

Mango allergy in a latex-sensitized patient

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Key words: cross-reactivity; latex allergy; mango allergy.

• ALLERGY to such different fruits and nuts as banana, chestnut, kiwi, or avocado has frequently been described. However, only a few cases of mango allergy have been reported (1-4). Like other food allergens, that of the mango can produce oropharyngeal syndrome, urticaria, and sometimes anaphylaxis (1-3), possibly causing life-threatening complications. Contact dermatitis of the face and lips (4) and asthma (5) after mango consumption have also been described.

On the other hand, allergy to tropical fruits has been identified in many latex-

An IgE-mediated reaction of rhinoconjunctivitis and dyspnea.

sensitized patients in whom cross-reactivity between latex and fruits – the so-called latex-fruit syndrome – has been shown (5). We report a case of mango allergy in a latex-sensitized patient. A 45-year-old nurse had been diagnosed as having latex sensitization 3 years before. Her clinical manifestations when in contact with latex included rhinoconjunctivitis, bronchial asthma, and contact urticaria. She usually avoided contact with latex. The patient had occasionally eaten mango for 2 years, without problems. After eating one fresh mango, she immediately suffered oral allergy syndrome, rhinoconjunctivitis, cough, and dyspnea requiring treatment with antihistamines, inhaled β_2 -agonists, and corticosteroids. Skin prick-prick tests with fresh mango were

positive (2+) in the patient but negative in three healthy controls. Class 3 mango-specific IgE levels (RAST) were moderately increased (10.4 kU/l). An immunoblot inhibition assay was performed to investigate possible cross-reactivity between mango and latex (Fig. 1). The results showed IgE binding to mango proteins of 25–50 kDa in the patient serum, as well as IgE binding to latex proteins of 40 kDa. Cross-reactivity between mango and latex was not shown.

Only a few cases of mango allergy have been reported, to our knowledge. However, the real frequency may be underestimated because of low consumption of this fruit in the northern hemisphere. The mango plant contains several

substances thought to be sensitizers such as cardol, uroshiol, β -pinene, and limonene (1). The hypersensitivity mechanism leading to anaphylactic reactions is probably mediated by IgE, as shown by the skin test positivity and IgE RAST detection. Other possible mechanisms of less severe reactions, such as contact urticaria, would be mediated by antibodies of IgG4 type (1).

Latex allergy is common among certain professionals at increased risk such as health-care workers. Many latex-sensitized patients show the latex-fruit syndrome, which consists of oropharyngeal syndrome and sometimes anaphylaxis (5). Cross-reactivity between latex and some fruits and nuts – banana,

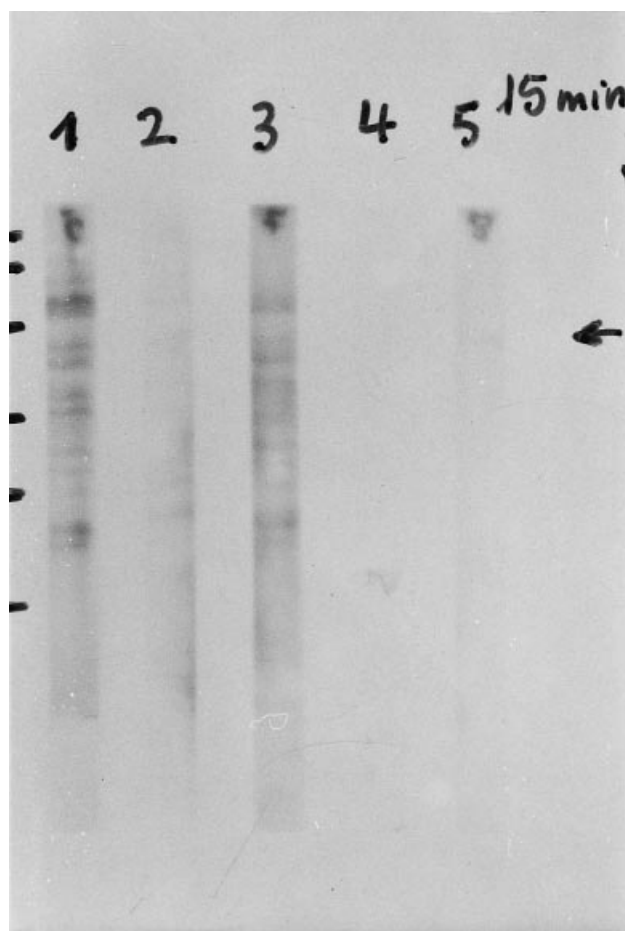


Figure 1.

