
Clinical characteristics of melon (*Cucumis melo*) allergy

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Background: Although melon is a frequent allergy-eliciting fruit, allergic reactions to melon have rarely been reported.

Objective: To evaluate and describe the clinical characteristics of melon allergy in melon-allergic patients.

Materials and Methods: We evaluated patients allergic to melon and a control group of patients allergic to pollen. The diagnosis of melon allergy was based on a convincing clinical history, positive skin test results (prick-by-prick test), and positive results on oral challenge tests to melon.

Results: A total of 161 patients were included in the study: 66 in the melon allergy group and 95 in the pollen control group. The melon allergy group included 35 female and 31 male patients with a mean age of 26.6 ± 2.7 years (range, 5–61 years). Although all patients had oral symptoms, 13 (19.7%) of the patients had extraoral symptoms and none experienced generalized urticaria or anaphylaxis. Excluding other Cucurbitaceae fruits, peach, fig, and kiwi most frequently elicited positive skin test results and symptoms. Up to 23% of melon-allergic patients had a concomitant latex sensitization. Melon allergy was especially linked to pollen allergy, since all the melon-allergic patients were also allergic to pollen. Some differential features with respect to the pollen allergy control group were a higher prevalence of asthma (odds ratio [OR], 2.13; $P < 0.05$) and a statistical increase in the frequency of sensitization to several tree and weed pollens, including *Ulmus* (OR, 42.8) and *Ambrosia* (OR, 22.4).

Conclusion: The most important conditions linked to melon allergy are pollen allergy (100%), allergy to other nonrelated fruits, mainly peach (up to 62%), and latex sensitivity (up to 23%). Some differential features of the pollinosis in melon allergy were a higher prevalence of asthma and a higher frequency of sensitization to several weed and tree pollens.

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INTRODUCTION

Melon (*Cucumis melo*) is a fruit belonging to the Cucurbitaceae family. Evidence suggests that melons were first cultivated in Egypt as early as 2400 BC. Melon spread slowly to Europe from Africa and Asia, becoming popular in Spain in the 15th century. It was soon introduced to America from Spain. Melon is mainly produced in semiarid regions, and thus California accounts for 70% of the total production in the United States. Today, melon is a fruit known throughout the world and largely consumed in a variety of ways: fresh as a fruit dessert or in salads, cooked (soup, stew, curry, stir-fry), pickled, canned, or used for syrup or jam.

Although allergy to fruits is a frequent cause of food allergy,^{1–8} little attention has been paid to this type of allergy. Recently, a large number of reports concerning fruit allergy focused mainly on allergy to Rosaceae fruits. In this sense, some interesting data have been reported, such as the description of a panallergen, Pru p 3 or lipid transfer protein, as the major peach allergen^{9,10} or the finding that up to 20% of peach allergy is not linked to pollen allergy.^{11,12}

Melon has been reported to be a frequent allergy-eliciting fruit in some areas in the United States¹³ and the second most frequent allergy-eliciting fruit in Spain,⁸ where fruit allergy is the most important cause of food allergy in adult patients.^{1,14} Despite these data, allergic reactions to melon have rarely been reported. The aim of this study is to evaluate and describe the clinical characteristics of melon allergy in a population of melon-allergic patients.

MATERIALS AND METHODS

Study Population

We selected melon-allergic patients to evaluate the characteristics of melon allergy and a control group of patients allergic to pollen to compare the pollen allergy between the 2 groups. Melon-allergic patients in both groups were diagnosed by criteria of symptoms of previous melon allergy reactions, positive skin prick-prick test (SPPT) results, and positive oral challenge test results to melon. The patients gave their written consent to participate in the study.

Questionnaire

A detailed clinical history was obtained for each patient, and patients filled out a questionnaire to permit statistical management of the data. The following parameters were established: age, sex, age at the first episode of allergy to melon, years from the first episode, number of episodes, evolution time, type of symptoms, and symptoms to latex and other

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foods, especially vegetables, fruits, nuts, and mustard. We also inquired about pollen allergy, such as allergic respiratory symptoms (rhinitis or asthma), year of the first episode, age at the first episode, and evolution period.

