergènes, Cédex, France). Raw persimmon, apple, celery root, carrot, potato, and hazelnut were tested by means of prick-to-prick tests (PPTs) in all patients, and bell peppers, grapes, kiwi, and banana were tested in some of the patients. In addition, intracutaneous testing was performed for birch and mugwort pollen (Stallergènes) if they elicited negative test results in SPTs.

DBPCFC

DBPCFC was conducted with fresh persimmon, using passion fruit as placebo and to disguise the taste of persimmon. The per-

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simmon drink contained 100 g of persimmon, 50 g of yogurt, 5 g of honey, 40 g of passion fruit, 20 g of water, 15 g of lemon juice, 5 g of sugar, and 25 g of cream (total, 260 g); the placebo drink contained 50 g of yogurt, 10 g of honey, 90 g of passion fruit, 20 g of apple, 10 g of sugar, 10 g of raspberry syrup, 30 g of cream, and 2 g of Nesvital (total, 222 g). No intake of antihistamines, tranquilizers, or antidepressive agents was allowed 5 days before testing. Four control persons tested the drinks and could not distinguish between persimmon and placebo.

For DBPCFC, we used a spit-swallow protocol if necessary, an analogue to a method described elsewhere.[6,10] The drink remains in the oral cavity for 1 minute, and we wait for reactions during 15 minutes before continuing.

First, the patients were challenged with one teaspoon (5 g) of placebo and continued with the first drink (one teaspoon of persimmon [2.15 g] or placebo drink) if no reaction occurred. The dose was doubled with each challenge up to 8 teaspoons. The drinks were then switched, and the second drink was given in the same setting. If reactions were mild and not visible to the physician, we tried to reproduce them twice by switching drinks back and forth again, with 4 teaspoons (20 g) each. If no reaction occurred at all or no difference could be made between the drinks, we carried out the swallow protocol, only allowing one drink to be taken a day and starting with 1 tablespoon (10 g), doubling the dose every 15 minutes until the drink was finished. The second drink was consumed in the swallow protocol on another day.

A reaction was regarded as positive when the patient consistently reacted to the persimmon drink 3 times but not to the placebo. If reactions were observed clinically (erythema, asthma, urticaria, or edema) or were very bothering to the patient (nausea), we allowed the protocol to be shortened.

After the DBPCFC, SPTs with both drinks were performed to exclude a sensitization to components of the placebo drink.

Total and specific IgE

Diagnostic measurements of total and allergen-specific IgE were performed with the CAP-FEIA system (Pharmacia, Uppsala, Sweden). Specific IgE to persimmon was measured by using a commercial enzyme allergosorbent test (EAST) according to the instructions of the manufacturer (Allergopharma, Reinbek, Germany). For the EAST assay, antigens were coupled to cyanogen bromide–activated filter paper disks, as previously described.[11]

EAST inhibitions

For EAST inhibitions, sera were diluted (sera 152 and 291, 1:2; serum 907, 1:5) in incubation buffer (0.05 mol/L TRIS-hydroxymethyl-aminomethane, 0.15 mol/L NaCl, 1.0% Tween-20, and 0.3% BSA [pH 7.4]). For each disk, 50 μL of serum dilution was incubated with 50 μL of inhibitor solution (persimmon, birch pollen, grass pollen, ragweed pollen, latex, and skim milk extract: 500, 250, or 50 μg/mL; rBet v 1, rBet v 2, bromelain glycopeptide, fibrin glycopeptide: 100, 10, or 1 μg/mL diluted in incubation buffer).

