

Can exposure to latex cause adhesion formation?

THE prevalence and severity of latex allergy have increased markedly in the last decade, especially in health-care workers, due to increasing exposure to natural rubber products. Clinical symptoms vary from contact eczema to various allergic phenomena, including even life-threatening anaphylaxis (1). We tested whether latex allergy might also lead to enhanced adhesion formation.

Postoperative adhesions are abnormal unions between peritoneal surfaces which occur after almost every intra-abdominal surgical operation and can lead to a number of complications. It is known that starch powder on surgical gloves contributes to the process of adhesion formation (2). Recently, it was realized that glove powder functions as a carrier

of latex allergens (3). The present study aimed to test the hypothesis that the peritoneum may become sensitized to latex, and, upon rechallenge via contact with surgical gloves, may respond with pronounced adhesion formation.

This was tested by immunizing rats with glove eluates, followed by intraperitoneal challenge with latex and scoring of adhesion formation.

Female inbred Wistar rats weighing about 250 g were used (HSD, Zeist, The Netherlands). The experimental protocol was approved by the Ethical Committee on Experimental Animal Research of Erasmus University. Rats were immunized three times at 2-week intervals with lavage fluid of surgical gloves. Two powdered latex gloves (Baxter, type 2D7255I) were cut into strips of 1 × 10 cm and washed for 1 h in 500 ml phosphate-buffered saline (PBS) by gentle stirring. The solution was decanted, divided into 10-ml aliquots, and stored at -20°C. A sample was lyophilized and weighed to determine the amount of substance in the lavage solution (1.5 mg/ml; 0.6% protein). The rats were immunized subcutaneously, as well as intraperitoneally, with 0.5 ml of lavage solution per site. Similar immunizations were repeated after 14 and 28 days. Sham immunizations were performed with PBS only. Fourteen days after the last immunization, the adhesion experiment was started,

Once sensitized by latex exposure, the peritoneum may respond to re-exposure with enhanced adhesion formation.

Table 1. Immunization with eluates from powdered surgical gloves provokes enhanced adhesion formation upon challenge with latex

Immunization	Challenge	Adhesion	
		Mean ± SEM	Range (median)
1) Eluate	Starch	41 ± 5%	12.5–68.75% (50%)
2) PBS	Starch	47 ± 6%	25.0–62.5% (34.25%)
3) Eluate	Latex	51 ± 6%	37.5–81.25% (50%)
4) PBS	Latex	24 ± 9%	6.25–62.5% (9.25%)

Eluate: washing fluid from powdered surgical gloves immersed for 1 h in PBS (phosphate-buffered physiologic saline). Rats were immunized three times with 1 ml of lavage fluid that contained 1.5 mg of glove substance; 14 days after last immunization, uterus horns were smeared with either starch or latex to provoke adhesions. Starch: cornstarch. Latex: freeze-dried ammoniated crude latex sap. Adhesion scores are given in percentage (mean, range, and median). Group 4 vs 2 and 3: $P < 0.05$.

the uterine horn adhesion model being used, as described previously (4). Briefly, the two uterus horns were sutured to the lateral abdominal peritoneum by three 5/0 Surgilene (Braun, Melsungen, Germany) sutures positioned at 0.7-cm intervals. Subsequently, using cotton tips, both uterus horns were smeared with either about 1 mg cornstarch (Sigma) or 1 mg freeze-dried latex sap. Lyophilized latex (12% protein) was produced from natural ammoniated latex sap (obtained from Regent, UK). Fourteen days after the operation, the rats were killed, and adhesion formation was assessed by scoring the presence of adhesions, as described earlier (4). For each rat, the average score of two uterus horns was taken. Each experimental group contained eight animals. Statistical analysis was done with the Kruskal-Wallis one-way ANOVA, followed by the Mann-Whitney-U test.