Skin Test

We performed SPPTs to fresh melon pulp in duplicate, following the technique described by Dreborg and Foucard.¹⁵ The Alk-Lancet needle (Alk-Lancet; Alk-Abelló, Hørsholm, Denmark) was used for skin tests, which were performed according to European Academy of Allergology and Clinical Immunology guidelines.¹⁶ A result was considered positive when a wheal of 3 mm was obtained.

Skin prick tests to commercially available melon extracts (Leti SA, Madrid, Spain, Bial-Aristegui, Bilbao, Spain, and Stallergenes SA, Paris, France) were performed to evaluate the availability of commercial melon extracts to detect patients sensitized to melon. We also performed SPPTs to other fresh fruits (*Malus domestica* [apple], *Musa acuminata* [banana], *Ficus carica* [fig], *Vitis vinifera* [grape], *Actinidia deliciosa* [kiwi], *Citrus sinensis* [orange], *Pyrus communis* [pear], *Ananas comosus* [pineapple], and *Prunus persica* [peach]) and skin prick tests to other commercially available allergens (*Prunus dulcis* [almond], *Corylus avellana* [hazelnut], *Arachis hypogaea* [peanut], *Juglans regia* [walnut], *Castanea sativa* [chestnut], *Helianthus annuus* [sunflower seeds]), latex, and *Brassica sinapsis* [mustard]).

Skin prick tests to an aeroallergen commercial battery (pollens, mites, fungi, cat, and dog) were performed, including the following pollens: 5 grasses (*Lolium*, *Phleum* [timothy grass], *Phragmites*, *Secale* [rye grass], and *Cynodon* [Bermuda grass]); 6 weeds (*Ambrosia* [ragweed], *Artemisia*, *Chenopodium*, *Parietaria*, *Plantago* [English plantain], and *Taraxacum*); and 5 trees (*Betula* [birch], *Fraxinus* [ash], *Olea*, *Populus*, and *Ulmus* [elm]).

Melon Challenge Test

The oral challenge tests were performed with fresh melon pulp, following the open method previously described.⁸ Briefly, each dose was chewed and then swallowed. The melon doses were gradually increased until a positive response was obtained or until the entire melon portion was eaten without symptoms. The in vivo tests with fresh melon were performed from March to December, which is the melon season in Spain. Oral challenge test was performed in all patients from the melon allergy group. Oral challenge test in the control group was performed in patients who reported symptoms (10 patients) or those who had a positive SPPT result to fresh melon pulp (13 patients).

Statistical Methods

Quantitative variables were reported as medium, SD, and range. Qualitative variables were described by absolute (n) and relative (percentage) frequencies. To evaluate the association between 2 qualitative variables, individual 2 × 2 contingency tables were constructed. The magnitude of the association was estimated by the odds ratio (OR) and 95%

confidence intervals (95% CIs). The statistical significance was evaluated by the χ^2 test. All *P* values were 2-tailed.

RESULTS

A total of 161 patients were included in the study: 66 patients in the melon allergy group and 95 pollen-allergic patients as a control group. The characteristics of patients belonging to the control group have previously been reported.⁸ Up to 13.7% of pollen-allergic patients had a positive SPPT result to fresh melon pulp; however, only 7.4% of the patients in the pollen allergy group had melon allergy based on positive results on the skin test and oral challenge test.

Age and Sex

The melon allergy group was composed of 35 female (53%) and 31 male (47%) patients. The mean age of these patients was 26.6 ± 2.7 (range, 5–61 years). The age distribution of melon-allergic patients is showed in Figure 1. The age of most patients ranged from 11 to 40 years, although there was predominance between 21 and 30 years. The mean age of patients beginning to experience melon allergy symptoms was 18.7 ± 2.6 years (range, 2–56 years).

