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Component-resolved diagnosis of peach allergy and its relationship with prevalent allergenic pollens in China

To the Editor:

Today, molecule-based component-resolved diagnosis is more commonly used because of its advantage in distinguishing species-specific allergens from cross-reactive allergens and predicting the potential risk of severe reactions. Sensitization patterns of foods comprising the component allergens and relevant manifestations exhibit obvious geographic differences.[1,2] Peach allergy is common in China; however, the sensitization to peach allergens and the relevance of the sensitization pattern for the clinical presentation remain unclear.

The most important peach allergens, Pru p 1, Pru p 3, and Pru p 4, belong to the protein family of pathogenesis-related protein 10 (PR-10), nonspecific lipid transfer protein (nsLTP), and profilin, respectively. It is well known that PR-10–related oral allergy syndrome usually follows birch pollen sensitization.[2] The nsLTPs used to be considered pollen-unrelated "true" food allergens. However, *Platanus acerifolia*, *Olea europaea*, and mugwort pollen nsLTPs were successively reported to have a close relationship with peach nsLTP, namely Pru p 3,[3-5] despite the controversy on the initiation pathway of primary sensitization.[3,6,7] Mugwort is the most important allergenic pollen allergen in late summer and autumn in China, especially in the northern region, where pollinosis is a major health problem.[8] A very recent study from China concluded that Art v 3 played a dominant role in peach allergy as a primary sensitizer.[9]

Here we present sensitization patterns of peach and their clinical significance in China and further analyze the relationship between pollen sensitization and peach allergy. Eighty-seven patients with positive peach-specific IgE levels were included in this study. Thirty-eight were symptomatic on peach exposure (19 only presented with oral allergy syndrome, which consists of isolated oropharyngeal symptoms, and 19 had ≥1 after manifestations: generalized urticaria, allergic rhinitis, asthma, gastrointestinal symptoms, or even hypotension with or without oral reactions). Forty-nine patients were sensitized but tolerant to peach (peach-tolerant group). Total IgE and specific IgE against peach, rPru p 1 and rPru p 3, rBet v 1, rBet v 2, mugwort, and nArt v 3 were detected by using the ImmunoCAP system (Thermo Fisher, Uppsala, Sweden). Because Pru p 4 reactivity was proved to be completely correlated with Bet v 2, we tested rBet v 2 (birch profilin) instead of rPru p 4. This research protocol was approved by the local ethics committee, and written consent was obtained from each patient.

All patients were from northern China, where mugwort and birch are 2 of the most common allergenic pollens that lead to seasonal rhinitis, asthma, or both in late summer and autumn, as well as spring, respectively.[8] There were no differences in sex, age, family history of allergic disease, personal history of eczema or drug allergy, and total IgE levels between symptomatic and asymptomatic patients (Table I). Peach-specific IgE levels of symptomatic patients were markedly higher than those of asymptomatic patients (median, 6.11 vs 2.68 kU/L; $P = .023$; Table I).

The total positivity rate of IgE response to rPru p 1, rPru p 3, and rBet v 2 was 47.4%, 55.3%, and 34.2% in the symptomatic group, respectively (Table I), and therefore rPru p 3 was the major allergen of peach in our population. The 3 most common sensitization patterns of peach were monosensitization to rPru p 1, cosensitization to rPru p 3 and rBet v 2, and monosensitization to rPru p 3, which involved 81.6% of all patients with peach allergy (Table I).

Although no differences were found in the prevalence of birch pollinosis between symptomatic and asymptomatic patients, birch pollen sensitization, as represented by positivity for serum IgE (using 0.35 kU/L as a cutoff), was more common in the symptomatic group than in the asymptomatic group (84.2% vs 65.3%, respectively; $P = .047$), as was specific IgE against rBet v 1 (Table I). Moreover, rPru p 1 IgE levels were significantly related to rBet v 1 levels (Spearman $\rho = 0.934$; $P < .001$). This might be a reasonable explanation for the higher positivity rate of IgE response to rPru p 1 in the symptomatic group (Table I). Therefore rPru p 1 was a clinically relevant allergen of peach in our population. Furthermore, rPru p 1 sensitization was more frequently observed in the subgroup with oral allergy syndrome, which was most likely due to the higher prevalence of birch pollinosis and rBet v 1 sensitization in the oral allergy syndrome subgroup (Table I).

The prevalence of mugwort pollinosis and pollen sensitization did not differ between symptomatic and asymptomatic patients, but mugwort pollen sensitization did affect the types of symptoms of peach allergy (Table I). Mugwort pollen and nArt v 3 sensitization was more frequently seen in patients with systemic symptoms, which was in accordance with the higher frequency of rPru p 3 sensitization in the SS group. Art v 3 IgE levels were also in close relationship with rPru p 3 IgE levels (Spearman $\rho = 0.718$, $P < .001$).