chestnut, kiwi, or avocado – has been described [5]. Biologic cross-reactivity between latex and mango has also been reported [5]. Cross-reactivity is believed to be due to a common epitope shared by both latex and fruits. Several plant proteins such as profilins, endo-1,3- β -glucosidases, and patatin have been suggested, although the responsible proteins have not yet been identified [5]. We could not show cross-reactivity between latex and mango in our patient, so we made the diagnosis of independent mango allergy in a latex-sensitized patient. Therefore, it is important to bear in mind that not every case of fruit allergy in a latex-sensitized patient can be diagnosed as latex-fruit syndrome if the proper biologic tests have not been performed.

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Accepted for publication 6 April 1999
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ISSN 0105-4538

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IL-4R α gene Ile50Val polymorphism

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Key words: atopy; interleukin-4 receptor; Ile50Val polymorphism; Singapore.

● ATOPY is the genetic predisposition to the development of high levels of IgE against normally innocuous allergens, and it frequently manifests clinically as allergic asthma, eczema, or rhinitis. In a Japanese population, homozygotes for the Ile50 allele polymorphism in the extracellular domain of the α chain of the interleukin-4 receptor complex (IL-4R α)

have been found to be in excess among the atopics, leading the authors to

conclude that IL-4R α is a major atopy locus (1). To confirm this association, we determined the frequency of the Ile50Val genotype in relation to atopy in the three main ethnic groups in the Singapore population.

Subjects were recruited from patients attending the Asthma Clinic of the Children's Medical Centre, National University Hospital. Atopy was defined as the presence of any clinical symptom of allergic asthma, allergic rhinitis, or atopic eczema, either a total serum IgE level 2 SD above mean for age (2), and/or the presence of sensitization to the house-dust mite *Dermatophagoides pteronyssinus* as demonstrated by skin prick test or FAST. Controls were volunteers recruited from hospital and institute staff and were defined as individuals without any history of the above-mentioned atopic diseases, a normal serum IgE level corrected for age, and negative reaction to *D. pteronyssinus* on FAST.

Genomic DNA was extracted from blood with the QIAamp Blood Kit (QIAGEN GmbH,

There was no association in Chinese, Malay, and Indian atopics.

Germany). Separate PCR reactions were performed with the two allele-specific sense primers (corresponding to nucleotide positions 383–398) and a common antisense primer 5'-CGCTGGGCTTGAAGGAG-3' corresponding to nucleotide positions 514–530 in the IL-4 receptor gene. The sequence for the Val50-specific primer was 5'-GAAGCCCACACGTGTG-3'; for Ile50, it was 5'-GAAGCCCACACGTGTA-3'. Amplification products were detected by ethidium bromide staining after electrophoresis through a 2% NuSieve gel. To confirm the accuracy of genotyping, we performed cycle sequencing on 13 samples. The region of interest was amplified with AmpliTaq Gold and primers 5'-CGGAATTCCGAGGCCACACGTGT-3' and 5'-CGCTGGGCTTGAAGGAG-3'. Bands were excised from agarose gels after electrophoresis and purified with the QIAquick Gel Extraction Kit (QIAGEN GmbH, Germany). Cycle sequencing was performed with the ABI PRISM BigDye Terminator Cycle Sequencing Ready Reaction Kit (PE Applied Biosystems, USA). Products were analyzed with the ABI 377 Genetic Analyzer. Data from sequencing and AS-PCR genotyping concurred in all cases.

A total of 241 individuals (108 controls, 133 atopics) from the three major ethnic groups in Singapore were genotyped for this polymorphism. Association analyses were performed separately for each ethnic group. Fisher's exact tests were used for comparing distribution and detecting associations. The allelic distributions conformed to the Hardy–Weinberg equilibrium.

Frequency distributions of the three genotypes are presented in Table 1. We were unable to replicate Mitsuyasu et al.'s (1) finding of an excess of homozygotes for the Ile50 allele among the atopics. Instead, there was overrepresentation of the Val50 allele in the atopic group compared to the controls, but this was statistically significant only for the Indians ($P < 0.05$). Ile50 was found to be the more abundant allele for controls in all three groups, whereas it was the rarer allele for