

CONTACT POINTS

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Occupational airborne contact allergy to cyanamide and dibenzyl phosphite

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Key words: airborne contact allergy; chemical workers; cyanamide; dibenzyl phosphite; occupational; phosphorylcreatine.

Case Report

A 45-year-old man had been employed as a chemical worker since 1996 in an industry producing pro-drugs. In May 2001, the factory began to produce phosphorylcreatine, with a half-enclosed working cycle, and our patient was deputed to supervise its synthesis. After 20 days, he developed severe eczematous dermatitis with diffuse, symmetrical involvement of the face, including the eyelids, the region under the chin and the retroauricular folds. The symptoms disappeared when away from the workplace, with the aid of topical corticosteroid treatment and systemic antihistamines. On return to work on the same job, the patient developed a new dermatitis episode. He was then relocated to a different area of the plant and the dermatitis did not recur.

The patient was patch tested with the Italian Society of Allergological, Occupational and Environmental Dermatology (SIDAPA) standard series with negative results. Further patch testing was performed with the substances used in the synthesis of phosphorylcreatine (Table 1). Readings were made on D2 and D4.

Table 1. Patch test results

	D2	D4
SIDAPA standard series	–	–
Benzyl alcohol 1% pet.	–	–
Cyanamide 1%, 0.1% and 0.01% pet.	++	++
Phosphorylcreatine raw 5% and 1% pet.	++	++
Phosphorylcreatine 'pure grade' 5% pet.	+	+
Phosphorylcreatine 'pure grade' 1% pet.	–	–
Pyridine 1% pet.	–	–
Dibenzyl phosphite 1%, 0.1%, 0.01% and 0.001% pet.	++	++
Sarcosine methyl ester hydrochloride 5% pet.	–	–

SIDAPA = Italian Society of Allergological, Occupational and Environmental Dermatology.

The patient showed positive reactions to dibenzyl phosphite, cyanamide and phosphorylcreatine. Photopatch tests with the above substances were negative. 5 healthy volunteers were negative to cyanamide 1% pet. and dibenzyl phosphite 0.1% pet.

Discussion

Phosphorylcreatine synthesis includes the use of sarcosine methyl ester hydrochloride, dibenzyl phosphite and cyanamide as reagents. Most likely, sensitization to phosphorylcreatine was due to the presence of dibenzyl phosphite and cyanamide residues, because patch tests with phosphorylcreatine pure grade 5% and phosphorylcreatine pure grade 1% gave a weak positive and a negative reaction, respectively. Cyanamide is an organic amide used in different settings, such as in chemistry, in antirust solutions, or in drugs for treating alcoholism. There have been sporadic reports of contact dermatitis from cyanamide, especially in health-care workers preparing and adminis-

tering drugs to treat alcoholism (1, 2). Calnan reported a case of a positive patch test to cyanamide in a chemist who had handled the substance (3).

To our knowledge, this is the 1st report of allergic contact dermatitis from dibenzyl phosphite [phosphonic acid bis(phenyl-methyl)ester: (HO)P(OCH₂C₆H₅)₂] (Fig. 1), a substance used in the pharmaceutical industry as a phosphorylating agent for converting pro-drugs.

The eczematous dermatitis we observed affected a chemical worker delegated to quality control procedures in the industrial synthesis of phosphorylcreatine. Chemical workers not directly involved in industrial production processing, but assigned to supervising or coordinating plant procedures, are usually considered at low risk of occupational dermatitis (4). For this reason, they may become inclined to disregard the use of protective measures because they are not directly exposed to chemical products. In our case, the failure to observe protective measures and the presence of phosphorylcreatine and

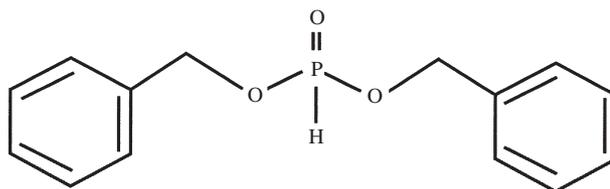


Fig. 1. Chemical structure of dibenzyl phosphite.

its reagents in the environment favoured the onset of airborne allergic contact dermatitis.