Comparisons of rPru p 3 and nArt v 3 IgE levels were conducted between rPru p 3–sensitized patients with and without

TABLE I. Clinical characteristics and sensitization profiles of patients included in this study

Characteristics	Symptomatic patients (n = 38)	Asymptomatic patients (n = 49)	*P* value
Male sex, no. (%)	15 (39.5)	23 (46.9)	.517
Age, median (range)	24.5 (6-49)	26 (2-49)	.264
Positive family history of atopy, no. (%)	16 (42.1)	21 (42.9)	1.000
Total IgE (kU_A/L), median (range)	219.5 (24.4-5000)	273 (16.6-2643)	.691
Peach-specific IgE (kU_A/L), median (range)	6.11 (0.39-64.1)	2.68 (0.32-18.5)	.023
Other known food allergies, no. (%)	38 (100.0)	31 (63.3)	<.001
Atopic eczema	3 (7.9)	5 (10.2)	1.000
Drug allergy, no. (%)	14 (36.8)	16 (32.7)	.821
Pollens, no. (%)			
Birch symptoms	17 (44.7)	13 (26.5)	.076
ImmunoCAP	32 (84.2)	32 (65.3)	.047
rBet v 1	19 (50.0)	12 (24.5)	<.001
Mugwort symptoms	18 (47.4)	29 (59.2)	.270
ImmunoCAP	26 (68.4)	41 (83.7)	.094
nArt v 3	17 (44.7)	25 (51.0)	.233
Other symptoms	8 (21.1)	13 (26.5)	.554
ImmunoCAP	6 (15.8)	8 (16.3)	1.000
Sensitization profile of peach, no. (%)			
rPru p 1	14 (36.8)	5 (10.2)	.003
rPru p 1 + rBet v 2	0	1 (2.0)	ND
rPru p 1 + rPru p 3	3 (7.9)	4 (8.2)	1.000
rPru p 1 + rPru p 3 + rBet v 2	1 (2.6)	0 ND	
rPru p 3	8 (21.1)	14 (28.6)	.424
rPru p 3 + rBet v 2	9 (23.7)	6 (12.2)	.161
rBet v 2	3 (7.9)	11 (22.4)	.067
Other	0	8 (16.3)	ND
Total rPru p 1	18 (47.4)	10 (20.4)	.008
Total rPru p 3	21 (55.3)	24 (49.0)	.561
Total rBet v 2	13 (34.2)	18 (36.7)	.807
	SS (n = 19)	**OAS (n = 19)**	
Pollens, no. (%)			
Birch symptoms	5 (26.3)	12 (63.2)	.022
ImmunoCAP	17 (89.5)	15 (78.9)	.374
rBet v 1	6 (31.6)	13 (68.4)	.023
rBet v 2	11 (57.9)	2 (10.5)	.002
Mugwort symptoms	12 (63.2)	6 (31.6)	.051
ImmunoCAP	16 (84.2)	10 (52.6)	.036
nArt v 3	13 (68.4)	4 (21.1)	.003
Sensitization patterns of peach, no. (%)			
rPru p 1	3 (15.8)	11 (57.9)	.007
rPru p 1 + rPru p 3	1 (5.2)	2 (10.5)	ND
rPru p 1 + rPru p 3 + rBet v 2	0	1 (5.2)	ND
rPru p 3	4 (21.1)	4 (21.1)	1.000
rPru p 3 + rBet v 2	9 (47.4)	0	ND
rBet v 2	2 (10.5)	1 (5.2)	ND
Total rPru p 1	4 (21.1)	14 (73.7)	.005
Total rPru p 3	14 (73.7)	7 (36.8)	.022
Total rBet v 2	11 (57.9)	2 (10.5)	.002

Comparison of categorical variables between groups was conducted by using the Pearson χ^2 or Fisher exact tests. Cutoff values for positive ImmunoCAP results were 0.35 kU/L for specific IgE antibodies against extracts of pollens and peach and 0.30 kU/L for specific IgE antibodies against allergenic components of pollens and peach.
ND, Not determined because of a small number of patients; *OAS*, oral allergy syndrome; *SS*, systemic symptoms.

relevant clinical symptoms to further analyze the relationship of mugwort pollinosis with peach allergy.

A total of 45 patients were sensitized to rPru p 3 in this study, but only 21 patients presented with relevant manifestations (rPru p 3 allergy), among whom 13 patients also had mugwort pollinosis and the remaining 8 patients only had peach allergy. In addition, 15 of the 24 rPru p 3–sensitized but tolerant patients (rPru p 3 tolerance) had mugwort pollinosis, and the remaining 9 patients were free of both peach allergy and mugwort pollinosis. Mugwort pollinosis was defined as seasonal rhinitis, asthma, or both occurring only in late summer and autumn, together with a confirmed positive serum IgE response to mugwort pollen extract. No difference was found in rPru p 3 IgE levels between the patients with rPru p 3 allergy and the tolerant patients (median, 2.32 vs 2.43 kU/L; $P = .724$). Art v 3 levels were remarkably higher than rPru p 3 levels in patients with mugwort pollinosis, whereas rPru p 3 IgE levels were comparable with Art v 3 IgE levels in patients without mugwort pollinosis (Fig 1). No