

Occupational asthma to lobster and shrimp

SEAFOOD is known to cause occupational asthma. To our knowledge, lobster has been reported to cause occupational asthma in only one instance (1). We here report for the first time a case of occupational asthma caused by both lobster and shrimp.

■ A 34-year-old woman started working in a fishmonger shop in 1991. She had no previous history of asthma but had complained of hay fever for 10 years. She had never smoked. Two years after beginning her job, she developed urticaria when she had to handle lobster and shrimp. Six months later, she complained of rhinitis as well as cough and wheezing when she was cooking lobsters and handling raw shrimp. She was asymptomatic during weekends and on holidays. She was treated by albuterol as needed, but her symptoms progressively worsened. A few months later, after ingestion of a shrimp, she complained of itching, swelling in the mouth, and then stomach pain. She stopped working in February 1995 and her symptoms rapidly improved. Skin tests performed by the prick method with 22 common inhalants were entirely negative. Skin tests to seafood with commercial extracts (Hollister-Stier Aurora, ONT, Bencard, Mississauga, ONT; Omega, Montreal, QUE, Canada): clam, oyster, scallop, and mussel were negative whereas skin tests to lobster (Omega), shrimp, and crab (Hollister-Stier and Bencard) showed immediate whealing. Total IgE levels were increased (179 kU/l, normal values < 30).

■ Specific inhalation challenges were performed in June 1995. FEV₁ was 3.03 l (predicted: 2.88 l (2); 105% of predicted) and FVC was 3.5 l (predicted: 3.35 l; 104% predicted). On the control day, the subject was exposed to a saline solution (0.9% NaCl). No significant change in FEV₁ occurred afterward (Fig. 1). Nonspecific bronchial responsiveness to methacholine was assessed at the end of the day with a Wright nebulizer (output = 0.14 l/min) at tidal volume breathing for 2 min, by the procedure of Cockcroft et al. (3). PC₂₀ was 16 mg/ml (borderline hyperresponsiveness). On the next day, she inhaled increasing dilutions of the aqueous lobster extract prepared by cooking lob-

sters in ion-depleted water for 15 min. Meat of cooked lobster was removed and homogenized with PBS for 3 min. After mixing overnight at 4°C, extracts were centrifuged. The supernatant was concentrated and dialyzed against PBS. The subject experienced an immediate asthmatic reaction after 18 min of nebulization (23% fall of FEV₁). PC₂₀ was significantly decreased at the end of the day (3 mg/ml). PC₂₀ reversed toward normal values on the next day (28 mg/ml). Two days later, we exposed her to dilution of shrimp powder (10 mg/ml) nebulized in an exposure chamber. She experienced an immediate asthmatic reaction after a 1-h period of exposure (26% fall of FEV₁). PC₂₀ was 21 mg/ml at the end of the day.

■ The subject's serum was assayed for IgE antibodies by RAST. The serum was tested in duplicate, and results were expressed as percent binding of the amount of radiolabeled anti-IgE added to the disk. The patient showed substantial reactivity to lobster (5.8%), shrimp (16.7%), crab (3.6%), and crawfish (4.5%), while showing a negative reaction (1.1%) to the control disk HSA. The reactions were greatest to lobster and shrimp, the reaction to shrimp being approximately three times that to lobster.

*Eat – do not inhale –
your seafood.*

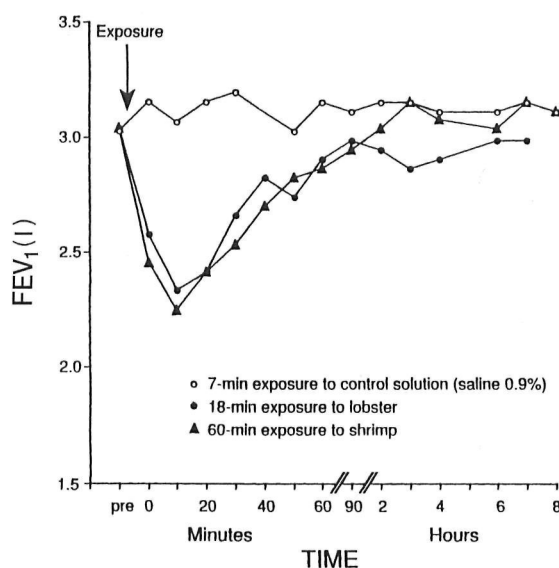


FIGURE 1 Results of specific inhalation challenges to lobster and shrimp.

■ Crustaceans are known to induce occupational asthma in workers employed in the seafood industry (4). Occupational asthma and immunologic sensitization caused by shrimp has been reported in several instances. Desjardins et al. (5) reported a prevalence of immediate sensitization of 16% in 56 food company workers. Although lobster is a shellfish frequently used in the food industry, occupational asthma caused by lobster is unusual. Indeed, to our knowledge, it has been described in only one instance, by Patel & Cockcroft (1) in a chef who prepared lobster. This subject was also sensitized to some other seafood: haddock, cod, oyster, clam, and scallop. Skin tests to shrimp were not done, but the subject complained also of allergic symptoms after ingestion of shrimp.

Occupational asthma and immunological sensitization caused by both lobster and shrimp can be explained by the fact that our subject was exposed to both kinds of shellfish and/or cross-reactivity between these two kinds. Although this was not examined in the present case, this cross-reactivity has been previously demonstrated by RAST inhibition in other cases (4), and the presence of shared allergic determinants between shrimp and lobster has been clearly described.

Interestingly, our subject was nonatopic and developed occupational asthma to high-molecular-weight agents. She also showed changes in PC₂₀ after the immediate reaction to lobster, but not to shrimp. No explanation of these unusual findings can be proposed.

The prevalence of immunologic sensitization and of occupational rhinoconjunctivitis and asthma caused by lobster remains to be assessed in the primary processing industry as well as among fishmongers.

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Antibodies to cow's milk proteins in chronic urticaria

Key words: cow's milk protein-reactive antibodies; specificity and isotype distribution; RAST-negative adults; urticaria/angioedema.

ANTIBODIES reactive to cow's milk protein (CMP) have been demonstrated in the serum of non-atopic patients with chronic urticaria (CU) (1). We searched for such serum antibodies in cow's milk-intolerant patients with CU and healthy volunteers. We used the Western-blot procedure to estimate which isotypes were represented most among reactive antibodies in both diseased and healthy persons.

■ The study was performed in patients with recurrent urticaria/angioedema, i.e., >6 weeks' duration, and healthy, randomly selected blood donors. Before undergoing dietary regimens, all patients were examined to exclude known causes of chronic urticaria (2). Those selected filled in the questionnaire to exclude occupational allergy, current therapy including birth-control pills, surgical or other therapeutic implants, reaction to insect bites, or inhalants.

■ The protocol is shown in Fig. 1. Diets were designed according to published schemes (3, 4). An E/P diet (elimination of or provocation with milk and dairy products) was introduced twice. The improvement or worsening of skin eruptions was estimated by a clinical scoring system (3, 5). No medication was allowed during the study. The IgE antibodies reactive to CAS (casein), ALA (α -lactalbumin), and BLG (β -lactoglobulin) were determined in serum by enzyme immunoassay (RAST, Pharmacia Diagnostics, Sweden). Blood samples for the Western-blot analysis were taken on the latest day of the second provocation diet (E/P diet) in the morning.

■ The SDS-PAGE procedure and the Western-blot analysis were performed as follows. After removing

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