Extracts, allergens, and N-glycans

Persimmon and skim milk extract were prepared from fresh material by a modification of a low-temperature extraction procedure, according to the methods of Vieths et al[11] and Rudeschko et al.[12] Fresh persimmon fruits were cut into short pieces and ground in a mortar under liquid nitrogen to a fine powder. This powder was then added to prechilled 4-hyxdroxy-4-methyl-2-pentanone (–20°C, 1:2 wt/vol) in several portions, and the obtained suspension was homogenized with a spinning blade homogenizer. Chilled acetone (–20°C) was added to a final fruit/solvent ratio of 1:5. After the suspension was stored overnight at –20°C, it was filtrated over a suction filter and washed twice with chilled acetone (–20°C) and once with chilled acetone-diethyl ether (1:1, –20°C). The filter residue was lyophilized. One gram of the obtained acetone powder was extracted for 40 minutes at 4°C with 20 mL of PBS (10 mmol/L potassium phosphate and 150 mmol/L NaCl [pH 7.4]). The suspension was centrifuged for 30 minutes at 20,000*g*, and the supernatant was filtered through a 1.2-μm membrane.

Ragweed pollen extract was obtained from MAST Diagnostica (Reinfeld, Germany), and latex and birch pollen extracts were obtained from Allergopharma. Recombinant Bet v 1 and rBet v 2 were purchased from Biomay (Linz, Austria), and grass pollen extract was a gift from Borstel Research Center (Dr A. Petersen, Borstel, Germany).

For the examination of cross-reactivity to carbohydrate determinants, we applied purified N-linked glycopeptides as inhibitors for EAST and blot inhibition. On the one hand, a glycopeptide with the glycan structure Manα1-6(Xylβ1-2)Manβ1-4GlcNAcβ1-4(Fucα1-3)GlcNAc and just 2 to 4 amino acid residues were used. It contains structures that are involved in IgE binding to cross-reacting carbohydrate determinants (CCDs), α1,3-linked fucose, and β1,2-linked xylose,[13] and it was prepared from bromelain, as previously described.[8,14] As a negative control, a glycopeptide with the common pentasaccharide core Manα1-6(Manα1-3)Manβ1-4GlcNAcβ1-4GlcNAc was used. It was prepared from bovine fibrin, as previously described.[13] The purity of the applied N-glycans was checked by means of mass spectrometry and SDS-PAGE (not shown).

Immunoblotting and immunoblot-inhibition experiments

Persimmon extract was separated by means of SDS-PAGE (T = 12.5%, C = 2.7%) under reducing conditions, applying 25 μg of protein per centimeter on 5.5-cm preparative slots. The separated proteins were transferred onto nitrocellulose membranes by means of semidry blotting.[16] The membrane was blocked with 0.05 mol/L TRIS-HCl, 0.15 mol/L NaCl, and 0.3% Tween-20 (pH 7.4). Cut nitrocellulose strips (0.3 cm) were incubated overnight in 1.0 mL of diluted patient serum (1:6.7). Sera were diluted in incubation buffer (0.05 mol/L TRIS-hydroxymethyl-aminomethane, 0.15 mol/L NaCl, 0.1% BSA, and 0.05% Tween-20 [vol/vol; pH 7.4]). Bound IgE were detected with mouse monoclonal anti-human IgE alkaline phosphatase conjugate from PharMingen (San Diego, Calif; 1:750, 4 hours, 1 mL) and subsequent staining with 4-nitroblue tetrazolium chloride and 5-bromo-4-chloro-3-indolylphosphate, which was purchased from BioRad (Hercules, Calif), as the substrate.

For blot inhibitions, sera were diluted 1:6.7 in incubation buffer and preincubated with 100 μg of protein from plant extracts or skim milk or 10 μg of N-glycans. Incubations of the blot strips and detection were performed as above.

RESULTS

Personal history, skin test results, and IgE determinations are summarized in Tables I to III. All patients had positive PPT responses to persimmon (Table I). Also, increased specific IgE levels to persimmon were determined in all patients (Table III).