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Allergic contact dermatitis due to perupok wood

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Key words: allergic contact dermatitis; occupational; patch test; perupok; woods.

Case Report

A 56-year-old botanist visited our hospital because he had come into contact with perupok, and 1 month after this, a rash spread all over his body. The result of skin biopsy suggested allergic contact dermatitis. A patch test with perupok sawdust (50% pet.) resulted in a ?+ reaction at D2, + at D3 and D7. This same

patch test preparation was negative in 10 normal control patients. The eruption disappeared after 2 weeks of treatment with oral prednisolone and topical corticosteroid.

Discussion

Perupok (*Lophopetalum dubium*, *Lophopetalum floribundum*) is a shrub mainly found in Southeast Asia. Although not as useful a wood as many others, it has recently been used more because of a serious shortage of wood throughout the world. There have been no cases of contact dermatitis from perupok until now.

Most cases of contact dermatitis from wood are occupational in forestry (1–3). The possibility of contact dermatitis from treatment of wood also has to be considered (4, 5). Our test wood did not have any such additives. We, therefore, diagnosed allergic contact dermatitis from perupok itself. Vigilance will continue to be required, if newly used woods are to be detected as causes of contact dermatitis (5, 6).

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Labial edema due to an acrylic dental prosthesis

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Key words: (meth)acrylates; allergic contact cheilitis; cross-sensitivity; dental prostheses; labial edema.

Occupational allergic contact dermatitis caused by (meth)acrylates is relatively common in dental personnel (1), whereas denture material-induced reactions to acrylates in dental patients are less common (2–7). Stomatitis (2–4), burning mouth syndrome (5) and cheilitis (6) are adverse oral mucous membrane reactions from acrylic dental prostheses in dental patients.

Case Report

A 72-year-old woman had had her metallic dental prosthesis changed to a metallic and acrylic one 3 years ago. Approximately a week after the change, she reported edema and a burning sensation of both lips that disappeared after the removal of the prosthesis and reappeared hours after its replacement. From that time onwards, because of these symptoms, she had used 3 different dental prostheses, made exclusively of acrylates, continuing to have flare-ups of edema of both lips that were progressively more intense and clearly related to their use. No erythema, ulceration or cheilitis were present. No wheals or other cutaneous lesions were observed.

Patch tests with the Spanish standard series, a dental screening series (Chemotechnique Diagnostics AB, Malmö, Sweden) and a (meth)acrylates series (Chemotechnique Diagnostics AB) were positive to methyl methacrylate 2% pet. (MMA), 2-hydroxyethyl methacrylate 2% pet. (2-HEMA), 2-hydroxypropyl methacrylate 2% pet. (2-HPMA), ethyleneglycol dimethacrylate 2% pet. (EGDMA), gold sodium thiosulfate and para-phenylenediamine (Table 1). Reading of patch tests with the (meth)acrylate series was negative after 30 min.

Table 1. Positive patch test results

	D2	D4
p-Phenylenediamine 1% pet.	++	++
Gold sodium thiosulfate 2% pet.	+	++
Methyl methacrylate 2% pet.	++	++
2-hydroxyethyl methacrylate 2% pet.	+	++
2-hydroxypropyl methacrylate 2% pet.	+	++
Ethyleneglycol dimethacrylate 2% pet.	+	++

According to the manufacturer, the components of the powder and liquid of the last acrylic denture base material used by the patient were: (1) powder containing polymethyl methacrylate, benzoyl peroxide and cadmium and ferric salts and (2) liquid containing MMA, EGDMA and hydroquinone. The patient started to use a dental prosthesis made of nickel and chrome, and the edema of the lips resolved.

Discussion

In contrast to the usual clinical presentation of allergic reactions to acrylics (4–6), our patient did not have typical oral symptoms. Instead, she developed recurrent episodes of edema of the lips as an expression of a delayed hypersensitivity reaction clearly related to the use of different acrylic prostheses. These ceased as soon as the exposure to the allergen stopped. Patch tests showed negative results after 30 min. Positive patch tests read at D2 and D4 to (meth) acrylates allowed the diagnosis.

