

CONTACT POINTS

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Latex, potato and tomato allergy in a restaurateur

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Key words: cross-sensitivity; food handlers; natural rubber latex allergy; occupational; potato; tomato.

A 39-year-old atopic woman presented with 2-year history of fingertip dermatitis. She owned and worked in a fish-and-chip and pizza restaurant. She gave a history of reacting to costume jewellery.

Patch testing to the European standard series showed positive reactions to nickel, cobalt and fragrance. Despite allergen avoidance, hand care and treatment with clobetasol propionate ointment, her dermatitis did not improve. At subsequent review, she complained that rubber gloves irritated her hands and that handling uncooked potatoes and tomatoes aggravated her dermatitis. In addition, she had noted that eating uncooked tomatoes caused oral tingling and facial erythema, though eating cooked tomatoes and potatoes had no adverse effects. RAST showed moderate IgE levels to natural rubber latex, tomato and potato. To avoid contact with potatoes, tomatoes and latex, she changed her job to retailing kitchenware and her hand dermatitis improved considerably.

It is well known that patients with latex allergy may have cross-reactions to foodstuffs such as kiwi fruit, banana, passionfruit, avocado and chestnut (1). Cross-reactions to potato and tomato have been recognized more recently (2, 3). Tomatoes and potatoes belong to the family Solanaceae and both contain the

protein patatin, which shows 60% homology with a latex protein allergen (3). Patatin has been identified as an IgE-binding protein and an allergen in children with positive prick tests to potato (4). Local sensitivity reactions to raw potato have previously been reported in patients who do not react to cooked potatoes, due to heat denaturing the allergenic proteins (5). As our patient had worked with potatoes and tomatoes for years before she ever started to wear rubber gloves it may be that her allergy to latex arose secondarily via primary sensitization to potato or tomato.

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Evaluation of cross-reactivity between budesonide and desonide

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Key words: budesonide; contact allergy; cross-sensitization; desonide, medicaments; topical corticosteroids.

Coopman et al. (1) classified topical corticosteroids into 4 cross-reacting groups, though this has been challenged (2). Desonide is chemically related to budesonide and, in the same Coopman et al. group, contact sensitization to desonide has been sporadically described (3–7).

Materials and Methods

After informed consent, 20 patients with allergic contact dermatitis from budesonide were patch tested with desonide (Galderma R & D, Sophia-Antipolis, France), 0.1% and 1% pet., using Al-tests (Imeco AB, Södertälje, Sweden). Readings were made at day 2 (D2), D4 and D7. All patients had previously been tested with the SIDAPA standard series, augmented with further allergens, including budesonide 0.1% pet., hydrocortisone 17-butyrate 1% eth. and tixocortol 21-pivalate 0.1% pet.

Results and Comment

The clinical data and patch test results of our patients are summarized in Table 1. Patch testing with desonide was positive (+ +) at D2 and D4 in only one patient, who had repeat-

Table 1. Clinical data and patch test results in 20 patients with contact allergy to budesonide

Patient	Sex	Age (years)	Location	Positive (+ +) patch test results
1	F	24	Hands	Hydrocortisone 17-butyrate
2	F	19	Eyelids; chin	PPD*; diaminodiphenylmethane; Disperse Orange 3; <i>para</i> -toluenediamine
3	F	35	Feet; thighs; submammary, pubic, perianal areas	Hydrocortisone 17-butyrate
4	F	19	Hands	Methylchloroisothiazolinone + methylisothiazolinone
5	F	44	Generalized	Hydrocortisone 17-butyrate; fragrance mix; imidazolidinylurea
6	F	55	Trunk; limbs	Lanolin alcohol
7	M	29	Wrists; retroauricular folds	Fragrance mix; PPD mix
8	F	35	Trunk	Nickel sulfate; neomycin; potassium dichromate
9	F	45	Feet; upper limbs; submammary folds	Hydrocortisone 17-butyrate; Myroxylon Pereirae resin; potassium dichromate; thiuram mix; PPD mix
10	F	36	Limbs	Hydrocortisone 17-butyrate; desonide 0.1% and 1%; lanolin alcohol; fragrance mix
11	F	51	Hands	—
12	M	78	Trunk; thighs	Hydrocortisone 17-butyrate
13	M	46	Upper limbs	PPD mix; PPD; epoxy resin
14	M	48	Feet	PPD mix; PPD; Disperse Blue 124; Disperse Red 1; Disperse Orange 3; Myroxylon Pereirae resin; neomycin
15	F	45	Hands	Myroxylon Pereirae resin; lanolin alcohol
16	F	56	Eyelids	Myroxylon Pereirae resin; lanolin alcohol; diaminodiphenylmethane
17	M	59	Feet; hands; limbs; face	Mercaptobenzothiazole
18	F	38	Face; back	Nickel sulfate; fragrance mix; PPD; benzocaine
19	F	53	Abdomen	Myroxylon Pereirae resin; lanolin alcohol; fragrance mix; paraben mix
20	M	48	Limbs	Hydrocortisone 17-butyrate

*PPD, *para*-phenylenediamine.

edly used several topical corticosteroids, including desonide, suggesting concomitant sensitization rather than cross-sensitization.

Among topical corticosteroids, budesonide is one of the most frequent sensitizers (8). Our preliminary findings suggest a lack of cross-reactivity between it and desonide, despite of their close chemical similarity.

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Father and child with milk allergy and positive reactions to latex gloves on prick and use testing

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Key words: anaphylaxis; casein; glove; latex; milk allergy; prick test.

Casein is the main component of cow's milk proteins, and it is also one of the major allergens of milk allergy. It is added to many kinds of food-stuffs, dairy products and medical supplies because of a high emulsification and water-holding capacity (1).

Case Reports

Case no. 1

A 42-year-old man with no previous history of food allergy or exposure

Table 1. Results of prick test and CAP-RAST of case no. 1

Prick test	Score	CAP-RAST	UAmL ⁻¹ (class)
milk (as is)	4 +	milk	36.6 (4)
yoghurt (as is)	4 +	cheese	38.1 (4)
cheese (as is)	4 +	casein	39.1 (4)
calpis (as is)	4 +	latex	<0.34 (0)
sausage (as is)	4 +	α -lactalbumin	0.52 (1)
α -casein (100 μ g mL ⁻¹)	4 +	β -lactglobulin	<0.34 (0)
β -casein (100 μ g mL ⁻¹)	4 +		
κ -casein (100 μ g mL ⁻¹)	3 +		
latex glove*	2 +		
nonammoniated latex 1 mg mL ⁻¹	(-)		

*Dunlop examination glove.

