desensitizing treatment, performed subcutaneously with an aqueous latex extract in a patient with allergy to NRL, resulted in serious side-effects (anaphylactic reaction requiring the use of epinephrine) (2).

Therefore, we employed an alternative method of desensitization in a 23-year-old female medical student. She had a history of local erythematous papular rash, asthma, and rhinitis when wearing latex gloves.

A complete allergologic evaluation (including specific skin prick and patch tests; measurement of specific IgE; and contact, mucous, and sublingual challenge tests) was carried out. Skin prick tests with a 1-cm^2 piece of surgical latex glove material (prick by prick method) and a standard latex skin test reagent (0.5 mg/ml; ALK Abelló, Madrid, Spain), and specific anti-NRL IgE immunoassay (Pharmacia UniCAP, Uppsala, Sweden) were positive.

The patch tests using the standard latex preparation of the prick tests and a 1-cm^2 piece of surgical latex material were negative.

To confirm the diagnosis of allergy to latex, we performed specific challenge tests. The contact glove-challenge test was performed with the patient wearing a latex glove on a hand until symptoms appeared, the mucous challenge was performed with the patient holding a latex glove finger in her mouth until the occurrence of symptoms, and the sublingual challenge was performed by putting under the patient's tongue increasing doses of latex for 3 min every 20 min until symptoms appeared. The contact glove-challenge test (rhinitis, asthma, and local erythematous papular rash with pruritus after 20 min), the mucous challenge test (local erythema with some itchy wheals and itching of throat after 10 min), and the sublingual challenge test (submaxillary erythema with pruritus, nausea, and dysphagia after 20 min with one drop of a solution containing 25 μg of latex diluted 1:10) were all positive. These findings are consistent with the occurrence of IgE-mediated allergy to latex.

We decided to attempt a rush sublingual desensitization to NRL. In this protocol, the patient put under her tongue increasing doses of latex extract (ALK Abelló) for 3 min every 20 min and then spit them out.

After obtaining informed consent, the desensitization was begun with an initial dose of a solution containing 25 μg of latex diluted 1:10. On day 1, the patient received a cumulative dose of 28×10^{-10} μg of latex, on day 2 she received 2.8 μg, and on day 3 the desensitization protocol was successfully completed, reaching the cumulative dose of 500 μg of latex with no side-effects.

After the rush desensitization, the contact and the mucous challenges were negative: the patient could wear latex gloves for 6 h and hold a latex glove-finger in her mouth for 1 h without symptoms. Desensitization was maintained with five drops of a solution containing 500 μg/ml of latex once a day.

Although this study has been carried out only in one patient, it provides evidence that a successful therapeutic approach to latex allergy is possible with a sublingual desensitizing treatment. However, further studies are necessary to confirm these results.

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Accepted for publication 21 August 2000
Allergy 2001: 56:86–87
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ISSN 0105-4538

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Wasp-sting-triggered LE

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Key words: apoptosis; isomorphic reaction; Köbner's phenomenon; subacute cutaneous lupus erythematosus; wasp sting.

• Lupus erythematosus (LE) is well known to be induced by several stimulating events such as exposure to sunlight, infectious and noninfectious inflammatory diseases including appendicitis (1), treatment with various drugs (e.g., procainamide, carbamacepine, chlorpromacine), and pregnancy. Surprisingly, Köbner's phenomenon has been only rarely described in patients with LE (2). Here we report on a female patient with subacute cutaneous lupus erythematosus (-SCLE) caused by an insect sting. A 79-year-old Caucasian woman with a 2-year history of SCLE presented with a purple-red, scaly plaque of approximately 10 cm in diameter on the back of the right hand (Fig. 1). She told us that she had been stung by a wasp 2 months before and had initially developed an edematous swelling in the mentioned area. Wasp-sting köbnerization of the underlying SCLE was suspected. To exclude induction of the LE by contact sensitization to one or more allergens of applied creams, she was patch tested with the standard series of the International Contact Dermatitis Research Group (ICDRG), an ointment series, and the topical treatments used. We did not detect any contact sensitization. A skin biopsy (right hand) showed orthokeratosis covering a hydropic degenerated epidermis in which lymphocytes were visible, pigment incontinence, discrete extravasation of erythrocytes, colloid bodies, and splitting of

First case of subacute cutaneous LE showing köbnerization after sting.

Figure 1. Dorsal aspect of right hand 2 months after wasp sting shows erythematosquamous plaque and erythematous papules.

the PAS-positive basement membrane. Total IgE was 110 kU/l, IgE against bee venom was 4.18 kU/l, and that against wasp venom was 10.0 kU/l. Because she did not suffer LE eruptions after other traumatic events such as nettle stings, it was suspected that an IgE-mediated allergic reaction had induced the SCLE skin lesions.

Köbner's phenomenon, or isomorphic reaction, first described by Heinrich Köbner in 1876, is defined as the development of skin lesions in previously normal skin after traumatization by either internal or external influences. It is well known in patients with psoriasis and has been reported in several other skin diseases such as lichen planus, vitiligo, bullous pemphigoid, etc. (3). In patients with LE, it has been only rarely described (2). Contact dermatitis, scratching effects, pressure from sock tops, exposure to liquid or silica, tattoos, operation scars, striae cutis distensae, thermal injury, and other traumatic events have been reported to induce LE (2). Our case is the first report of a wasp sting inducing SCLE, and it is of special interest because, in contrast to the cases reported so far, LE exacerbation could be observed not only within the irritated skin area but also elsewhere on the skin far away from this site. It was suspected that it was not the mechanical sting but an IgE-mediated inflammatory reaction that had triggered LE. Because autoantibodies are uncommon in LE triggered by köbnerization (2), it is reasonable to assume that such patients may have increased susceptibility to minimal traumatic events and could react with increased apoptosis of epidermal cells. Apoptotic keratinocytes are a hallmark of LE skin lesions (4). Moreover, lupus antigens, normally hidden within cells, are present in small surface blebs of cells undergoing apoptosis. In summary, the presented case demonstrates that Köbner's phenomenon seems to be a relevant mechanism in the pathogenesis of LE. Therefore, patients with LE should be asked about external and/or internal triggering events.

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Accepted for publication 5 September, 2000
Allergy 2001: 56:87–88
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ISSN 0105-4538

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