120-kDa LAD-1 antigen, as reported previously [1]. However, all the results of these immunoblotting studies were negative for both IgG and IgA. The result of IgG ELISA, using the noncollageneous 16a (NC16a) domain of 180-kDa bullous pemphigoid antigen, was negative. The diagnosis of LAD was established and we started therapy with the topical application of clobetasol propionate-contaning ointment daily. Within a week the itching of the skin disappeared and no new vesicles developed. In the following 4 months no recurrence was noted.

Several LAD-specific autoantigens have been reported so far. These proteins are 97-kDa [2], 285-kDa [3], 180-kDa, 230-kDa [4] and 255-kDa [5] antigens. It has been suggested that the 97-kDa LAD antigen is a processed form of the larger 120-kDa protein produced by cultured keratinocytes [6]. This 97-kDa LAD antigen is localized on anchoring filaments of normal human skin. In addition, this 97-kDa LAD antigen is shown to be a part of the extracellular domain of the 180-kDa bullous pephigoid antigen 2 [7]. Unfortunately, an immunoblot analysis using patient serum could not detect any antigens. This might be the reflection of low titer of the antibody in the patient serum. This low titer might be related to the mild clinical features in our case. Because the direct immunofluorescence showed very broad linear deposition with a slight granular deposition, this case might be considered to be concurrence of LAD and classical dermatitis herpetiformis. This may explain the negative results in both indirect immunofluorescence and immunoblotting studies. LAD was originally thought to be a subtype of dermatitis herpetiformis, but is now clear that LAD is an entirely different disease with no association to gluten-sensitive enteropathy.

LAD symptoms vary from mild pruritus to severe pruritus and burning sensation. The skin lesions comprise urticarial erythematous plaques, papules, vesicles and blisters. The blisters can arise from normal appearing skin or urticarial erytematous plaques and become erosive and hemorrhagic. The erythematous plaques are characterized by slow enlargement and migration. Our case is characterized by clinical features similar to contact dermatitis (dermatitis linearis). In spite of unique clinical features, our case is histologically and immunologically consistent with LAD. ■

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Occupational allergic contact dermatitis to cresyl glycidyl ether (CGE)

Due to their unique properties, such as high mechanical, chemical and thermal resistance, epoxy resin systems (ERS) are widely used in many trades and industrial fields and are a common cause of occupational allergic contact dermatitis. Sensitisation occurs not only to the resins, but also to hardeners and reactive diluents [1]. Population-based data on the epidemiology of ERS-sensitisation are scarce, but approximately 0.6%-1.1% of the general population are estimated to be affected. Men are sensitised more often than women [3, 4] probably related to their more frequent occupational exposure. However, only a fraction of the ERS components currently used in industrial applications are available for patch testing.

We report a 40-year-old male bricklayer and tile setter, seen in our inpatient department for the treatment of a severe spreading allergic contact dermatitis. There was no history of atopy, skin or internal diseases. He reported he had had a work accident one week previously: he contaminated his left upper leg with a 2-component adhesive while trying to open it. Two days later he developed dermatitis, not only on the affected upper leg but also on his hands. After therapy with topical steroids had been unsuccessful and his skin changes were spreading to affect the whole body, he was referred to our inpatient department for further diagnostics and therapy.

On his left upper leg and on his chest and abdomen there was an approximately 40×15 cm large erythema, with papulovesicles. Scattering eczematous lesions were found on the whole body *(figures 1A and B)*. Clinical examination revealed a good general status and a normal clinical examination; no other cutaneous abnormality or lymphadenopathy was found.

The patient received oral methylprednisolone (initially at a dose of 120 mg per day) and improved slowly within two weeks *(figures 1C, D)*. Tacrolimus 0.1% was used topically.

The qualitative analysis of the patient's occupationally used 2-component-adhesive revealed the presence of the putative allergens, epoxy resin, bisphenol A, the reactive diluent cresyl glycidyl ether, the hardeners isophoron diamine and m-xylylen diamine and benzyl alcohol as preservative.