Case 1 (patient 291)

A 26-year-old man had hay fever and asthma since early youth, and during recent years, he increasingly experienced allergic reactions to ingestion of food. After eating raw persimmon, he felt pruritus in both his hands and inguinae. In addition, he had urticaria, penis edema,

TABLE I. Personal history: Atopy, food allergy, and skin tests (only positive test results)

Patient No.	Age (y)	Sex	Rhino-conjunctivitis	Asthma	Food allergy	Positive skin test results
291	26	M	April-July for >15 y	Animal hair	OAS: ap, pe, nuts; urticaria and asthma: pe, zu, wm, cu to recent years	SPT: bi (+++), gr (++), rp (+), al (+++), as (+++), hp (+++), Dpt (+++), Dfa (+++), la (+), cd (+), ha (+), to (+); PPT: pe (+++), ap (+++), ce (+++), ca (+++), po (+++), persimmon drink (+), placebo drink (–); IC: mu (+)
152	17	F	March-September for 10 y	None	OAS: ca, ha; rhinoconjunctivitis: ki; nausea and vomitus: pe in recent years	SPT: bi (++), rp (++), al (++), as (+), hp (++), cd (++); PPT: pe (++), ce (+++), ca (+++), ap (+), ha (+), bp (+), persimmon drink (+), placebo drink (–)
907	24	M	April in recent years	Persimmon	OAS: ba, da, ol, ki, gp; rhinoconjunctivitis, nausea, and vomitus: pe in recent years	SPT: negative; PPT: pe (+++), ca (+), ba (+++), da (+), gp (+), ki (+), persimmon drink (++), placebo drink (–); IC: bi (+), mu (++)

OAS, Oral allergy syndrome; *IC,* intracutaneous testing; *al,* alder pollen; *as,* ash pollen; *ba,* banana; *bi,* birch; *bp,* bell peppers; *ca,* carrot; *cd,* cat dander; *ce,* celery; *cu,* cucumber; *da,* dates; *Dfa, D farinae*; *Dpt, D pteronyssinus*; *gp,* grapes; *gr,* grass pollen; *ha,* hazelnut; *ki,* kiwi; *la,* latex; *mu,* mugwort pollen; *ol,* olives; *pe,* persimmon; *po,* raw potato; *rp,* rye pollen; *to,* tomato; *wm,* watermelon; *zu,* zucchini.

TABLE II. Personal history: Symptoms to ingestion of persimmon and DBPCFC

Patient No.	Age (y)	Sex	Symptoms to ingestion	Symptoms to DBPCFC
291	26	M	Palmaplantar pruritus, urticaria, asthma	Nausea, pruritus, rhinorrhea, OAS
152	17	F	Nausea, vomitus	Nausea, OAS, rhinorrhea
907	24	M	Rhinorrhea, stomachache, asthma, vomitus, tachycardia	OAS, erythema of the lips

OAS, Oral allergy syndrome.

TABLE III. Results of total and specific IgE determinations

Patient No.	Total IgE (kU/L)	Sx1 (kU/L)	EAST,* persimmon (kU/L)	CAP* (kU/L)						
				Melon	Ragweed	Kiwi	Latex	Birch	rBet v1	rBet v2
291	207	80.5 (5)	2.08 (2)	1.03 (2)	ND	ND	0.85 (2)	32.2 (4)	31.9 (4)	8.1 (3)
152	483	ND	2.05 (2)	ND	ND	2.47 (2)	9.13 (2)	>100 (6)	2.47 (2)	<0.35
907	253	5.2 (3)	7.22 (3)	4.3 (3)	5.3 (3)	3.3 (2)	9.2 (3)	2.3 (2)	0.4 (1)	<0.35

Sx1, IgE screening test for common inhalational allergens; *ND,* not determined.
*EAST and CAP classes are shown in parentheses.

and bronchial asthma. Similar symptoms occurred after ingestion of cucumber, watermelon, and tomato.

The DBPCFC result was positive for persimmon. The patient reacted with nausea after 8.6 g and 17.2 g in the spit protocol, a burning of the oral cavity, and rhinorrhea after 8.6 g of persimmon after switching drinks back and forth. No reaction occurred with placebo in the spit protocol. Prick test responses were positive for the persimmon drink and negative for placebo.

Case 2 (patient 152)

A 17-year-old girl had hay fever from March to September since early youth, and during recent years, increasingly experienced allergic reactions to ingestion of food. After eating raw persimmon, she felt dizzy and sick and had to vomit. Symptoms also occurred after ingestion of raw carrots, hazelnuts (oral allergy syndrome), and kiwi (rhinoconjunctivitis).