Table 2. Results of prick test and use test of case nos. 1 and 2

	Prick test		Use test	
	Case no. 1	Case no. 2	Case no. 1	Case no. 2
Gammex (no casein)	(-)	(-)	(-)	(-)
Triflex (contains casein)	2 +	1 +	(+)*	(+)**
Dunlop (contains casein)	2 +	2 +		

*Erythema and itching. **Itching.

Table 3. Results of prick test and CAP-RAST of case no. 2

Prick test	Score	CAP-RAST	UAmL ⁻¹ (class)
milk*	4 +	milk	90.8 (5)
cheese*	4 +	cheese	97.8 (5)
sausage (as is)	4 +	casein	96.7 (5)
powdered skimmed milk*	4 +	α -lactalbumin	<0.34 (0)
α -casein (100 μ g mL ⁻¹)	4 +	β -lactglobulin	1.75 (2)
β -casein(100 μ g mL ⁻¹)	2 +		
κ -casein(100 μ g mL ⁻¹)	1 +		

*Allergen scratch extract (Torii®)

to cow's milk had developed, when 39 years old, several anaphylactic reactions such as generalized urticaria, dyspnoea, nausea and diarrhoea after eating various milk products such as yoghurt, cheese, Calpis (concentrated lactic acid drink) and some sausages. Prick tests with regard to all of these products were all 4 + positive (2). The prick test reactions to α -, β - and κ -casein were also positive: 4 +, 4 + and 3 +, respectively (Table 1). He was eventually diagnosed as having casein anaphylaxis. He also developed a 2 + positive prick test reaction to Dunlop latex glove extract (3) containing casein. However, the patient responded with a negative reaction to non-ammoniated latex (NAL). Triflex glove extract, which contained casein, elicited a 2 + positive reaction, but Gammex glove extract, which did not contain casein, elicited a negative

reaction (Table 2). A use test was performed by applying a Gammex glove to one wet hand, and a Triflex glove to the other wet hand for 1 h. On the Triflex hand, erythema and itching occurred, but there was no change on the Gammex hand. These results indicated that the patient reacted to the casein included in the natural rubber latex (NRL) glove, and not to the latex itself. We asked three particular manufactures of NRL gloves whether NRL gloves contained casein. Gammex (Ansell) does not contain casein, but Dunlop (Dunlop) and Triflex (Allegianse) both contain latex (1).

Case no. 2

A 4-year-old boy, the son of case no. 1, when 3-years and 4-months old, developed diarrhoea and a cold sweat appearing about 10 min after

eating a hamburger, French fries and some fried chicken. He subsequently became pale and then fainted. These symptoms disappeared about 2 h later. Prick test reactions to cow's milk, cheese, sausage and powdered skimmed milk were all 4 + positive (Table 3). These foodstuffs turned out to contain casein. The specific IgE antibody to casein was also detected (96.7 UAmL⁻¹). These results, together with the patient's history, led to the same diagnosis in case no. 2 as in case no. 1. Both Triflex and Dunlop glove extracts containing casein elicited 1 + and 2 + positive reactions, respectively, but Gammex glove extract elicited a negative reaction. Use tests with each finger of Triflex and Gammex glove were then examined for 90 min. Although itching appeared on the Triflex finger, there was no change on the Gammex finger (Table 2).

Discussion

The NRL gloves are widely used for operations and medical examinations. There is a possibility that the NRL gloves containing casein may come into contact with the skin or mucous membrane of people who have casein allergy. We have to consider sufficiently the danger of casein anaphylaxis caused by contact with NRL gloves containing casein. In order to prevent casein anaphylaxis, the packaging for NRL products should clearly indicate if they contain casein. Casein is included not only in foodstuffs and daily products, but also in some medical supplies such as Meiact (oral antibiotic). Therefore, medical workers need to take casein allergy into consideration if there is a possibility the patient may have an allergy to cow's milk, and efforts to prevent casein anaphylaxis need to be made by using medical supplies without casein.

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Contact dermatitis from textile and dye allergens requires a high index of suspicion for diagnosis

A review of 18 cases

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Key words: allergic contact dermatitis; disperse dyes; ethylene urea melamine formaldehyde; occupational; PPD; textile dermatitis; uniform dermatitis.

In this report we describe 18 patients who were positive to dyes and textile chemicals on patch testing, during a 4½-year period.

Patients and Methods

All 18 patients had been patch tested, on grounds of clinical suspicion, to a modified British Contact Dermatitis Group standard series, and a series consisted of 18 dyes and 4 textile chemicals (Table 1), using Finn Chambers on Scanpor tape applied to the upper back for 2 days (D), readings being made at D2 and D4.

Results

Of the 18 patients, 3 (17%) were male and 15 (83%) female. The median age of presentation was 38 years (range 18–71: mean 41, 95% confidence interval 6.7). The duration of history ranged from 1 week to 34 years (median 4 years; mean 7.6 years; 95% CI 4.2). A personal history of atopy (hayfever, asthma or childhood eczema) was present in 13 (72%), with a previous history of eczema found in 11 (including 2 with discoid eczema).

All 18 subjects reacted to at least 1 allergen in the dyes and textile chemicals series, several reacting to more than 1 allergen (Table 1). In the standard series, 5 patients were positive to PPD, 3 to formaldehyde and 3 to fragrance mix. 4 of the 5 PPD-positive subjects also reacted to 1 or

more disperse dyes (the other having formaldehyde as a cosensitivity).

1 man worked in a clothing factory and handled various textiles as part of his occupation. 4 patients, 2 nurses, a checkout operator, and a bingo club worker, had to wear a uniform at work and this may have been implicated in their dermatitis in each case.

In 10 cases, specific garments (in 1 case, shoes) were identified as potentially causative. In 4 cases, this was coloured underwear, including tights or stockings, usually black, dark blue or, in 1 case, brown 'Y-fronts'; in 2 cases, trousers; and in 1 case each, socks, rubber gloves, leggings, blouse (part of a uniform), numerous garments from occupational exposure and shoes. Uniforms were implicated in the 4 patients who wore them; particularly black or dark-blue trousers.

Discussion

Contact sensitivity to textile dyes is not uncommon (1), the frequency in our clinic being similar to previous reports (2, 3), as was the sex distribution (2–6). Only 1 of our patients worked in the textile industry, a similar proportion to that found in an Italian study (5). This 1 patient of ours had dermatitis confined to the hands, which may be an unsuspected localization of textile dye dermatitis (7). 4 of the patients wore a uniform to work, their suspected garments being part of this uniform. These uniforms were of synthetic/cotton mixes and dark-blue, black or lilac in colour. A group of nurses was found to be allergic to an anthraquinoid dye (Vat Green 1) in their navy-blue uniforms in 1971 (1), but we are not aware of any more recent such reports.

