

**704 Occupational Management of the Type I Latex Allergic Patient**  
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We are reporting our clinical experience with 73 patients diagnosed with occupationally related type I latex-allergy from December 1994 to May 1999. There is some controversy whether latex-sensitive health care workers must be removed from the workplace to avoid respiratory exposure to latex aeroallergens. We did a retrospective chart review of 73 patients with type I symptoms corroborated by positive PST and/or positive CAP/AlaSTAT *in vitro* assays. Patient management, based on interaction between the patient and the treating physician, was coded as the following: (A) patients switched to non-latex gloves and returned to the same working environment, powdered or powder-free; (B) patients switched to non-latex gloves and returned to work while co-workers also switched to powder-free latex or non-latex gloves; (C) patients vocationally rehabilitated and removed from the workplace. 51 patients (70%) were able to return to work with a simple change of gloves (A), 6 (8%) were returned to a more restrictive environment (B), and 16 (22%) were taken out of the workplace (C). 11 of the 16 Type C patients had originally been managed as Type B but needed to be rehabilitated when their employers failed to accommodate them with powder-free environments. In 14 of the 16 Type C cases, the treating physician decided to remove the patient from work. All 57 (78%) of the patients who have remained at work have no longer suffered from or complained of progressive IgE mediated latex-allergic symptoms. According to our clinical experience, many latex-positive health care workers may safely return to their workplace when simply switched to non-latex gloves. Management still needs to be individualized based on the nature of work, the severity of the patient's symptoms, and the patient's personal risk tolerance.

**705 Cross-reactions in the Latex-Fruit Syndrome: A Relevant Role of Chitinases but Not of Cross-reactive Carbohydrate Determinants**  
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Cross-reactions among latex and plant foods have been widely reported. Although the cross-reactive components have not been well identified, class I chitinases seem to be responsible, at least in chestnut, avocado and banana. We sought to evaluate the potential role of chitinases and complex glycans as cross-reactive determinants linked to latex-food allergy.

Extracts from 20 different plant foods and from latex were obtained. These preparations were immunodetected with anticomplex glycans and antichitinase sera raised in rabbits, as well as with sera from patients with latex-fruit allergy and sera from patients allergic to latex without food allergy. Immunoblot inhibition assays were also carried out by using a class I chitinase purified from avocado or a latex extract as inhibitors.

Reactive proteins of 30 to 45 kd (putative class I chitinases) were recognized by both specific polyclonal antibodies to chitinases and sera from patients with latex-fruit allergy in cherimoya, passion fruit, kiwi, papaya, mango, tomato, and flour wheat extracts. Both the latex extract and the class I chitinase from avocado strongly or fully inhibited the IgE binding by these components when tested in immunoblot inhibition assays. Additional bands of 16 to 20 kd, 23 to 28 kd, and 50 to 70 kd were detected by the antichitinase serum but not with the patient sera. The putative 30- to 45-kd chitinases present in different food extracts did not react with a pool of sera from subjects allergic to latex but not to fruits. Very different immunodetection patterns were produced with the anticomplex glycan serum and the sera from allergic patients.

In conclusion, putative class I chitinases seem to be relevant cross-

reactive components in foods associated with the latex-fruit syndrome. On the contrary, cross-reactive carbohydrate determinants are not important structures in the latex-fruit cross-sensitization.

**706 Immunological Responses of Mice Following Administration of Natural Rubber Latex Proteins by Different Routes of Exposure**  
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Although the prevalence of IgE-mediated latex allergy has increased over the past decade, the circumstances that culminate in sensitization remain uncertain. The objective of these studies was to evaluate the role which sensitization route plays in the development of latex allergy using murine models representative of potential exposure routes by which health care workers (topical and respiratory) and spina bifida patients (subcutaneous) may be sensitized. BALB/c mice administered latex proteins by the subcutaneous, topical, intranasal or intratracheal routes exhibited dose responsive elevations in total IgE. Subcutaneous and intratracheal exposures resulted in comparable IgE levels > 8,000ng/ml 30 days after initial protein administration. Topical applications induced IgE production (~6,000ng/ml) in abraded and non-tape stripped mice following daily exposures for 7 weeks. Intranasal instillation resulted in the lowest total IgE concentrations (<1,500ng/ml). Active Cutaneous Anaphylaxis and *in vitro* splenocyte stimulation initially demonstrated specificity of the murine immune response to latex proteins in subcutaneously sensitized mice. Subsequently, immunoblot analysis was used to compare latex-specific IgE production amongst sensitization routes. Immunoblots of IgE from subcutaneously sensitized mice demonstrated recognition of latex proteins with molecular weights near 14 kDa and 27 kDa. These protein sizes are consistent with the molecular weights of major latex allergens (hev b 1 and hev b 3) to which high percentages of spina bifida patients develop antibodies. The 27 kDa latex protein was not evident following blotting of sera from mice sensitized by any other route. Mice sensitized by intratracheal or topical administration exhibited combined IgE recognition of latex proteins near 14 kDa, 35 kDa and 92 kDa; these molecular weights are similar to other latex allergens (hev b 6, hev b 2 and hev b 4) commonly recognized by IgE of health care workers. Sera from intratracheally sensitized mice demonstrated unique recognition of the 92 kDa latex protein. Mice sensitized to latex proteins by topical, intranasal, or intratracheal exposures exhibited bronchoconstriction (increased PENH) as measured by whole body plethysmography following respiratory challenge with latex proteins; subcutaneously sensitized mice were unresponsive. These differences in latex specific IgE immunoblot profiles, and the altered pulmonary responses, amongst the four different sensitization routes suggest that exposure routes leading to sensitization may play a role in determining the primary allergen(s) and the clinical manifestation of the allergic responses in latex allergy.

**707 Cytokine Profile in Latex-sensitized Health Care Workers Before and After Removal of Powdered Natural Rubber Latex Gloves in Their Workplaces**  
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Occupational exposure to natural rubber latex (NRL) has led to sensitization of healthcare workers (HCWs). The source of aerogen NRL in health care facilities are powdered NRL-gloves. Elimination of powdered NRL-gloves from the workplaces eliminated the detectable airborne NRL and permitted sensitized and allergic personnel to remain on the job [Allmers et al., J Allergy Clin Immunol 1998; 102: 841-6]. In an intervention study, examination of latex