The DBPCFC result was positive for persimmon. The patient reacted with slight itching of the throat after 1.8 g and 8.2 g in the spit protocol and nausea, itching of the throat, sneezing, and rhinorrhea after 55 minutes and a total of 100 g in the swallow protocol. No reaction occurred after placebo in the spit-swallow protocol. Prick test responses were positive for the persimmon drink and negative for placebo.

Case 3 (patient 907)

A 24-year-old man had hay fever during spring for several years and increasingly experienced allergic reactions to ingestion of food. After eating raw persimmon, he experienced rhinorrhea, stomachache, and conjunctivitis after 10 minutes and asthma, accelerated heartbeat, and the need to vomit after 15 minutes. He previously had an oral allergy syndrome to dates, green olives, and kiwi.

The DBPCFC result was positive for persimmon. The

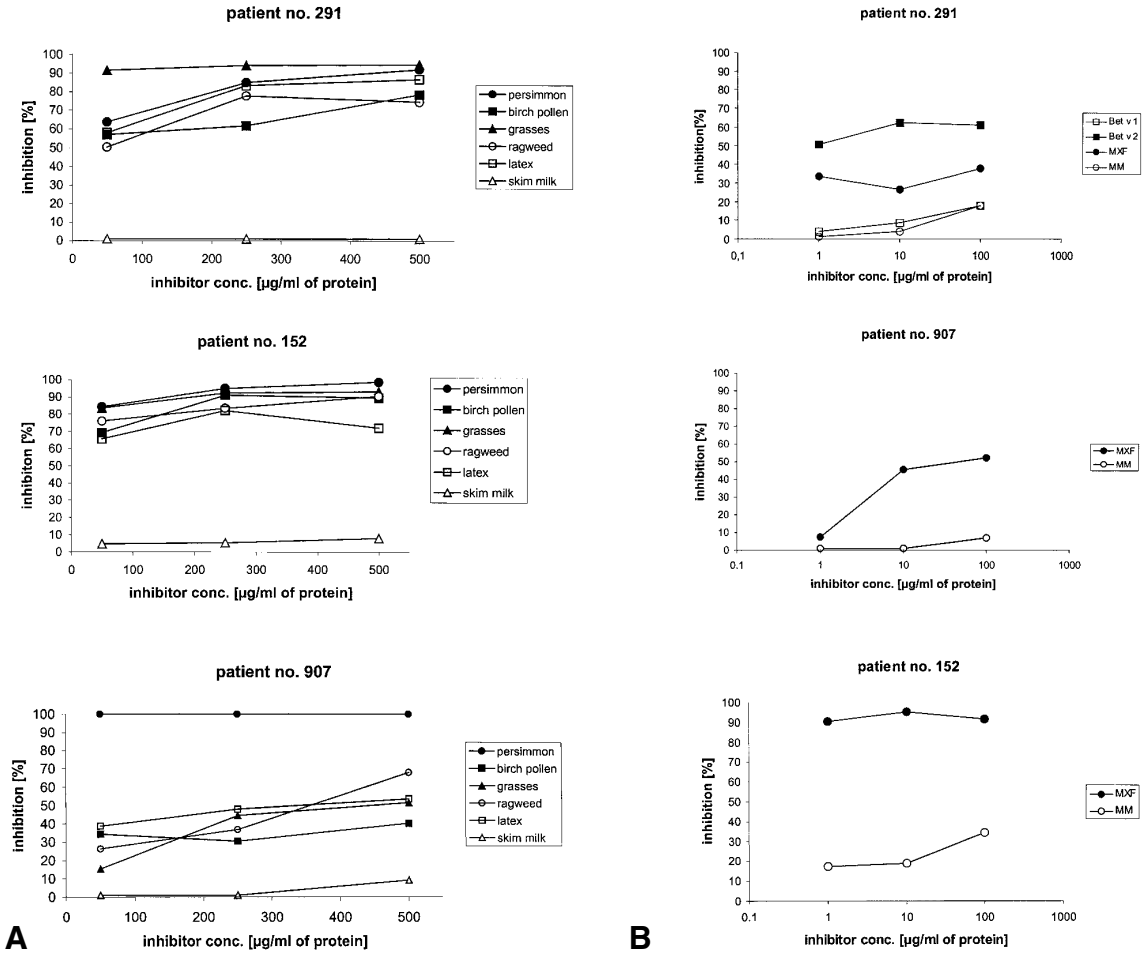

FIG 1. A, Dose-related EAST inhibition with plant extracts. **B,** Dose-related EAST inhibition with recombinant allergens and glycopeptides.