Atypical clinical forms of allergic reactions to acrylic dental prostheses have been previously reported (3, 7): chronic urticaria without mucosal or perioral lesions (7) and stomatitis and edema of the tongue, lips, eyelids and hands (3). In our case, patch testing with monomeric acrylic resins and repeated exposure to the prosthesis provided confirmation of contact allergy. Patch testing showed a positive reaction not only to MMA but also to 2-HEMA, 2-HPMA and EGDMA, some of which may be explained by cross-reactivity (8).

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Irritant paronychia with onychodystrophy caused by cyanoacrylate nail glue

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Key words: artificial nails; cosmetics; cyanoacrylate; irritant contact dermatitis; nail glue.

Case Report

A 37-year-old woman presented with a 1-month history of paronychia, onycholysis and onychodystrophy of all the fingernails. She had used preformed acrylic nails for 3 consecutive months, applying frequently (once or twice a week) a nail glue (Fing'rs[®] nail glue, Zürich, Switzerland) to keep the artificial nails adherent to the nail plates. The packaging of the glue declared the presence of cyanoacrylate, without specifying which one, and lacked a full list of ingredients. After the sudden onset of paronychia, she decided to remove the plastic nails and saw dystrophic changes of fingernails.

The patient was patch tested with the Italian standard series (meth)acrylates series (Chemotechnique, Malmö, Sweden), her own nail glue 10% pet. and the glue as is directly applied on the skin. Patch testing was positive (++) at D2 and D4 only to nickel sulfate. Discontinuation of the use of artificial nails resulted in rapid healing of the paronychia and gradual improvement in the onychodystrophy, with residual onychorrhexis after 6 months.

Discussion

Cyanoacrylate glues are used as adhesives for preformed acrylic nails. Contact allergy to cyanoacrylates is considered to be unlikely, as they bind strongly with keratin and rapidly polymerize on the skin. Nevertheless, allergic contact dermatitis from cyanoacrylates, especially ethyl cyanoacrylate, contained in nail glues has been reported (1–3). Ena et al. (4) also described a case of acute leukonychia, developing after accidental penetration of a cyanoacrylate glue onto the nail plate.

The toxic effects, related to long-term application of cyanoacrylate glues on nail plates and folds, are unknown; the frequency of onychodystrophies caused by these glues may be underestimated, considering that the use of preformed artificial nails occurs on already dystrophic nails in many cases.

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Contact urticaria from dill

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Key words: allergy; contact urticaria; dill; IgE; plants.

Foods and spices of the parsley family (Umbelliferae, formerly Apiaceae) include anise, caraway, coriander, cumin, celery, lovage, fennel, parsley, carrot and dill. Some of these have been associated with various allergic reactions including contact dermatitis, anaphylaxis, gastroenteritis and asthma (1–4). We report a patient with contact urticaria who handled several different plants.

A 32-year-old housewife with a personal history of atopy was investigated in our department. She reported that during the previous 1 year, she had developed symptoms

when handling dill plants. There was a history of rhinitis and asthma.

Serum total immunoglobulin E (IgE) was 325 kUL⁻¹ (HYCOR Biomedical Inc.-IZASA, Barcelona, Spain). Specific IgE to dill (and other allergens) was detected with enzyme-linked immunosorbent assay (ELISA), which was performed with the commercial kit (HYCOR) using activated paper disc as solid phase.

The allergenic extract was specially prepared at 5% w/v in phosphate-buffered saline (PBS) (0.15 M), pH 7.2, using the following procedure: 5 g of dill in 100 ml of PBS; extraction overnight at room temperature with stirring; centrifugation at 25 000 × g for 15 min; supernatant prefiltered and dialysed against PBS containing thimerosal in a tube with cut-off of 3500 Da at 4°C for 24 h; filtration with Millipore filters of 0.8 µm. The same procedure was followed to obtain the extracts from anise, caraway, coriander, cumin, celery, lovage, fennel, parsley and carrot.