Melon Allergy Symptoms

All of the melon-allergic patients had oral symptoms. Thirteen patients also experienced extraoral symptoms (19.7%); among them, 5 patients (7.6%) reported gastrointestinal symptoms, 3 patients (4.5%) reported conjunctivitis, 3 patients (4.5%) reported contact urticaria, 1 patient (1.5%) reported rhinitis, and 1 patient (1.5%) reported contact urticaria and conjunctivitis. Oral symptoms preceded extraoral symptoms, and none of the patients experienced generalized urticaria or anaphylaxis. The characteristics of patients who had extraoral reactions to melon are shown in Table 1.

Melon Allergy and Skin Test

The results of SPPTs with fresh melon pulp were positive in all patients in the melon allergy group. All of the patients were also skin tested with 3 commercially available melon extracts, and a positive skin test result was elicited in 12% of the patients in the first case, 17% in the second case, and 90% in the third case.

Figure 1. Age distribution of melon-allergic patients.

Table 1. Clinical Features of Patients with Extraoral Symptoms

Age, y/Sex/Age of onset, y	Local symptoms	Extraoral symptoms	Pollen symptoms
28/F/18	OS	C	RC
32/F/18	OS	CU	RC, A
28/F/19	OS	C	RC
17/F/16	OS	CU	RC, A
31/M/27	OS	GIS	RC, A
20/M/16	OS	GIS	RC
27/M/13	OS	CU, C	RC, A
12/F/7	OS	C	RC, A
20/F/8	OS	CU	RC, A
29/F/17	OS	GIS	RC, A
29/M/23	OS	R	RC
53/M/18	OS	GIS	RC, A
29/F/28	OS	GIS	RC, A

Abbreviations: A, asthma; C, conjunctivitis; CU, contact urticaria; GIS, gastrointestinal symptoms; OS, oral symptoms; R, rhinitis; RC, rhinitis/conjunctivitis.

Melon Allergy and Other Foods

In general, most of the fresh fruits and nuts were often able to elicit some positive skin test results. Excluding other Cucurbitaceae fruits, as many as 92% of the patients had positive skin test results to kiwi, but fig, peach, pear, apple, and grape also frequently elicited positive skin test results. The frequency of positive skin test results and symptoms to fruits and nuts is shown in Figure 2.

Many of these positive skin test results were not accompanied by symptoms on eating the food. The fruit most frequently self-reported by the patients to elicit symptoms was peach, which caused reaction in 62% of patients. Fig

(48%) and kiwi allergy (42%) were also frequently reported by melon-allergic patients. Forty-seven percent of patients reported symptoms caused by some nuts, with walnut (35%) and hazelnut (18%) most frequently implicated.

An interesting aspect of the evaluation was the latex allergy. Twenty-three percent of the patients were sensitized to latex as showed by skin tests, but as many as 13% had a clinical latex allergy.

Pollinosis and Melon Allergy

All of the patients were sensitized to pollens and had symptoms of pollen allergy, so there were no non-pollen-allergic patients in the melon allergy group. The results of pollen skin tests in both groups and the OR and 95% CI for each pollen are shown in Table 2. Some differential features of the pollinosis in melon allergy were a higher prevalence of asthma and a higher frequency of sensitization to several weed and tree pollens. We should first emphasize the high sensitization frequency to all pollens tested in the melon allergy group; the lowest sensitization frequency was higher than 65.6% (*Ambrosia*), excluding *Parietaria* pollen. The increase in pollen sensitization frequency was statistically significant for most of the tree and weed pollens, with the exception of *Parietaria*. This increase in sensitization frequency had its highest OR in pollens such as *Ulmus* (42.8), *Ambrosia* (22.4), *Chenopodium* (20.1), and *Populus* (16.1), increasing from less than 20% of the patients to more than 80%.

A total of 22 patients (33%) had rhinoconjunctivitis and 44 (67%) had rhinoconjunctivitis and asthma. Meanwhile, in the pollen control group, 49 patients (52%) reported rhinitis and 46 (48%) reported asthma. The prevalence of asthma among pollinic patients allergic to melon was statistically higher than

Figure 2. Frequency of positive skin test results and self-reported symptoms to fruits and nuts.