patient reacted with an oral allergy syndrome (itching and burning of the oral cavity and slight erythema on the margin of the lips) after 1.8 g of persimmon in the spit protocol and 8.2 g twice after switching drinks back and forth. No reaction occurred after placebo in the spit protocol. Prick test responses were strongly positive for the persimmon drink and negative for placebo.

Inhibition assays

The results of the dose-dependent EAST inhibition are shown in Fig 1, *A* and *B*. The specificity of the assay was demonstrated by the very strong inhibition obtained with persimmon extract as the inhibitor in all sera, whereas the applied negative control (skim milk) induced no inhibition (Fig 1, *A*). To the other plant extracts used as inhibitors (birch, grass, ragweed, and latex), patient sera 152 and 291 showed a very similar inhibition pattern. IgE antibodies from both patients were strongly inhibited by these extracts. A distinct but weaker inhibition for the plant extracts other than persimmon was obtained with serum 907 (Fig 1, *A*).The high cross-reactivity to all plant extracts points to the existence of CCDs in the persimmon extract. This was verified by the results of the inhibition with N-glycans. With all sera, an inhibition with the bromelain glycopeptide was measurable, whereas the inhibitory effect of the control glycopeptide was weak (Fig 1, *B*). In serum 152 the highest level of IgE binding to CCDs (90%) could be detected, and the maximal inhibition for sera 291 and 907 was about 40% and 50%, respectively. In the EAST inhibition experiment of patient serum 291, the only serum with a positive CAP result to rBet v 2, the panallergens rBet v 2 and rBet v 1 (negative control) were used as additional inhibitors. Only rBet v 2 produced a maximal inhibition of approximately 60%. Together with the maximum inhibition of the bromelain glycopeptide, a sum of about 100% was obtained with this serum.

Cross-reactivity of persimmon allergens was further investigated by means of blot inhibition. The immunoblot-inhibition results with both reactive sera 152 and 907, which presented IgE binding on immunoblots, are shown in Fig 2. With both sera, multiple IgE-reactive bands above 25 kd were recognized. The almost total inhibition with persimmon extract (lane 2)