A few months later the patient was patch tested with the German Contact Dermatitis Society series which showed

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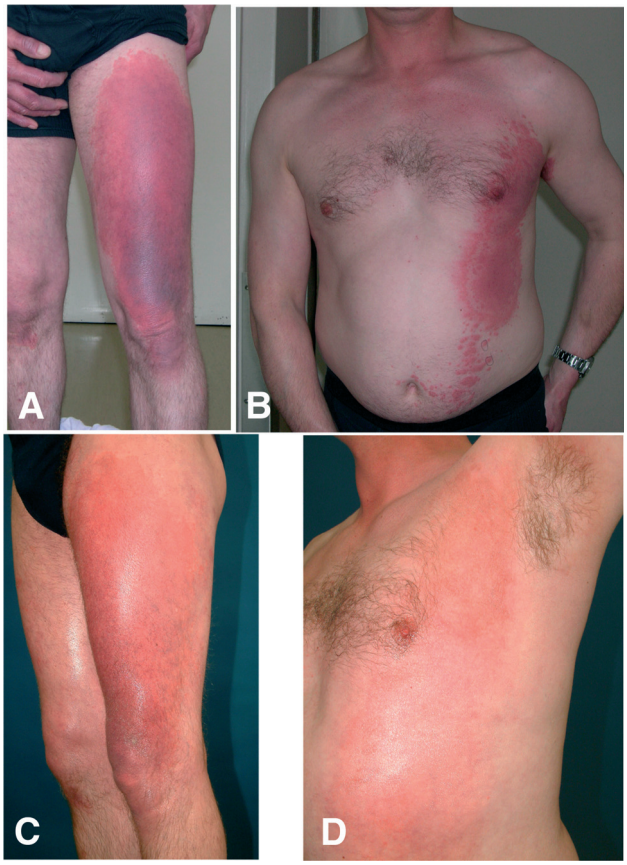

Figure 1. A, B) Acute eczematous lesions with papulovesicles on the contaminated left upper leg and on the chest and abdomen. Scattering eczematous lesions on the whole body. **C, D)** skin status at discharge from the inpatient department

positive reactions to epoxy resin based on bisphenol A (DGEBA-R), 4,4`-diaminodiphenylmethane, butyl glycidyl ether, cresyl glycidyl ether and phenyl glycidyl ether *(table 1)*.
The 2 basic components of an ERS are the resin and a hardener. Diglycidyl ether of bisphenol A is the monomer and the most important contact allergen in epoxy resins based on bisphenol A (DGEBA-R). Contact allergy to epoxy resins of the bisphenol F type (DGEBF-R) has been shown to be at least as common as contact allergy to DGEBA-R, but shows a high cross-reactivity [2]. Sensitisation occurs not only to the resins but also to hardeners and reactive diluents.
The hardener m-xylylendiamine which elicited positive test reactions in 13.8% in the EPOX 2002 study and therefore has been shown to be a frequent allergen [1] was also included in our patient's 2-component adhesive. However, it is not commercially available for patch testing due to its low stability. Thus, further investigations are necessary to establish a patch test preparation suitable for routine testing.
The reactive diluents phenyl glycidyl ether (11.5%), cresyl glycidyl ether (6.8%) and butyl glycidyl ether (3.4%), which are cross-reactive due to their chemical relation as aromatic glycidyl ethers and are established for patch testing and commercially available, played a minor role as contact allergens in the EPOX 2002 multicenter study [1], but were crucial for our patient.
However, not all components were available for patch testing and therefore maybe not all contact allergies could be detected. Further investigations are necessary to improve diagnostics in suspected contact allergy to ERS, which is important because of the wide occupational use of these substances in many trades and industrial fields. ■

Table 1. Patch test results (components of the patient's occupationally used 2-component-adhesive are underlined).

Patch test results	Test conc.	48 h	72 h
Epoxy resin based on Bisphenol A	1% pet.	+	+
4,4-Diaminodiphenylmethane	0.5% pet.	(+)	+
Butyl glycidyl ether	0.25% pet.	+	++
Cresyl glycidyl ether	0.25% pet.	++	+++
Phenyl glycidyl ether	0.25% pet.	++	+++
Bisphenol A	1% pet.	-	-
Benzyl alcohol	1% pet.	-	-
Isophoron diamine	0.5% pet.	-	-

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Development of multiple basal cell carcinomas in mycosis fungoides treated with oral bexarotene

Bexarotene belongs to a subclass of retinoids called "rexinoids" because of their selective binding to nuclear retinoid X receptors (RXRs) [1]. It inhibits the growth of several tumor cell lines of lung, breast, hematopoietic and squamous epithelial origin [2]. In addition, topical bexarotene proved effective for the treatment of basal cell carcinoma (BCC) [3].
A 60 year old Caucasian woman presented with a 20-year history of plaque stage mycosis fungoides (MF). Prior treatments included topical corticosteroids, a treatment cycle with intravenous gemcitabine (1,200 mg/m^2), a PUVA treatment cycle (total UVA dose: 80 J/cm^2) in 1989, two treatment cycles with total skin electron beam therapy (total dose: 24 Gray) in 1998 and 1999, and 6 treatment cycles with narrow-band UVB (total UVB