As in other studies, the dye with the highest frequency was Disperse Blue 106. Ethylene urea melamine formaldehyde, a resin used in the textile industry as a finishing product to improve fastness in pigment printing and as a binder for permanent-pressed garments, was also relatively frequent. PPD was not a reliable indicator of textile dye allergy (4), being positive in only 5 patients. A high index of suspicion is required to patch test patients with dyes and textile chemicals.

Table 1. Number of positive patch test reactions to the allergens in the dyes and textile chemicals series (several patients had more than 1 reaction)

Dye/textile chemical	% Vehicle	No. of patients reacting
Disperse Blue 106	1% pet.	4
Ethylene urea melamine formaldehyde	5% pet.	4
4-Aminophenol	*1% pet.	3
Disperse Blue 124	1% pet.	3
Disperse Blue 85	1% pet.	3
Disperse Orange 13	1% pet.	3
Disperse Orange 3	*1% pet.	3
Disperse Red 1	*1% pet.	3
Disperse Red 17	*1% pet.	3
Disperse Yellow 3	*1% pet.	3
Disperse Blue 3	*1% pet.	2
Disperse Blue 35	1% pet.	2
Disperse Orange 1	1% pet.	2
Basic Red 46	1% pet.	1
Dimethylol dihydroxyethylene urea	4.5%	1
	aq.	
Disperse Blue 1	*1% pet.	1
Disperse Blue 153	1% pet.	1
Disperse Brown 1	1% pet.	1
Dimethylol propylene urea	1% pet.	0
Disperse Red 11	*1% pet.	0
Disperse Yellow 9	1% pet.	0
Tetramethylol acetylenediurea	5% pet.	0

*Supplied by Trolab, the rest by Chemotechnique.

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Occupational allergic contact dermatitis from falcarinol

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Key words: Araliaceae; carrot; celery; common ivy; cross-sensitivity, *Daucus carota*; didehydrofalcarinol; falcarinol; food handlers; *Hedera helix* L; occupational; parsley; Umbelliferae

Several vegetables commonly used in cooking are members of the Umbelliferae family: carrot (*Daucus carota*), parsley (*Petroselinum hortense*), parsnip (*Pastinaca sativa*) and celery (*Apium graveolens*) (1). Carrots contain pinene, limonene, other terpenes (2) and falcarinol, this last substance also being present in common ivy (*Hedera helix*), a member of the Araliaceae family. Both falcarinol and didehydrofalcarinol have been reported as causing allergic and irritant contact dermatitis (3, 4).

Case Report

A 57-year-old school cook presented with a 5-year history of recurrent dermatitis on her hands and forearms, predominantly the left hand (Fig. 1). She also had episodic facial dermatitis. All lesions were work-related.

Patch tests with our standard series and plants series (including terpenes; Trolab) were negative. Patch tests with samples of the vegetables handled gave the main reactions given in Table 1.



Fig. 1. Erythematous scaly lesions of the fingers and thenar eminence of the left hand.

Table 1 Main reactions of patch tests with samples of vegetables handled

	D2	D4
Carrot (root)	++	++
Parsley (stem)	++	++
Celery (stem)	++	++
Alcoholic extract of carrot 1% pet.	++	++
Acetone extract of carrot 1% pet.	++	++
Alcoholic extract of parsley 1% pet.	++	++
Acetone extract of parsley 1% pet.	++	++
Alcoholic extract of celery 1% pet.	++	++
Acetone extract of celery 1% pet.	++	++

Patch testing was ++ to falcarinol 0.03% pet. but negative to didhydrofcalcarinol 0.3% pet. Patch tests with pieces of common ivy were also ++. With these vegetables, scratch-chamber tests with fresh material were also positive. Ten controls tested with the same materials were negative. RAST results were: carrot, positive class 2; celery, positive class 1; and parsley, positive class 2.

Our patient was advised to avoid handling any members of the Umbelliferae family and the lesions disappeared after 2 weeks.

Discussion

Preparing food carries a high risk of occupational contact dermatitis (5), the commonest vegetables to sensitize being garlic, onion, tomato and carrot in descending order of frequency (6). In contrast, the commonest Type I vegetable sensitizers similarly rank tomato, carrot, cucumber, lettuce and potato (7).

It is possible that some of the celery dermatitis attributed to psoralen phototoxicity (8) may instead have been caused by falcarinol sensitization (3). Falcarinol may also be present in anticellulitic creams and gels, shampoos, hair-lotions, teas and expectorants (4), though in eliciting rather than inducing concentrations.

The cut surface of the carrot root gives the strongest patch test reactions (9). The dermatitis mainly affected our patient's non-dominant hand because the patient held vegetables with this, while cutting them with her dominant hand, and the allergen was able to penetrate domestic rubber gloves (10). Most pa-

tients with positive patch tests to carrot are able to tolerate eating it, though such vegetables eaten raw or cooked can cause cheilitis and perioral dermatitis (9), as well as glossal oedema (11) and anaphylaxis (12). However, this latter allergen could be a thermolabile protein (12) as, when the carrots were boiled, no symptoms appeared.

Cross-sensitization may occur with other members of the Umbelliferae family such as parsley, celery and parsnip (9). A number of Araliaceae family plants, such as common ivy, *Schefflera actinophylla* (umbrella tree) and *Schefflera arboricola* (13), should be also avoided by falcarinol-allergic patients (14).

In patients suspected of contact dermatitis due to any of these vegetables and plants, falcarinol should be considered as a possible allergen and should be added to the patch tests.

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Simultaneous contact sensitivity to hydroxystearic acid and C18-36 acid triglyceride in lip glosses

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Key words: allergic contact dermatitis; C18-36 acid triglyceride; cosmetics; cross-sensitivity; hydroxystearic acid; lip glosses.

A 24-year-old woman was seen in September 2001 with pruritic oedematous erythema and scaling on her lips. After newly using about 30 cosmetics over 6 months, she had developed this within 3 months. A positive patch test reaction was seen to 3 of her lip glosses, as is, at D2, D3 and D7. She was then patch tested with 21 ingredients of these lip glosses. Only hydroxystearic acid and C 18–36 acid triglyceride 10% pet. were positive at D2 and D3 (Table 1). All 3 lip glosses

contained these 2 substances. The patient was further patch tested with stearic acid 0.1%, 1% and 10% pet., stearyl alcohol 0.1%, 1% and 10% pet., glyceryl stearate 0.1%, 1%, 10% and 20% pet., and diisostearyl malate 10% and 30% pet. Only glyceryl stearate 10% and 20% pet. were positive at D3. Patch tests with 10% pet. of hydroxystearic acid and C 18–36 acid triglyceride, 10% stearic acid and stearyl alcohol, 20% pet. of glyceryl stearate, and 30% diisostearyl malate in 6 control subjects were negative at D2 or D3.