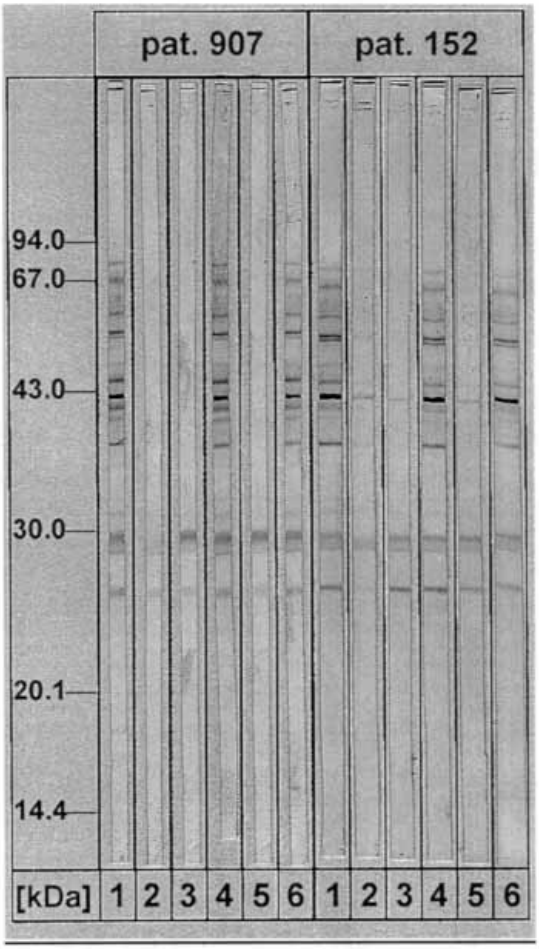

FIG 2. Blot inhibition experiments with sera from patients 907 and 152. *Lane 1,* No inhibitor; *lane 2,* 100 μg of protein from persimmon extract; *lane 3,* 10 μg of bromelain glycopeptide; *lane 4,* 10 μg of fibrin glycopeptide; *lane 5,* 100 μg of protein from birch extract; *lane 6,* 100 μg of protein from skim milk extract.

shows the high specificity of the experiment. Only the band at about 25 kd seems to be nonspecific because it was not fully inhibited by persimmon extract in patient 907. The blot inhibition with the N-glycans corresponded strongly to those obtained in the EAST experiment. All bands above 30 kd disappeared with the bromelain glycopeptide as the inhibitor (lane 3). This effect was not found with the control peptide (lane 4). With both sera, a band at approximately 30 kd could be detected. It was inhibited with persimmon extract but not with the N-glycans, birch, and skim milk extracts.

DISCUSSION

This is the first report on allergy to ingestion of persimmon in 3 patients, including detection of specific IgE and characterization of allergens and cross-reactivity. A report on one case of anaphylactic reaction to persimmon has been previously published; however, in this case specific IgE to persimmon could not be found, and the relevant food leading to this reaction was not clear because no DBPCFC was performed.[17]

In all 3 cases of our study, allergy to persimmon could be demonstrated by positive PPT and positive DBPCFC responses, which are required to verify the relevance of the sensitization.[18] Increased specific IgE levels to persimmon were detected in all 3 patients. In the DBPCFC, symptoms were generated in the spit protocol as an oral allergy syndrome in 2 patients. One patient who underwent the swallow protocol later had rhinitis and nausea. Symptoms were similar to reactions described after eating persimmon in the personal history. The patients were all atopic, one with sensitization to perennial and seasonal allergens and food and the others with sensitization only to seasonal allergens and food.

In all patient sera high levels of IgE to cross-reactive carbohydrate determinants were measured by means of EAST inhibition. IgE binding to persimmon proteins was partly inhibited by the bromelain glycopeptide and all plant extracts. The main IgE-binding structures in CCDs of bromelain glycopeptide are α1,3-linked fucose and β1,2-linked xylose. The possibility of reactions occurring to protein contaminations of the glycopeptide was excluded by means of mass spectrometry and gel electrophoresis analyses of the carbohydrate moiety isolated from bromelain. The results of the EAST inhibition were additionally confirmed by blot inhibitions with the 2 reactive sera in this assay. The detection of persimmon allergens ranging from 30 to 70 kd by the patients' IgE was inhibited by persimmon, birch pollen extract, and the bromelain glycopeptide.

In the patient who did not react in the immunoblot experiment, a cross-reactivity to profilin was found with the EAST assay. With recombinant Bet v 2, an inhibition of about 60% was obtained. The positive CAP result to rBet v 2 verified the sensitization of this patient to birch profilin. A conformational epitope on profilin may explain the negative results of immunoblotting after electrophoresis under reducing and denaturing conditions.

In addition to the cross-reactive structures probably involved in persimmon allergy, at least for one patient (907), there might be a pollen-independent persimmon allergen. In the EAST inhibition only persimmon extract totally inhibited the IgE binding, whereas the N-glycans and all other extracts used only led to a partial inhibition. A possible explanation could be the 30-kd allergen, which is detected in immunoblot inhibition for 2 patients and could only be inhibited with persimmon extract.

In addition to this finding, however, the fact that persimmon is yet another food with cross-reacting carbohydrate determinants, as well as profilin, leads us to the assumption that these (atopic) individuals were primarily sensitized to pollen. These cross-reactive allergens are important panallergenic structures that are also involved in allergy to apple, peach, zucchini, celery, lychee, and jackfruit.[5,6,8,19,20]

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