Table 2. Sensitization Frequency to Grass, Tree, and Weed Pollens in Melon-Allergic Patients and Pollen-Allergic Patients (Control Group)

Pollen	Frequency, %		OR (95% CI)*	P value
	Melon allergy group	Pollen allergy group		
<i>Phleum pratense</i>	98.4	97.9	1.4 (0.1–15.7)	0.78
<i>Lolium perenne</i>	98.4	97.9	1.4 (0.1–15.7)	0.78
<i>Cynodon dactylon</i>	96.8	87.4	4.6 (1.1–19.2)	0.02
<i>Secale cereale</i>	95.2	91.6	1.9 (0.5–7.5)	0.31
<i>Ambrosia trifida</i>	87.1	24.2	22.7 (10.5–49.1)	<0.001
<i>Artemisia</i> sp	65.6	29.5	4.5 (2.3–8.6)	<0.001
<i>Chenopodium album</i>	95.2	49.5	21.4 (8.0–56.9)	<0.001
<i>Parietaria judaica</i>	49.2	38.9	1.6 (0.8–2.9)	0.23
<i>Plantago lanceolata</i>	95.2	64.2	11.7 (4.1–33.2)	<0.001
<i>Taraxacum vulgare</i>	79.0	36.8	6.4 (3.2–12.7)	<0.001
<i>Olea europeae</i>	98.4	82.1	14.2 (2.8–70.6)	<0.001
<i>Fraxinus excelsior</i>	93.5	65.3	8.2 (3.1–21.9)	<0.001
<i>Ulmus glaba</i>	90.3	18.9	42.8 (19.1–97.3)	<0.001
<i>Betula alba</i>	88.7	45.3	10.2 (4.6–22.6)	<0.001
<i>Populus nigra</i>	90.3	34.7	18.8 (8.3–42.3)	<0.001

Abbreviations: CI, confidence interval; OR, odds ratio.

* Association between each pollen and melon allergy. There was statistical significance ($P < 0.05$) when 1.0 was not included in the CI.

that observed in the pollen control group (OR, 2.13; 95% CI, 1.11–4.08; $P < 0.05$).

When evaluating the first allergy (to melon or pollen) reported by melon-allergic patients, we found that symptoms of pollen allergy were the first to appear in 44 patients (67%), symptoms of melon allergy were the first to appear in 15 patients (23%), and symptoms to melon and pollen allergy appeared in the same year in 7 patients (11%).

DISCUSSION

Melon is a frequent cause of allergy to fresh fruits.^{8,13,14,17,18} The aim of this article was to describe the clinical features of melon allergy, in which the diagnosis was based on a convincing clinical history, positive SPPT results, and a positive open oral challenge test result to melon. Double-blinded challenge test has been considered the “gold standard” for the diagnosis of food allergy.^{19–21} The blindness of a provocation test in fresh fruit allergy raises many problems, which are summarized by Ortolani et al.²² In addition to this, some recipes to mask fresh fruits in the double-blinded challenge test have been reported, although they have not been validated.²³

Melon allergy is mainly experienced by people younger than 30 years. Although some reports^{7,24,25} have described a predominance of female patients with fruit allergy, the results of our study showed no predominance of either sex.

Melon allergy usually presents as oral symptoms. Nonetheless, 19.7% of the patients in our study experienced extraoral symptoms. In general, allergy to melon is not a serious disease, since most people continue eating the fruit and none of the patients we studied experienced generalized urticaria or

hypotension. Previous data on melon allergy^{13,17} also indicate that melon allergy is not a serious condition. This was reinforced by previous experience in our area, where the existence of systemic reactions through ingestion of other fruits (ie, peach) has been reported.¹² Nonetheless, these data are different from those of Rodriguez et al,¹⁸ who reported 2 severe reactions, which were not confirmed by open or blind challenge tests.