Discussion

Stearic acid and glyceryl stearate are rare contact sensitizers, though widely used in cosmetics (1). Stearic acid, an emollient and emulsifier, conforms to the formula $\text{CH}_3(\text{CH}_2)_{16}\text{COOH}$ (1). Hydroxystearic acid has an additional –OH group (Fig. 1) to increase its stability as an emulsifier, especially in cosmetic creams.

Cross-reactions between stearic acid, stearyl alcohol, and glyceryl stearate has been studied, and, due to their similarities in chemical structure, might be expected (2). Our case showed cross-reactivity to stearic acid and glyceryl stearate, suggesting that some structure in stearic acid may be the antigenic determinant.

C18-36 acid triglyceride (Synchrowax HGLC[®]), is the triester of gly-

Table 1. Patch test results

	D2	D3
Lip gloss A as is	+	+
Lip gloss B as is	+	+
Lip gloss C as is	+	+
Hydroxystearic acid 10% pet.	+	+
C18-36 acid triglyceride 10% pet.	+	+
Other ingredients pet.	–	–
Stearic acid		
10% pet.	–	–
1% pet.	–	–
0.1% pet.	–	–
Stearyl alcohol		
10% pet.	–	–
1% pet.	–	–
0.1% pet.	–	–
Glyceryl stearate		
20% pet.	+	+
10% pet.	+	+
1% pet.	–	–
0.1% pet.	–	–
Diisostearyl malate		
30% pet.	–	–
10% pet.	–	–

cerin and C18-36 acid (3) (Fig. 1), and is an alternative to natural wax to provide stable and uniform properties in lipsticks and emulsions. No previous case of contact dermatitis from hydroxystearic acid and/or C18-36 acid triglyceride has been reported.

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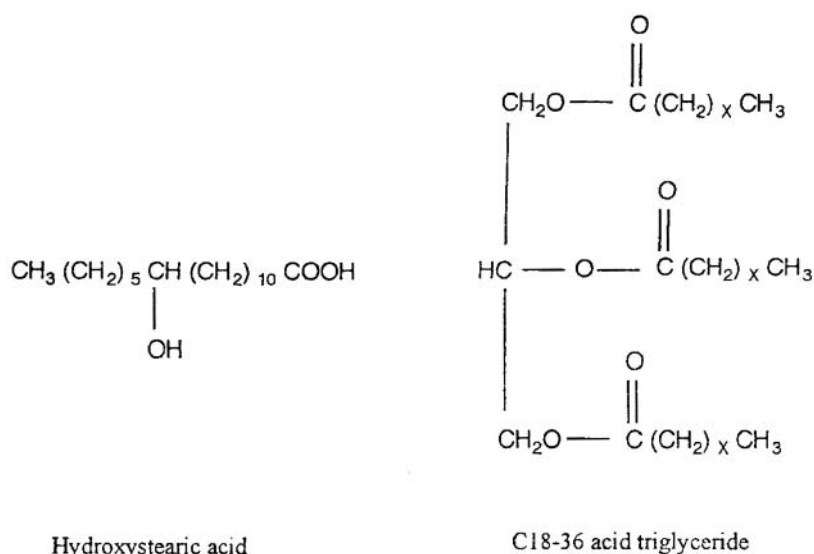


Fig. 1. Chemical structures of hydroxystearic acid and C18-36 acid triglyceride.

Conjunctivitis and bronchial asthma: symptoms of contact allergy to 1,3,5-tris(2-hydroxyethyl)-hexahydrotriazine (Grotan BK)

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Key words: 1, 3, 5-tris(2-hydroxyethyl)-hexahydrotriazine; antimicrobials; asthma; biocides; conjunctivitis; contact dermatitis; formaldehyde releaser; Grotan BK; metalworking fluids; occupational; preservatives.

1,3,5-Tris(2-hydroxyethyl)-hexahydrotriazine (Grotan BK) is mainly used as a bactericide in metalworking fluids (1). It has been reported (2–4) to be a cause of allergic contact hand dermatitis. To our knowledge, this is the first time conjunctivitis associated with bronchial asthma following exposure to this substance has been reported.

Case Report

A 45-year-old man, with no atopic history, was referred by an ophthalmologist because of complaints of red conjunctivae and eczema on the backs of his hands in relation to his work in a company that produced artificial fibers. He had been working there for 6 years, and his complaints had started 2 years before referral. At the same time, he was being investigated for dyspnea, wheezing, and nasal obstruction.

Both his skin and bronchial symptoms appeared after 3–4 h of exposure to biocides, industrial oils,

and an antistatic. He did not wear protective gloves or goggles. After a few days off work the symptoms would clear, and oral corticosteroids had had to be administered only once. His symptoms would recur each time he went back to work. His colleagues had reported no such symptoms.

Clinically, we noted an erythema of his face (no photodistribution) and severe conjunctivitis. His history and clinical signs suggested allergic contact dermatitis.

None of the standard series' allergens gave a positive reaction (Table 1). Patch tests with the biocides, oils, and antistatic showed a strongly positive reaction to hexahydrotriazine, tested in a 1% aqueous solution. He showed a slight reaction to the antistatic, which was thought to be an irritant effect. A formaldehyde radio-allergosorbent test (RAST) was negative.

Discussion

We report a Type IV-allergic reaction to Grotan BK, a formaldehyde releaser used as a biocide in industrial oils. Our patient came in contact with this commercial biocidal product that contains at least 25% 1,3,5-tris(2-hydroxyethyl)-hexahydrotriazine. The formaldehyde patch and RAST were negative. We assume that the liberator itself was the only allergen involved (3). We confirmed an occupational contact allergy with the clinical appearance of hand eczema and conjunctivitis associated with bronchial asthma.

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Allergic contact dermatitis from trideceth-2-carboxamide monoethanolamine (MEA) in a hair dye

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Key words: allergic contact dermatitis; cosmetics; hair dye; trideceth-2-carboxamide monoethanolamine.

Case Report

A 56-year-old woman presented with an itchy eczema of the scalp 3 days after application of a hair dye. The same hair dye (Excellence crème 8.34 Golden Copper Blonde by L'Oréal® Paris, France) had been used by the patient for several years without any complications. The dermatitis par-

Table 1

	D2	D4
Standard series	–	–
Antistatic as semiopen test	? +	? +
1,3,5-tris(2-hydroxyethyl)-hexahydrotriazine (1% aq.)	+	++
RAST formaldehyde: negative		

tially settled with a potent topical corticosteroid, but she continued to have a less-inflammatory scaly scalp consistent with psoriasis. This appeared to be a köbner phenomenon following contact dermatitis. There was no family history of psoriasis. She had no other features of psoriasis prior to the scalp dermatitis and none subsequently. Her scalp psoriasis has persisted despite treatment.