No visible peritoneal reactions were observed when immunized animals were challenged with freeze-dried latex. There also were no differences in the strength or texture of the adhesions among the experimental groups. The mean adhesion scores obtained in the different groups are summarized in Table 1. Animals that were immunized or sham-immunized responded similarly to a local challenge with starch, leading to mean adhesion percentages of 47 and 41, respectively. However, immunized animals showed statistically significant higher adhesion formation than sham-immunized controls, when challenged with latex (mean adhesion scores: 51% and 24%; $P < 0.05$). In addition, adhesion formation provoked by latex in sham-immunized rats was significantly lower than adhesion formation in the starch group (24% vs 47%; $P < 0.05$), suggesting that latex per se had no direct inflammatory effect on the peritoneum.

Our hypothesis was that the peritoneum might be a participant in latex allergy and that exposure of an "allergic peritoneum" to latex gloves might

add to adhesion formation. Adhesion formation provoked by glove powder might then proceed along two pathways:

- 1) direct peritoneal irritation by starch
- 2) an immunologic reaction of the peritoneum to latex antigens occurring in a subgroup of patients sensitized to latex.

The present results suggest that the peritoneum can indeed be sensitized to latex antigens, and, upon challenge, can respond with increased adhesion formation. This notion may provide a new interpretation of earlier findings by Grant et al. (5) that experimental peritonitis may be provoked in guinea pigs by immunization with glove powder. Unknown components in glove powder were held responsible for the observed delayed hypersensitivity. We now believe that these components are latex proteins. If our observations can be confirmed clinically, increased risk of adhesion formation is another reason why surgical patients identified as sensitized to latex should not be exposed to latex gloves.

Key words: adhesion formation; latex allergy; rats; surgical gloves.

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Food allergy or scombrototoxin poisoning?

VARIOUS mechanisms, toxic, parasitic, or infectious, may simulate allergy symptoms.

At the end of dinner in a restaurant, Mr S., aged 59 years, with a 20-year history of grass-pollen allergy, developed facial erythema, which rapidly extended to the neck and upper part of the thorax, accompanied by a feeling of hotness, pruritus, headache, and, in the next hour, malaise with an impression of rigor, and a blood pressure fall to 9/6 needing emergency treatment, but there was no diarrhea or bronchospasm. The symptoms gradually subsided over 4-5 h.

The patient was referred to our allergy center 3 weeks later with a diagnosis of food allergy. The patient interview did not identify other comparable previous episodes but showed that the dinner had consisted of fish soup containing sea bream, angler, sea perch, salmon, bay leaf, salt, pepper, tomatoes, and rapeseed oil; mayonnaise containing ketchup and garlic; and a slice of tuna with Greek rosé wine.

Prick tests with the commercial battery were negative (crab, prawn, lobster, spiny lobster, whiting, cod, hake, freshwater fish, sardine, tuna, and garlic). Slight positive results were recorded for hops, almond, and hazelnut. No clinical symptom was found retrospectively for those substances, and the

patient ate them without any problems. Under inpatient monitoring, tests consisting of intake of rosé wine and fish soup were conducted and were negative. Testing for IgE was positive for *Dactylis*, but negative

*This probably
histamine-induced
shock is often confused
with food allergy.*

for cod, tuna, mackerel, and garlic.

Further inquiries showed that the slice of tuna served to the patient was of an unusual color and texture, although the taste seemed normal. It would appear that the tuna had been thawed the day before use. Since then, the patient had eaten tuna with no reaction.

A diagnosis of scombrototoxin poisoning was finally adopted.

This disease is frequently unrecognized and confused with allergy to fish (1). In fact, it is poisoning related to the presence in tuna and other fish of a toxin known as scombrototoxin after the scombroid family of fish, particularly tuna and mackerel. Other species of fish have since been considered responsible (dolphin, herring, pilchard, anchovy, sardine, salmon, bluefish) (2, 3). In the USA, 30-150 cases were reported each year to the Centers for Disease Control between 1978 and 1981, and there was a major epidemic in 1974, involving 232 people, caused by canned tuna (4).

The symptoms generally develop in the first minutes or 2 h after ingestion of the contaminated

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