Allergy to fresh fruit is commonly described in the context of pollen allergy, but we reported that 1 of 5 patients allergic to peach was not allergic to pollens.¹¹ Nonetheless, the results of this study show that there were no non-pollen-allergic patients who had melon allergy; all patients had a concomitant pollinosis. These data are different from those of peach allergy¹¹ but reinforce the previous data reported by Anderson et al,¹³ in which the patients were exposed to different pollens.

Association and cross-reactivity between melon and some pollens have been reported (eg, melon-ragweed¹³ or melon-plantago and grasses²⁶). It is possible that pollen-fruit associations might vary according to the prevalence of certain pollens in each area and the dietary habits of the population. In this sense, we should remember that grasses and trees make up the major pollinating plants in the Madrid area, and, in contrast with other geographic areas, some major allergenic weeds (*Ambrosia* or *Parietaria*) or trees (*Betula*) are rarely found or are totally absent.^{11,27} Our results confirmed these previous associations, but they also showed a higher percentage of sensitization to several weed and tree pollens, spreading the pollens-melon associations. We also observed that the prevalence of asthma among pollinic patients allergic to melon was higher than that observed in the pollinic control group (OR, 2.13). To the best of our knowledge, the higher frequency of asthma in pollen-allergic patients with concomitant fruit allergy has only been reported in the literature by 2 works concerning melon and peach allergy.^{11,13} In our opinion, this finding should be evaluated in other areas and with other foods, since it could represent a risk factor for asthma.

It has been reported that patients allergic to vegetables often have positive skin test results and symptoms to other plant-derived foods. Anderson et al¹³ established a link between melon and banana, since 32% of the patients were allergic to both fruits. In our experience, patients allergic to melon were usually sensitized to several fresh fruits and nuts as showed by a positive skin test result, mainly to kiwi, fig, peach, and pear. Nonetheless, peach (62%), fig (48%), nuts (47%), kiwi (42%), and banana (37%) were the fruits most frequently reported to elicit symptoms by melon-allergic patients. These data suggest the existence of a spread of immunologic and clinical cross-reactivity. Future studies should be performed to determine which antigens are responsible for this spread in cross-reactivity (eg, profilin, carbohydrate cross-reactive determinant, lipid transfer protein).

Patients allergic to latex often experience the so-called latex-fruit syndrome through ingestion of some foods.²⁸ Fruits implicated have mainly been banana, avocado, and chestnut, although other foods have also been involved. This

was attributed to cross-reactivity to class I chitinases.²⁹ The concomitant pollen allergy in patients allergic to latex seems to be an important factor in the determination of sensitization to the other foods, such as melon, which might be attributed to profilin sensitization.^{30–32} In our study, allergy to latex was not a condition linked to most melon-allergic patients; however, it is remarkable that up to 23% of melon allergy had a concomitant latex sensitization, although only 13% of patients had a clinical latex allergy.

The sensitivity of commercially available melon extracts was variable, once more demonstrating the absence of standardization and the need to improve these tests.^{22,33–37} The absence of standardization of food extracts is a current problem in daily clinical practice. Today, it is possible to quantify the major allergen content¹⁶ and biologic activity³³ in food-derived extracts. In our opinion, it would be necessary to standardize and to label food extracts to end any confusion.

CONCLUSION

We have reported clinical features of melon allergy and associations to other fruit and pollen allergies. The most important conditions linked to melon allergy are pollen allergy (100%), allergy to other nonrelated fruits, mainly peach (up to 62%), and latex sensitivity (up to 23%). Some differential features of the pollinosis in melon allergy were a higher prevalence of asthma and a higher frequency of sensitization to several weed and tree pollens. Future immunologic studies should be performed to elucidate the antigens involved in these findings.