Patch testing to the European standard series, a hairdressing series (Trolab, Hernal, Reinbek, Germany) and to the 3 components of the hair dye (crème colourant, crème developer and after-colour conditioner), in 10% and 50% pet., was performed. No reaction was seen to para-phenylenediamine (PPD) or related compounds. Patch testing to the crème developer was positive at D2 (++) and D4 (+++) to both 10% and 50% pet. Patch testing was performed to the full ingredients of the hair dye kindly provided by the manufacturer. At D2 and D4 there was a +++ reaction to trideceth-2-carboxamide MEA (0.5% aq.) – a constituent of the crème developer.

The reaction at D2 and persisting to D4 would be suggestive of an allergic contact dermatitis; however, an irritant reaction could not be fully excluded. We therefore decided to assess the irritancy of 0.5% aqueous trideceth-2-carboxamide MEA. All patients referred to patch testing in a 3-week period were patch tested to the European standard series (Trolab) in addition to 0.5% aqueous trideceth-2-carboxamide MEA, kindly provided by the manufacturer. All 14 patients had a negative reaction at D2 and D4 to 0.5% aqueous trideceth-2-carboxamide MEA, suggesting that this compound has low irritancy under patch testing conditions.

Discussion

Contact dermatitis from hair dyes is frequently due to PPD or related aminobenzene compounds and from hair bleaches to ammonium salts (1). Contact dermatitis from other intermediary components of hair dye has previously been reported (2). Dyeing the hair to achieve the desired colour can be a complicated process. Excellence crème takes advantage of 2 separate reactions. Firstly, the hair is

died with PPD, then it is lightened to the required colour with hydrogen peroxide. The colourant and developer are mixed together and used immediately to carry out this 2-step process almost simultaneously. An open application test to the hair dye is recommended in the product information leaflet, but it only applies to the colourant because the developer will oxidize once opened. Trideceth-2-carboxamide MEA helps stabilize the viscosity of the mixture of the colourant and developer. Allergic contact dermatitis to monoethanolamine has been reported; however, to our knowledge this is the first case report of allergy to trideceth-2-carboxamide MEA or to any carboxamide.

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Contact leukoderma secondary to occupational toluenediamine sulfate exposure

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Key words: contact leukoderma; cross-sensitivity; hair dye; occupational; toluidinediamine sulfate.

Case Report

A 38-year-old white man presented with a 6-month history of eczema, initially around the neck and wrists, and then more widespread. There was no personal or family history of atopy.

He worked in a company which manufactured hair dyes. 8 months prior to the onset of the rash, he changed his job within the firm and packed toluidinediamine sulfate (TDS) powder, which is used as a hair dye. He wore latex gloves under blue nitrile gloves, a paper overall and a dust mask for this work. However, the environment was dusty and his skin was regularly contaminated. This was usually so severe that, at the end of his shift, he always took a shower before leaving work.

The rash improved greatly with topical corticosteroids and he was patch tested to the European standard series, a hairdressing series and also 5% and 1% aq. dilutions of the patient's own TDS sample. On D2, he had positive (+) reactions to para-phenylenediamine (PPD) and TDS, which remained on D4 (+). There was a slight reaction (?+) to the 5% aq. dilution of toluidinediamine sulfate at D2, and at D4 there was a positive reaction (+) to the 5% aq. dilution and also a slight reaction (?+) to the 1% aq. dilution.

Following the patch tests, his employer changed his work environment and duties, and the eczematous rash settled over a period of 6 weeks. However, he was left with hypopigmented patches of skin within the areas in which the rash developed. There was no depigmentation at the positive patch test sites. His auto-antibody screen and antinuclear antibody were negative.

Discussion

Contact leukoderma can occur secondarily to a preceding allergic contact dermatitis or from the toxic results of depigmenting chemicals such as monobenzyl ether of hydroquinone (1), phenols (2) and catechols (3). Leukoderma following al-

lergic contact dermatitis can be similar to vitiligo both clinically and histologically. If there is gradual coalescence of hypopigmented macules, without perifollicular sparing, contact leukoderma is more likely (4). Depigmentation at the patch test sites of patients with leukoderma secondary to allergic contact dermatitis is not necessary to make the diagnosis (5).

Contact dermatitis from hair dyes is well recognized. Toluenediamine sulfate is a derivative of para-phenylenediamine that is found in hair dyes, where it can cause allergic contact dermatitis. Among 809 hairdressers patch tested to a hairdresser's series, 8% were positive to TDS and 15% to PPD (6).

Contact leukoderma occurring following contact with hair dyes is rare. Taylor et al. (4) reported 4 patients with contact leukoderma in areas where hair dye was applied: 3 were patch tested and 2 were positive to PPD. Progressive depigmentation above the upper lip (7) followed the application of a moustache colouring solution, but with no associated dermatitis, and patch tests to both PPD and the product were negative. A patient (8) applied a hair dye and developed contact dermatitis from PPD, the confirmatory patch test subsequently depigmenting.

Our patient developed leukoderma secondary to allergic contact dermatitis from occupational tolunedi-amine sulfate exposure. Contact leukoderma secondary to tolunedi-amine sulfate has not previously been reported, and hairdressers and their clients need to be aware of this potential side-effect.

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Photosensitivity induced by paroxetine

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Key words: cutaneous adverse drug reaction; paroxetine; photoallergic contact dermatitis.

Case Report

A 59-year-old woman, with no personal or family history of atopy, presented with dermatitis in photoexposed areas. 2 years previously, she had been diagnosed with mixed connective tissue disease and, after receiving chloroquine for 3 months, had shown sustained improvement in systemic disease. Subsequently, however, she developed depression, which was successfully treated with paroxetine, a selective serotonin reuptake inhibitor. For the past year she had been receiving paroxetine 20 mg/day and omeprazole (a proton pump inhibitor) 20 mg/day with good clinical response. In July 2001 she developed itching mainly in sun-ex-

posed sites which did not clear with oral antihistamines. 3 weeks later, an itchy, vesicular, exudative eruption appeared in photoexposed areas (face, neck, V of neck, forearms and hands), sparing the rest of the skin. All drugs were stopped and patch tests and photopatch tests (10 J/cm) were performed with the following results.

True test was negative. Photopatch testing with a standard series plus omeprazole 1% aq., 5% aq., and 10% aq., and paroxetine 1% aq., 5% aq., 10% aq., showed positive reactions at D4 on irradiated sites only to paroxetine 1% aq. (+), paroxetine 5% aq. (++) and paroxetine 10% aq. (++). Photopatch tests with all 3 dilutions of paroxetine in 20 controls were negative.

