

*Wellington Asthma Research Group
Wellington School of Medicine and Health Sciences
PO Box 7343
Wellington
New Zealand
E-mail: rob.siebers@otago.ac.nz

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Turnip and zucchini: new foods in the latex-fruit syndrome

C. Pereira*, B. Tavares, G. Loureiro, M. Lundberg
C. Chieira

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Since the first description of systemic reaction induced by plant-derived food in

a patient allergic to latex (1), several reports of cross-reactivity between latex, fruits and vegetables had been described. In 1994, Blanco et al. (2) suggested the designation latex-fruit syndrome to describe this syndrome.

Turnip (*Brassica rapa*) belongs to the *Brassicaceae* family also called *Cruciferae*. This is known as the mustard or cabbage family. Turnip is a root vegetable commonly grown in temperate climates worldwide for its white, bulbous taproot.

Zucchini (*Cucurbita pepo*), also known as courgette, belongs to *Cucurbitaceae* family. It is a small summer marrow or squash, also commonly called Italian squash. Despite being a fruit, zucchini is considered to be a vegetable in culinary and is traditionally picked when very immature.

We report the case of a female patient, 42 years old, worker in the mineral

Turnip and zucchini: new vegetables responsible for cross-reactivity between latex and plant-derived foods.

extraction industry for several years, with significant exposure to rubber, describing multiple episodes of anaphylaxis. The first episode took place 30 min after ingestion of chestnut when she was 25 years old. After this, she reported other episodes after ingestion of almond nut, hazelnut, banana, kiwi, mango, avocado, fig and tomato and recently also with both raw and cooked zucchini and turnip. The symptoms always included oral allergy syndrome, urticaria, angioedema and glottis oedema. All the episodes needed medical attention in the Emergency Department. She also describes an anaphylaxis episode after blowing latex balloons. The only medical care with exposure to latex material was at 23 years old when she had normal delivery.

After the first observation in our Allergy Department (2005), it became clear a clinical history suggestive of latex-fruit syndrome.

The skin prick tests with commercial extracts to aeroallergens, latex (ALK Abelló, Madrid, Spain), fruits, nuts, vegetables and legumes (Leti, Madrid,

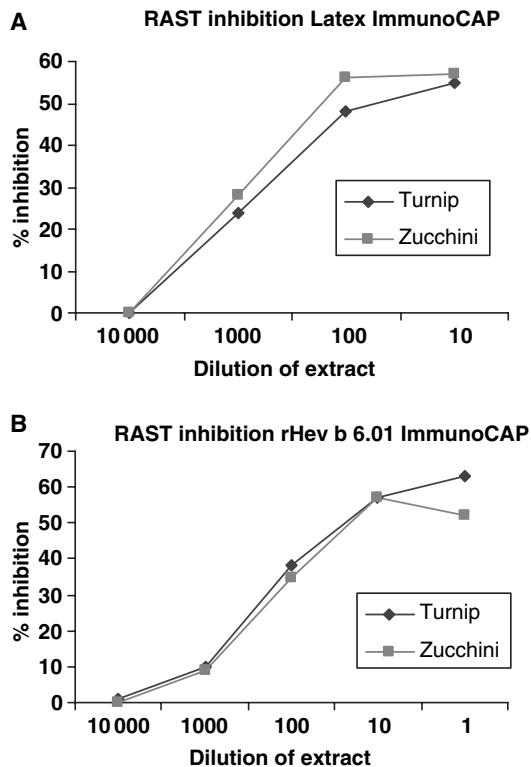


Figure 1. RAST inhibition with turnip and zucchini extracts. (A) RAST inhibition of latex ImmunoCAP. (B) RAST inhibition of rHev b6.01 ImmunoCAP.

Spain) revealed positive (wheal diameter 3 mm above the negative control) to kiwi, pear, banana, watermelon, cherry, passion fruit, walnut, pistachio and latex (wheal diameters of 4, 5, 5, 6, 5, 5, 4, 3 and 12 mm, respectively; positive control 8 mm and negative control 0 mm).

The skin prick-prick tests to several raw foods and latex revealed positive to latex, chestnut, walnut, pistachio nut, blackberry, tomato, zucchini and turnip (wheal diameter of 12, 13, 5, 4, 4, 5, 7 and 5 mm; positive control 6 mm and negative control 0 mm).