The solid phase was prepared from the extracts.

We detected specific IgE to dill (IgE ELISA Score (IES): 5.6), anise (IES: 2.5), cumin (IES: 3) and carrot (IES: 4). We did not detect specific IgE to the other members of the parsley family. Prick tests were positive to dill (++++), anise (++) and carrot (++) and negative to the other extracts. All these extracts were negative in 6 non-atopic and in 6 atopic control subjects. The patient had positive prick test (Bial-Aristegui, Bilbao, Spain) and specific IgE (HYCOR) to grass and *Olea europaea* pollen extracts. Both skin tests and ELISA to salmon were negative.

Patch tests with extracts of dill and other members of the parsley family extracts were applied for D2, and readings made at D2, D3 and D7. All these extracts were negative in the patient and in 6 non-atopic and 6 atopic control subjects.

The various members of the parsley family have antigens that are either in common or cross-react (4, 5). The patient reported here demonstrated positive tests to other members of the *Umbelliferae* in addition to dill.

To our knowledge, this is the 1st case report of allergic contact

urticaria caused by dill. The patient no longer had symptoms after avoiding further contact with dill.

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Occupational allergic contact dermatitis from polyurethane/methacrylates in windscreen repair chemical

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Key words: fingertips; hand dermatitis; occupational; semiopen test; UV-curable resins; (meth)acrylic resins; windscreen.

Case Report

A 24-year-old man was referred with recurrent occupational dermatitis. Lesions had started on his fingertips 3 weeks before his seeking medical advice. They had then spread to palmar aspect, perionychium and dorsum of his fingers. The patient had been employed for 6 months in

a small company and his job consisted in replacing windscreens and repairing small defects. For this last task, he mostly worked with a glass cleaner, a wiper and a primer and a chemical system to repair windscreen defects. This system was used either to repair big defects with a pressure system injecting resin, or employed as such to repair small defects, the resin being applied directly to the defect. The patient did not use the pressure system, but applied the liquid resin directly without protective gloves. This mono-component resin Glass Medic[®] repair system (AGSI, Kempston, UK) available in a small bottle, was applied to small defects, covered with a transparent plastic film and then rapidly cured under ultraviolet (UV) light (around 5 min). The safety data sheet indicated that the chemical was composed of polyurethane/methacrylic resin at >50% concentration, acrylic acid and maleic acid. Patch tests were performed, using the Finn Chamber technique (Epitest, Tuusula, Finland), with the revised ICDRG standard series (1), additional allergens, methyl acrylate, ethyl acrylate, butyl acrylate and 2-hydroxyethyl acrylate, each 0.1% pet. and methyl methacrylate 2% pet. The patient's own materials were tested in semiopen tests, on 5 × 5 cm² areas: the windscreen cleaner and the diluents as is, and the windshield repair polyurethane/methacrylate-based resin 1 p. 1000 (v/v) in acetone. A positive (+) reaction was observed after 3 days to the resin only, which persisted for more than 2 weeks. After several severe relapses of his dermatitis due to further minimal contact with contaminated gloves, the patient almost completely avoided contact with this product and the lesions progressively cleared. In this small company, the windshield repair chemical system was then made available only in encapsulated form.

Discussion

This report illustrates a recently described mode of sensitization to (meth)acrylic compounds, which can occur in people working in the car or car-glass repair industry. The first case of dermatitis from a glass repair chemical based on (meth)acrylic UV-curable resins was reported by Pedersen (2) in a workman repairing minor damage in car windscreens. The patient had positive reactions

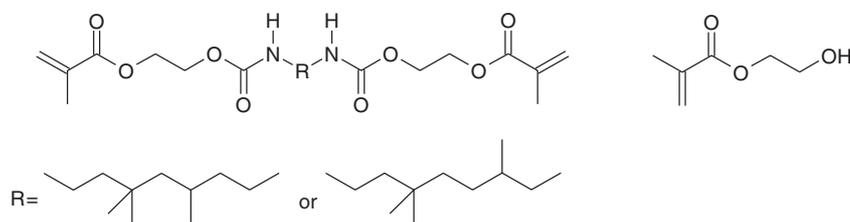


Fig. 1. Chemical formulae of polyurethane/dimethacrylate and 2-HEMA (5).