Discussion

Drug-induced photosensitivity (1) is more frequent in southern Europe because of greater sun exposure (2). Nevertheless, considering the daily use of such drugs, we would agree with Darvay et al. (3) that photoallergic reactions are unusual.

Paroxetine has previously been described as causing cutaneous vasculitis (4), alopecia (5), spontaneous ecchymosis (6), angioedema (7) and polyserositis (8). To the best of our knowledge, paroxetine has never previously been reported as a photosensitizer, though an earlier publication (9) described clinical suspicion unsupported by photopatch testing.

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Acute fingertip dermatitis from a temporary tattoo and quantitative chemical analysis of the product

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Key words: allergy; fingertip dermatitis; para-phenylenediamine (PPD); temporary tattoos.

Patch testing of patients with skin reactions to temporary tattoos shows a positive reaction to para-phenylenediamine (PPD) (1–5).

Case Report

A 24-year-old otherwise healthy man was referred with a vesiculobullous reaction on his fingertips (Fig. 1). Four years before, he had had a temporary tattoo on the right side of the neck. Following this, he had noticed reactions on his face related to black hair colouring. He tolerated bleaching of his hair. Two months before the presenting episode, he had had a temporary tattoo on his left forearm without any reaction. Four days before the current eruption, he had used his fingertips to paint a friend with temporary tattoo paint bought in Thailand. Two days later, he had developed painful vesiculobullous eruptions on the fingertips which had been exposed to the temporary tattoo. Treatment with systemic prednisolone 25 mg daily, relieved the symptoms within 2 weeks. After tapering prednisolone treatment, the patient was patch tested with the European standard series, using Scanpor tape and Finn Chambers (Epitest, Tuusula, Finland). He had a +++ reaction to PPD and no other positive reactions.

Chemical analysis

The temporary tattoo sample obtained from the patient was analysed for PPD, using a previously described method (6). In brief, approximately a 1 g sample was extracted in a mixture of 15 mL acetonitrile and 10 mL phosphate buffer containing 1-heptanesulphonic acid and sodium ascorbate. The sample extract was analysed by high performance liquid chromatography (HPLC), employing diode array detection. Matching both the retention time and the UV spectrum of the HPLC-peak of the sample extract with the retention

time and UV spectrum of the HPLC-peak of PPD reference standard identified PPD in the sample extract. Determination of how much PPD was in the sample extract was made from a calibration curve of the standard PPD. The tattoo sample being found to contain 0.43% (w/w) PPD, recovery of PPD from the spiked sample was found to be 80%.

Discussion

The present case is the first time that a temporary tattoo reaction has been described on the fingertips and the first time that quantitative chemical analysis has been performed, demonstrating the presence of PPD, at a concentration of 0.43%. The fingertip reaction was unusual in causing severe pain and being oedematous and vesiculobullous. This is due to PPD being a potent allergen, and being applied at a high concentration. Similarly, PPD-containing hair colours can cause severe oedema of the face, not otherwise seen from cosmetic products. PPD is allowed in hair colours, according to the EU Cosmetics Directive, at a concentration up to 6% (7). The present case adds further evidence that potent allergens should not be permitted in consumer products unless they are used at restricted concentrations unlikely to induce or elicit contact allergy.

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Fig. 1. Vesiculobullous reaction on fingertips of patient.

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Contact dermatitis from anti-inflammatory gel containing hydroxyethyl salicylate

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Key words: allergic contact dermatitis; 2-hydroxyethyl salicylate, medicament.

One of the commonest causes of allergic contact dermatitis in the elderly are topical agents used to treat stasis ulcers and venous eczema. Contact sensitivity to hydroxyethyl salicylate (ethyleneglycol monosalicylic acid ester), an active ingredient of Venosin[®] gel (Klinge Pharma, Munich, Germany), Bayolin[®] cream (Bayer Austria, Vienna, Austria) and Ambenat[®] ointment (Merckle, Blaubeuren, Germany), has been reported in 1 case only (1). Allergies to other salicylates also seem to be rare (2–4).

Case Report

A 62-year-old woman with chronic venous insufficiency, and muscle and back pain applied various antirheumatic and anti-inflammatory gels

Table 1. Composition of the topical medicaments used by the patient

Product	Active ingredients	Clinical reaction
Venosin [®] gel	aescin heparin	dermatitis
Bayolin [®] cream	2-hydroxyethyl salicylate heparinoid nicotinic acid benzylester	dermatitis
Hirudoid [®] gel	2-hydroxyethyl salicylate	tolerated
Vetren [®] gel	MPS-polysulphuric acid ester heparin	tolerated

Table 2. Patch test results to Venosin[®] gel and other anti-inflammatory topical medicaments used by the patient. Day (D) 2 and 3 readings

	% vehicle	D2	D3
Venosin [®] gel			
gel	as is	+	+++
gel without active ingredients	as is	–	–
aescin	1% aq.	–	–
heparin	0.05% aq.	–	–
2-hydroxyethyl salicylate	5% aq.	+	+++
Bayolin [®] cream	as is	+	+++
(containing 2-hydroxyethyl salicylate)			
Vetren [®] gel	as is	–	–
(free of 2-hydroxyethyl salicylate)			
Standard series			–

and creams. She had been using Hirudoid[®] gel (Sankyo Pharma, Munich, Germany), Venosin gel, Bayolin cream and Vetren gel (Byk Gulden Lomberg, Constance, Germany), some of these containing heparin, 2-hydroxyethyl salicylate or both (Table 1). Venosin gel and Bayolin cream caused an acute contact dermatitis on her back and thighs after 2D. Her previous history indicated that application some years ago had caused dermatitis. It can be assumed that sensitization to at least 1 of the substances occurred at that time.

She was patch tested with Venosin gel, containing aescin, heparin and 2-hydroxyethyl salicylate, this gel without the active ingredients, and Bayolin cream containing heparinoid and 2-hydroxyethyl salicylate. A +++ reaction to 2-hydroxyethyl salicylate was obtained after 3D (Table 2). There were no reactions to Hirudoid gel, Vetren gel containing heparin, or a standard series.

Discussion

Our patient had allergic contact dermatitis from Venosin gel containing 2-hydroxyethyl salicylate. To our knowledge, and in spite of the wide

use of topical gels containing salicylates, there is only 1 other case report of contact sensitization to 2-hydroxyethyl salicylate (1): a man with osteochondrosis dissecans developed an acute contact dermatitis after application of Dolo-Arthrosenex[®] gel.

Salicylates are widely used in many topical analgesics. Patients have benefited from their analgesic, antithrombotic and anti-inflammatory effects for 80 years.

Allergies to other salicylates also seem to be rare (2–4). Many such topical medicaments are over-the-counter products, which leads to wide use in the general population. Considering this, 2-hydroxyethyl salicylate seems to be a rare sensitizer.