The total serum IgE level was 34 IU/l and the serum-specific IgE to latex 3.2 KU/l (ImmunoCAP™, Phadia, Uppsala, Sweden). As the sensitization to turnip and zucchini were not previously described in the latex-vegetable-fruit syndrome, we decided to proceed with further investigation. The specific serum IgE to turnip and zucchini revealed 0.64 and <0.35KU/l, respectively. Previous incubation of serum with turnip and latex nondiluted extracts resulted in an inhibition of 99.3% and 93%, respectively, of turnip ImmunoCAPs reaction. Latex ImmunoCAP reaction was inhibited with both zucchini and turnip extract, respectively (Fig. 1A). Specific IgE levels to rHev b1 (Rk215), rHev b5 (Rk218) and rHev b6.01 (Rk219) were 0, 0.31 and 2.9 KU/l, respectively. Inhibition study of rHev b6.01 with nondiluted extracts of turnip and zucchini revealed an inhibition of 63% and 52.3%, respectively (Fig. 1B).

Despite cross-reactivity between zucchini and latex had been previously described (3) with this study, we could prove for the first time cross-reactivity between turnip and latex and identify Hev b6.01, the chitin-binding protein, as the protein responsible in this latex-fruit syndrome. Turnip and zucchini should be added to the long list of plant-derived foods cross-reactive to latex and sensitization to these vegetables must be assessed in patients with latex anaphylaxis.

*Serviço de Imunoalergologia
Hospitais da Universidade de Coimbra
Apartado 9057
3001-301 Coimbra
Portugal
Tel: +351 917205277
Fax: +351 239836262
E-mail: celsopereira@netcabo.pt

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Effectiveness of omalizumab in monozygotic twin sisters with severe allergic asthma

J. Just*, F. Sahraoui, V. Le Gros, A. Grimfeld

Between 5% and 10% of children worldwide suffer from asthma (1). The familial nature of asthma is well known; the risk for a first cousin of an asthmatic patient is multiplied by 2.5–3 compared with the general population (2). Studies of twins have confirmed this genetic component finding higher concordance of asthma among monozygotic (58.97%) than dizygotic twins (23.64%) (3). We report an exceptional clinical situation of monozygotic twin sisters with severe allergic asthma where one was treated with a monoclonal anti-IgE antibody (omalizumab), and the other with a placebo in a randomized, double-blind, placebo-controlled study (4).

Premature monozygotic twin sisters developed bronchopulmonary dysplasia secondary to hyaline membrane disease.

Omalizumab in twin sisters.

Afterwards, the two sisters had similar respiratory history; first, asthma with exacerbations related to viral infections in infancy, then at the age of 6, developing respiratory allergies to various aeroallergens with symptoms of seasonal rhinitis and persistent atopic dermatitis. Despite treatment with high doses of inhaled corticosteroids in combination with long-acting β_2 -mimetics and delayed-release theophylline, their asthma remained poorly controlled. Repeated severe exacerbations (>10 hospitalizations between 4 and 8) led to several stays in the mountains for climatic treatment. In 1998 at the age of 12, the twins were enrolled in a 32-week randomized, double-blind, placebo-controlled study (4). One received omalizumab and the other a placebo. The twin receiving omalizumab was able to reduce her long-term treatment with fluticasone to 500 $\mu\text{g/day}$, while the other required treatment with fluticasone at a dose of 1250 $\mu\text{g/day}$ and regular courses of oral corticosteroids due to persistent exacerbations. They were both then treated with omalizumab during a 6-year extension phase. During this time, there was clinical improvement with the absence of hospitalizations for severe attacks, disappearance of pollen-induced rhinitis and improved respiratory function (Fig. 1) under reduced long-term treatment with fluticasone 500 $\mu\text{g/day}$ and occasional administration of oral corticosteroids. The adult height of the twin who first received omalizumab was 159 cm compared with 156 cm for the other.

The effectiveness of omalizumab in adolescents has previously been demonstrated in a pooled analysis showing that the relative risk of an adolescent aged between 12 and <18 presenting an asthma exacerbation when treated with omalizumab was 0.470 compared with the control group (95% CI: 0.318–0.695; $P = 0.0002$) (5). Our observation of monozygotic twins one treated with omalizumab and the other with a placebo for 32 weeks, then both with omalizumab for 6 years, and living in the same environment, supports these findings and suggests the effectiveness of omalizumab on a specific phenotype of severe asthma.