to 2-hydroxyethyl methacrylate (2-HEMA) and methyl methacrylate, but his own material, namely TEAM GLAS, prep 20, was not tested. A recent report describes a patient with right thumb pulp and index finger dermatitis probably due to occupational contact with acrylic-based windscreen glass repair resin. He secondarily developed an allergic contact dermatitis due to an electro-surgical earthing plate coated with acrylic adhesive (3). Patch testing was positive to the patient's own resin 2% pet., and several (meth)acrylates. In the present report, the sensitizer was probably the polyurethane/methacrylate, a UV-curable polymer without CAS number. Polyurethane/methacrylate is prepared from (1) polyurethane components, namely a diisocyanate (toluene, hexamethylene or isophorone diisocyanate) and a polyol (polyethylene glycol PEG 200, PEG 400 or PEG 1000), (2) the UV-curable resin 2-HEMA and (3) other additives and catalysts (4). Its chemical formula is illustrated in Fig. 1. This polymer is used as an anaerobic sealant or threadlocker ingredient, for use on threaded fasteners and flange sealing applications. It can be used in 2-component glues used to bond metals and ferrites.

Clinical data are similar in patients with delayed hypersensitivity to (meth)acrylic resins: an acquired pruritic fingertip dermatitis that progressively spreads over the fingers and hands, with occupational features. When safety data sheets are lacking, patch testing can be performed with the patient's own material diluted in acetone in a semiopen test. In our case, a 1 p. 1000 concentration was able to elicit a positive reaction, probably without risk of active sensitization to any acrylic resins present. According to its preparation modalities and to its chemical formula, sensitivity to polyurethane/methacrylate could be detected by a positive reaction to

2-HEMA, this hapten either acting as the offending agent present as an impurity in the chemical or, more likely, being the epitope and marker of delayed sensitivity.

Like Pedersen (2), this report emphasizes a situation frequently observed in small companies. Workers often handle chemicals without gloves, these often being used only after the onset of dermatitis. As information on safety data sheets and packages is rarely read, caution should be reinforced for occupational allergens, with the labelling Xr43 clearly indicated on materials.

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Corticosteroid contact allergy from a nasal spray in a child

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Key words: allergic contact dermatitis; budesonide; child; medicaments; nasal spray; peri-nasal; topical corticosteroids.

Case Report

A 9-year-old boy presented with erythema, oedema and scaling around the nose and complaints of severe pruritus. The lesions persisted for 2 years and had started 3 weeks after beginning treatment with budesonide nasal spray (Pulmicort[®] Nasal Aqua; AstraZeneca, Barcarena, Portugal) for allergic rhinitis.

There was a history of previous use of other topical corticosteroids, as both creams and nasal sprays. Patch tests showed positive reactions to Pulmicort[®] nasal spray (as is), corticosteroid mix, tixocortol pivalate and budesonide, other corticosteroids tested all being negative (Table 1). The patient was instructed to stop the use of nasal sprays, and progressive improvement of the skin lesions was observed thereafter.

Comments

Budesonide is a synthetic, non-halogenated, moderately potent topical corticosteroid, with a 16 α -, 17 α -butylidene dioxy portion used in the treatment of inflammatory dermatoses and also widely in nasal sprays to treat rhinitis and bronchial asthma (1). It seems to be a more important sensitizer when used as a nasal spray (2).

Several cases of allergic contact dermatitis from this drug, in both ointments and nasal sprays, have been reported in adults, but antigen-determinant structure and cross-reactivity between this agent and the other corticosteroids remain unclear (3).

Considering the widespread use of budesonide nasal sprays, sensitivity to them is considered uncommon (4). In children, this must be even rarer; there being only 1 description of 2 cases of peri-oral allergic contact dermatitis from inhaled budesonide (5).