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Paronychia in cashew nut industry workers

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Key words: cashew nut; castor oil; occupational; paronychia; skin care products.

Impairment of the epidermal barrier of the proximal nail fold, through mechanical or chemical trauma, is a forerunner of chronic paronychia, because it encourages irritants, allergens and microorganisms (1–4).

Cashew nut industry workers are subject to dermatological lesions due to occasional contact with the main cashew nut shell liquid (CNSL) (5, 6), some of its components acting as both irritants (anacardic acid and other phenolic substances) and sensitizers (cardol) (5). The use of barrier creams to protect such workers has also been described (7), one such being castor oil (7).

Patients and methods

From previous research on contact dermatitis among workers who used only a mixture of castor oil and alco-

hol to protect their hands against CNSL (5), 20 workers, all female, were selected to be reexamined. Patch testing had already been carried out on these workers (5).

Results

Paronychia was observed in 9 workers; a further 16 presented with cutaneous lesions associated with CNSL contact (5). The only patient found to be sensitized to cardol showed no paronychia (5). Stopping the use of castor oil as a barrier cured their paronychia, even though they remained in contact with substances found to be sensitizers on patch testing (5). Their contact dermatitis from CNSL, however, got worse when the use of castor oil was stopped, recurrence of paronychia occurring when the oil was used again.

Discussion

We suggest that the paronychia exhibited by these workers is related to the mixture of castor oil and alcohol used to protect their hands. The seeds and plant from which castor oil is extracted have been associated with skin irritation (8). Additionally, the residue from castor bean oil extraction is associated with asthma and dermatitis among castor oil extractors, fertilizer workers, and farmers (8). Furthermore, nail alterations caused by other oils have been described (9).

This oil may protect by diminishing the lesions caused by CNSL, but it may also provoke paronychia manifestations. Further studies should be performed to confirm that castor oil is indeed causing the paronychia, and by what mechanism.

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Airborne contact dermatitis caused by the pesticide acephate

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Key words: acephate; airborne allergic contact dermatitis; organophosphates; pesticides.

Case Report

A 53-year-old woman noticed itchy papules on her ears a few days after

she collected fallen leaves in her garden, where her husband had sprayed the trees with the pesticide acephate. The initial eruption on her ears spread to her cheeks, forehead, and dorsa of the hands. Unexposed areas, such as where her spectacle frames protected the skin, were symptom-free. There was no history of contact allergy, atopic dermatitis or allergic rhinitis.

Patch tests with leaves and cosmetics were negative. A patch test with acephate (0.1% pet.) was positive at D2 and D3 (+/+ ICDRG criteria). This reaction was not enhanced by irradiation with 5 J/cm² of UVA at D2. Stopping using acephate resulted in disappearance of the dermatitis.

Comment

Acephate is an organophosphate foliar-spray insecticide, which is widely used in Japan. In a large Japanese study (1), 16.3% of farmers with contact dermatitis had positive patch test reactions to acephate 0.05% pet.

In a review (2), pesticides such as pyrethrum and tetrachloroacetophenone were cited as responsible for airborne contact dermatitis. To the best of our knowledge, this is the 1st report in English of airborne contact dermatitis due to acephate.

There have also been reports of photocontact dermatitis due to pesticides (3, 4). Our patient also developed erythema in anatomically shadowed areas of the skin such as the retroauricular and submandibular regions. In addition, her reaction to acephate on patch testing was not enhanced by UVA. Therefore, our case cannot be ascribed to photocontact dermatitis.

Matsushita et al. analysed 216 Japanese cases of contact dermatitis from pesticides (5). Most patients (96.6%) developed dermatitis from April to September, which correlated with the frequency of pesticide spraying in Japan. The face was the most frequent site of dermatitis, followed by the neck, forearms, hands, chest and back.

The most likely factor contributing to the causation of contact dermatitis from pesticides is insufficient clothing. Our patient's husband sprays acephate 1 × a month only from spring to summer every year,

and the patient suffered from dermatitis after the 1st spraying of the pesticide this year. She developed erythema on her face and hands, which she did not cover while gardening.

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Allergic contact dermatitis from occupational propyl gallate exposure

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Key words: allergic contact dermatitis; antioxidants; occupational; preservatives; propyl gallate; synthetic textile fibre manufacture.

Case Report

A 41-year-old man presented with marked erythema and oedema around the eyes. He worked at a plant which manufactured a synthetic textile fibre and used propyl gallate as a stabilizing agent. The previous day, he had been involved in cleaning a pre-mix hopper which had injected into it propyl gallate in a powdered form. He had performed the cleaning operation on several occasions previously, but felt that he might have become exposed to some unreacted propyl gallate on this occasion. His symptoms settled with redeployment away from potential sources of exposure of propyl gallate and the use of oral antihistamines. There was no relevant past medical history.

He was patch tested to the European standard series, together with propyl, octyl and dodecyl gallates. There was a positive reaction to propyl gallate (+ D2/+ D4) at 1% pet. and to an open test to a saturated solution of propyl gallate in ethanol (+ D2).

Discussion

Propyl gallate and other esters of gallic acid are used as antioxidants and preservatives. They are commonly added to cosmetics, medicaments and foods to prevent oxidation of unsaturated fatty acids. Propyl, octyl and dodecyl gallate have all been shown to be moderate to strong contact sensitizers, propyl gallate being the most frequent source (1). However, despite their extensive use, there are only a few reports of contact dermatitis from gallates.

Propyl gallate was first identified as a cause of lipstick dermatitis by Cronin (2). Since then there have been reports of allergic contact dermatitis from propyl gallate in lip balm (3) and in body lotion (4). Octyl and dodecyl gallate have also rarely been found to cause lipstick contact dermatitis (5).

Various topical medicaments containing propyl gallate as an antioxidant have been reported to produce contact dermatitis reactions (6–10).

In the food industry, butter, margarine and essential oils are common sources of propyl gallate, often combined with other gallate esters. Octyl and dodecyl gallate are known to cause allergic contact dermatitis in

bakers and other food handlers (11, 12). The gallates are listed in commercially packaged foods by the following E numbers: propyl gallate E310, octyl gallate E311 and dodecyl gallate E312. There have not yet been any reports of developing either an immediate Type I or a delayed Type IV hypersensitivity reaction to ingesting food containing the esters of gallic acid.

Reports of contact dermatitis from propyl gallate are rare despite its widespread use and it has been suggested that this may be due to immunological tolerance induced by oral exposure and the low concentration of the allergen in cosmetics (6, 13). Occupational exposure to propyl gallate in an industrial setting resulting in allergic contact dermatitis has not previously been reported.

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Comparative testing with budesonide in petrolatum and ethanol in a standard series

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Key words: budesonide; corticosteroid; ethanol; medicaments; patch testing technique; petrolatum; standard series.