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REFERENCES

1. Sociedad Española de Alergología e Inmunología Clínica y Alergia e Inmunología Abelló SA. *Alergológica: Factores Epidemiológicos Clínicos y Socioeconómicos de las Enfermedades Alérgicas en España: Alergia a Alimentos*. Madrid, Spain: Nilo Industria Gráfica; 1995.
2. Kivity S, Dunner K, Marian Y. The pattern of food hypersensitivity in patients with onset after 10 years of age. *Clin Exp Allergy*. 1994;24:19–22.
3. André F, André C, Colin L, Cacaraci F, Cavagna F, Cavagna S. Role of new allergens and of allergens consumption in the increased incidence of food sensitizations in France. *Toxicology*. 1994;93:77–83.
4. Martin Esteban M. Adverse food reactions in childhood: concept, importance and present problems. *J Pediatr*. 1992;21:1–3.
5. Hannuksela M, Lathi A. Immediate reaction to fruits and vegetables. *Contact Dermatitis*. 1977;3:79–84.
6. Ericksson EN. Food sensitivity reported by patients with asthma and hay fever. *Allergy*. 1978;33:189–196.
7. Eriksson EN, Formgren H, Sveno Niv SE. Food hypersensitivity in patients with pollen allergy. *Allergy*. 1982;37:437–480.
8. Cuesta-Herranz J, Lázaro M, Figueredo E, et al. Allergy to plant-derived fresh foods in a birch and ragweed-free area. *Clin Exp Allergy*. 2000;30:1411–1416.
9. Sánchez-Monge R, Lombardero M, García-Selles FJ, Barber D, Salcedo G. Lipid-transfer proteins are relevant allergens in fruit allergy. *J Allergy Clin Immunol*. 1999;103:514–519.
10. Pastorello EA, Farioli L, Pravettoni V, et al. The major allergen of peach (*Prunus persica*) is a lipid transfer protein. *J Allergy Clin Immunol*. 1999;103:520–526.
11. Cuesta-Herranz J, Lázaro M, Martínez A, et al. Pollen allergy in peach allergic patients: sensitization and cross-reactivity to taxonomically unrelated pollens. *J Allergy Clin Immunol*. 1999;104:688–694.
12. Cuesta-Herranz J, Lázaro M, De las Heras M, et al. Peach allergy pattern: experience in 70 patients. *Allergy*. 1998;53:78–82.
13. Anderson LB, Dreyfuss EM, Logan J, Johnstone DE, Glaser J. Melon and banana sensitivity coincident with ragweed pollinosis. *J Allergy Clin Immunol*. 1970;45:310–319.
14. Joral A, Villas F, Garmendia J, Villareal O. Adverse reactions to food in adults. *J Investig Allergol Clin Immunol*. 1995;5:47–49.
15. Dreborg S, Foucard T. Allergy to apple, carrot and potato in children with birch pollen allergy. *Allergy*. 1983;38:167–172.
16. Subcommittee on Skin Tests of the European Academy of Allergy and Clinical Immunology. Skin tests used in type I allergy testing [position paper]. *Allergy*. 1989;44(Suppl):1–59.
17. García Ortíz JC, Cosmes Martín P, López-Asunsolo A. Melon sensitivity shares allergens with *Plantago* and grass pollens. *Allergy*. 1995;50:269–273.
18. Rodríguez J, Crespo JF, Burks W, et al. Randomized, double-blind, crossover challenge study in 53 subjects reporting adverse reactions to melon (*Cucumis melo*). *J Allergy Clin Immunol*. 2000;106:968–972.
19. Bock SA, Lee WY, Remigio LK, May CD. Studies of hypersensitivity reactions to foods in infants and children. *J Allergy Clin Immunol*. 1978;62:327–334.
20. Sampson HA. Role of immediate food hypersensitivity in the pathogenesis of atopic dermatitis. *J Allergy Clin Immunol*. 1983;71:473–480.
21. Bernstein M, Day JH, Welsh A. Double blind food challenge in the diagnosis of food sensitivity in the adult. *J Allergy Clin Immunol*. 1982;70:205–210.
22. Ortolani C, Ispano M, Pastorello EA, Ansaloni R, Magri GC. Comparison of results of skin prick tests (with fresh foods and commercial food extracts) and RAST in 100 patients with oral allergy syndrome. *J Allergy Clin Immunol*. 1989;83:683–690.
23. Noé D, Bartemucci L, Mariani N, Cantari D. Practical aspects of preparation of foods for double-blind, placebo-controlled food challenge. *Allergy*. 1998;53(Suppl):75–77.
24. Ortolani C, Ispano M, Pastorello E, Bigi A, Ansaloni R. The oral allergy syndrome. *Ann Allergy*. 1988;61:47–52.
25. Asero R, Massironi F, Velati C. Detection of prognosis factors for oral allergy syndrome in patients with birch pollen hypersensitivity. *J Allergy Clin Immunol*. 1996;97:611–616.
26. García-Ortiz JC, Ventas P, Cosmes P, López-Asunsolo A. An immunoblotting analysis of cross-reactivity between melon, and plantago and grass pollens. *J Investig Allergol Clin Immunol*. 1996;6:3789–3382.
27. Subiza J, Jerez M, Jimenez JA, et al. Allergenic pollen and pollinosis in Madrid. *J Allergy Clin Immunol*. 1995;96:15–23.
28. Blanco C, Carrillo T, Castillo R, Quiralte J, Cuevas M. Latex