Cross-reaction studies are essential to recommending safe substitutes to the allergic patient (6). Cross-reaction evaluation is also important to define the best markers of corticosteroid hypersensitivity (7). Concerning budesonide cross-reactions, it is known that patients with allergic contact dermatitis from this drug do not cross-react to amcinonide, as also did occur in our case, while patients with contact allergic contact dermatitis from amcinonide generally do cross-react to budesonide (8).

In our case, it is likely that corticosteroid nasal sprays sensitized the patient to both budesonide and tixocortol pivalate. This report illustrates a very unusual allergic contact dermatitis from inhaled corticosteroids in a child.

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Table 1. Patch test results

Substance	Concentration (%)	Vehicle	D2	D4	D7
Pulmicort [®] nasal spray	As is	pet.	–	+	++
Corticosteroid mix	Tixocortol pivalate (1)	pet.	–	+	++
	Budesonide (0.1)		–	+	++
	Hydrocortisone-17-butyrate (1)				
Tixocortol pivalate	1	pet.	–	+	++
Budesonide	0.1	pet.	–	+	+++
Amcinonide	0.1	pet.	–	–	–
Betamethasone-17-valerate	1	pet.	–	–	–
Triamcinolone acetonide	1	pet.	–	–	–
Clobetasol-17-propionate	1	pet.	–	–	–
Hydrocortisone-17-butyrate	0.1	pet.	–	–	–
Alclomethasone dipropionate	1	pet.	–	–	–
Dexamethasone phosphate	1	pet.	–	–	–

Patch testing with thin-layer chromatograms

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Key words: clinical relevance; contact allergy; exposure; identification of sensitizers; isolation; paper chromatography; thin-layer chromatography; TLC; TLC-patch test.

When thinking of an inexpensive shortcut for the isolation and identification of a sensitizer, a Dutch investigation from the 1960s came to mind. In that study, paper chromatograms were patch tested in the process of identifying the sensitizer in tulips (1). In this paper, we report on an updated version of this methodology, thin-layer chromatography patch test/testing, and briefly on our own experience with it during the past 3 years.

Materials and Methods

Thin-layer chromatography (TLC) plastic roll 500 × 200 cm (silica gel 60F₂₅₄) (VWR International, Stockholm, Sweden) is cut into 18 × 18 cm strips. Samples to be tested are deposited on one spot each from a 10 µL capillary pipette, along a straight line with at least 2 cm between each spot. These samples are prepared in the same way, including diluent and concentrate, as when normally patch tested upon the back. 30 µL of each sample is then applied on the TLC plastic roller patch. This is double the volume used in the regular patch test procedure with filter paper in the small Finn Chamber (Epitest Ltd Oy, Tuusula, Finland). Double spots are applied for each sample that is to be investigated, one to be used as a patch test and the other to be used as a comparison and reference when reading the patch test. When the product tested is suspected to contain a specific allergen, this allergen is naturally also applied on the TLC plastic roll as a reference substance.

The 18 × 18 TLC plastic roll chromatogram is developed in a tank lined with solvent-saturated filter paper. It is attached in the tank, so that the chromatogram is straight, by clips on a welded stainless steel construction (Fig. 1). When the chromatogram is developed, the plate is investigated in UV light to detect spots that are not coloured. The spots are then marked on the chromatogram. In some cases, such as with diglycidylether of bisphenol A, the chromatograms are sprayed with reagents turning the non-visible spots violet (2).

If the separation is successful, each spot gives rise to a band of well defined and separated spots. These bands are cut out from the chromatogram in pieces of about 2.5 × 18 cm and are then applied on the upper arm using Scanpor (Norgesplaster A/S, Vennessla, Norway) tape. The position of the TLC-patch test (TLC-PT) is carefully marked. The TLC-PT is removed after 2 days and read after 1 additional day (D) and preferably also on D7 according to International Contact Dermatitis Research Group (ICDRG) criteria. When reading the patch tests, the 2nd chromatogram, deriving from the double sample, is used as a reference to determine to which spot/spots the positive reaction(s) correspond(s) to.

Results

We have used TLC-PT for products such as textiles, plastics, food, plants,

perfumes, drugs and grease, particularly in the following 3 situations.