Both the concentration and vehicle for optimal patch testing with budesonide remain controversial (1–6). While 0.01% w/w in petrolatum (pet.) was added to the European standard series in 2001 (1), a higher concentration (1.0%) in ethanol (eth.) has also been advocated (6).

Materials and methods

To compare the outcome of patch testing with budesonide 1.0% eth. and 0.01% pet. in consecutively

patch tested patients, 3 centres (Malmö, Leeds, and Manchester) added the 2 test preparations to their standard series from 1 September 2000 to 30 November 2001 (Leeds and Manchester) and 31 December 2001 (Malmö), respectively. Readings in Leeds and Manchester were performed on day (D) 2 and D4 and in Malmö on D3 or D4 and D7. Budesonide was purchased from Chemotechnique Diagnostics, Malmö, Sweden, by the Malmö clinic and the eth. test solution was to be made up by each centre every 3 months. The pet. test preparation, prepared at the beginning of the study by Chemotechnique Diagnostics from the same batch of budesonide as used in the eth. preparation, was sent out to the 2 English clinics from the Malmö clinic. The stability of the eth. test preparations in Malmö was investigated by high performance liquid chromatography (7).

Results and Discussion

Of 4033 patients (Malmö 1097, Leeds 1376, Manchester 1560) so tested, 31 were found to be allergic to budesonide (Table 1). Both test preparations detected the same number of allergic patients: 2 such patients were missed with the eth. preparation and another 2 with the pet. preparation.

Either preparation may therefore be used for screening purposes, though the pet. preparation has the advantages of stability (7) and ease of handling. Corticosteroids in eth. degrade (8, 9): in this study, investigation of the stability of budesonide in eth. over 26–65 days in the Malmö clinic showed that in 2/19 vials in use, the concentration of budesonide decreased by more than 20%, and in 3/19 the concentration increased by more than 20%, thresholds previously suggested for stability (7). Also, when eth. is used as the vehicle, it should also be tested as a control, because eth. itself may give irritant reactions (10).

Extremely strong patch test reactions have previously been reported with budesonide in eth. In the patients here reacting to both preparations, there were stronger reactions to eth. in 5 and stronger reactions to pet. in another 5. Budesonide 0.01% pet. gave stronger reactions at D2

Table 1. The yield of positive patients in the 3 centres and their patch test results

Patient	Budesonide 1.0% eth.				Budesonide 0.01% pet.			
	D2	D3	D4	D7	D2	D3	D4	D7
M 1	nd	+++	nd	+++	nd	++	nd	++
M 2	nd	++	nd	++	nd	++	nd	++
M 3	nd	++	nd	++	nd	++	nd	++
M 4	nd	?	nd	++	nd	–	nd	+
M 5	nd	++	nd	++	nd	++	nd	++
M 6	nd	++	nd	++	nd	++	nd	++
L 1	–	nd	+	nd	–	nd	+	nd
L 2	–	nd	+	nd	–	nd	–	nd
L 3	–	nd	+	nd	–	nd	+	nd
L 4	–	nd	++	nd	–	nd	–	nd
L 5	+	nd	++	nd	+	nd	+	nd
L 6	+	nd	++	nd	+	nd	+	nd
L 7	+	nd	++	nd	++	nd	++	nd
Ma 1	+	nd	+	nd	++	nd	++	nd
Ma 2	?	nd	++	nd	?	nd	++	nd
Ma 3	–	nd	++	nd	–	nd	++	nd
Ma 4	++	nd	++	nd	++	nd	++	nd
Ma 5	+	nd	++	nd	+	nd	++	nd
Ma 6	?	nd	++	nd	+	nd	+++	nd
Ma 7	+	nd	++	nd	+	nd	++	nd
Ma 8	++	nd	++	++	++	nd	++	++
Ma 9	+	nd	++	++	++	nd	++	++
Ma 10	++	nd	+++	nd	+++	nd	++	nd
Ma 11	–	nd	–	nd	+	nd	++	nd
Ma 12	+	nd	++	nd	++	nd	++	nd
Ma 13	–	nd	++	nd	?	nd	+	nd
Ma 14	–	nd	+	nd	–	nd	++	nd
Ma 15	+	nd	++	nd	++	nd	++	nd
Ma 16	++	nd	++	nd	+++	nd	++	nd
Ma 17	++	nd	++	nd	+++	nd	++	nd
Ma 18	–	nd	–	nd	–	nd	+	nd
Missed	11/25	1/6	2/25	0/6	9/25	1/6	2/25	0/6

M = Malmö; L = Leeds; Ma = Manchester; nd = not done; ? = doubtful reaction.

than did 1.0% eth. but by D4 this effect had disappeared.

In conclusion, similar results were obtained when patch testing with either test preparations. However, for screening, we advocate that budesonide should be tested in pet. for reasons of stability and practicality.

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Active sensitization to para-tertiary-butylphenol-formaldehyde resin

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Key words: active sensitization; para-tertiary-butylphenol-formaldehyde resin (PTBP-F-R); patch test.

Para-tertiary-butylphenol-formaldehyde resin (PTBP-F-R) is used in many adhesive formulations with industrial and domestic uses. PTBP-F-R 1% pet. is included in the European standard series of patch test allergens.

Case Report

A 32-year-old nurse presented with a 6-month history of hand dermatitis. Patch testing with the GEIDC standard series, a corticosteroid series and her own products were negative after 2 and 4 days.

She returned 5 weeks later because of pruritus on her back. Examina-

tion showed an erythematous macule at a site corresponding to PTBP-F-R. Re-patch testing with the standard series was positive to PTBP-F-R at 2 and 4 days. She had no history suggesting earlier sensitization to PTBP-F-R, so active sensitization by the initial patch test was diagnosed, in addition to irritant contact dermatitis of the hands.

Discussion

Active sensitization from patch testing with a standard series is rare (1). Its most frequent cause is too high a concentration of test substance; of less importance are the amount and duration of patch test application (1, 2). Adams warns that testing with substances of unknown composition that are brought in by patients presents the greatest hazard of patch testing (3).

To the best of our knowledge, this is the 1st report of active sensitization to PTBP-F-R 1% pet. Calnan reviewed 43 patients with active sensitization in 1967, and 1 was sensitized to PTBP-F-R 10% pet. In this review the most common active sensitizers were tetramethylthiuram disulphide and paraphenylenediamine (4). Estlander et al. reported 5 cases of active sensitization by para-tertiary-butylcatechol (PTBC) tested at 0.5–1%; only 1 of these had been exposed to PTBC in his work (5).

We continue to agree with the above authors (1–4) that the benefits of patch testing outweigh its rare adverse effects.

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