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- allergy: clinical features and cross reactivity with fruits. *Ann Allergy*. 1994;73:309–314.
29. Diaz-Perales A, Collada C, Blanco C, et al. Cross-reactions in the latex-fruit syndrome: a relevant role of chitinases but not of complex asparagine-linked glycans. *J Allergy Clin Immunol*. 1999;104:681–687.
30. Diez-Gomez ML, Quirce S, Cuevas M, et al. Fruit-pollen-latex cross-reactivity: implication of profilin (Bet v 2). *Allergy*. 1999;54:951–961.
31. Quirce S, Bombin C, Aleman A, Sastre J. Allergy to latex, fruit, and pollen. *Allergy*. 2000;55:896–898.
32. Levy DA, Mounedji N, Noirot C, Leynadier F. Allergic sensitization and clinical reactions to latex, food and pollen in adult patients. *Clin Exp Allergy*. 2000;30:270–275.
33. Cuesta-Herranz J, Lázaro M, Martínez A, et al. A method for quantitation of food biologic activity: results with peach allergen extracts. *J Allergy Clin Immunol*. 1998;102:275–280.
34. Herian AM, Bush RK, Taylor SL. Protein and allergen content of commercial skin test extracts for soybeans. *Clin Exp Allergy*. 1992;22:461–468.
35. Hefle SL, Helm RM, Burks AW, Bush RK. Comparison of commercial peanut skin test extracts. *J Allergy Clin Immunol*. 1995;95:837–842.
36. Sampson HA. Comparative study of commercial food antigen extracts for the diagnosis of food hypersensitivity. *J Allergy Clin Immunol*. 1988;82:718–726.
37. Norgaard A, Skov PS, Bindslev-Jensen C. Egg and milk allergy in adults: comparison between fresh foods and commercial allergen extracts in skin prick test and histamine release from basophils. *Clin Exp Allergy*. 1992;22:940–947.
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Erratum

There was an error in the article “‘Real-world’ effectiveness of daily controller medicine in children with mild persistent asthma” by Don A. Bukstein, MD, Allan T. Luskin, MD, and Avi Bernstein, BA, published in the May 2003 issue of the *Annals (Ann Allergy Asthma Immunol*. 2003;90:543–549). On page 544, in the second paragraph below Methods, the first sentence should read, “Consecutively enrolled children were assigned to treatment with either two 44- μ g puffs twice daily of inhaled fluticasone propionate (fluticasone subjects) or one 5-mg tablet daily of oral montelukast (montelukast subjects) on an alternating basis (**due to age and weight, 5 subjects received one 10-mg tablet daily**), ie, odd-numbered enrolled subjects received fluticasone, and even-numbered enrolled subjects received montelukast.” The authors regret the error.