- (1) When a patient patch tests positively both to a separate sensitizer such as a textile dye and to a coloured textile garment, TLC-PT can demonstrate that the patient tests positively both to the reference substance (textile dye) applied separately on the TLC plate and to the reference sensitizer (textile dye) in the textile.
- (2) When a patient patch tests positively to a compound product, it is valuable to test the ingredients to specify the allergy. When the ingredients cannot be obtained from the manufacturers, the dermatologist may test with the ingredients identified and already available. If the patient tests positively to one of these, TLC-PT can be used to show that the reactivity is directed towards only 1 substance, i.e. the reference sensitizer.
- (3) For certain products, TLC-PT may be a shortcut for the identification of a new or initially unknown sensitizer in a compound product. We have successfully used it for identification of the sensitizer in grease and are currently using it for the identification of sensitizers in food, plants and drugs.

To exemplify the methodology, the epoxy resin widely used in standard patch test series can be used as an example. Diglycidylether of bisphenol A, the monomer with a molecular weight of 340, is considered to be the major sensitizer

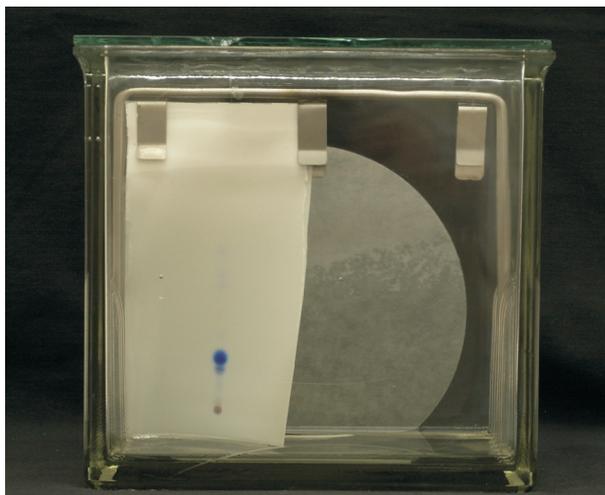


Fig. 1. Development of thin-layer chromatogram plastic roller that is straightened out and fixed to the tank in an upright position by attachment to metal clips.



Fig. 2. Positive thin-layer chromatograph-patch test reaction to a spot corresponding to the monomer (MW = 340) on the chromatogram shown in the figure in a patient positive to epoxy resin in the standard series.

and patch testing with a TLC-PT of this resin confirms this (Fig. 2).

The TLC-PT also has its limitations. All substances are not amenable to TLC, because they cannot be visualized on a chromatogram. However, even if a positive reaction is noted to a 'non-detectable' spot on a TLC-PT, the silica can then be scraped off and extracted and used for further investigations. The problem with the 'undetectable' sensitizer is that of developing a mobile phase to enable separation. Another limitation of TLC-PT is its detection limits. If the patch test reaction to the compound product is weak, the concentration of the sensitizer in the TLC chromatogram is likely to be below the individual elicitation level and thus result in a false negative reaction, unless this is not compensated for by applying a larger volume on the TLC plate.

In conclusion, TLC-PT is or can be a valuable tool for most dermatologists with an interest in contact dermatitis and its chemistry and who have some access to laboratory facilities.

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Contact allergy to decyl glucoside in antiseptic after body piercing

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Key words: alkyl glucosides; allergic contact dermatitis; antiseptics; CAS nos. 58846-77-8, 68515-73-1, 141464-42-8, 28211-18-9; coco glucoside; patch testing.

Case Report

A 29-year-old woman with no history of allergy had an umbilical piercing in August 2002. For wound care, she used $\times 2$ daily a chlorhexidine digluconate-based antiseptic gel. After 2 weeks, she developed acute eczema on the treated area that spreads over the whole abdomen and cleared after the withdrawal of the gel and topical corticosteroids. Patch testing was performed with the revised ICDRG series (1), additional allergens, the patient's own antiseptic gel, a similar antiseptic solution containing the same ingredients at the same percentages but without decyl glucoside or hydroxyethylcellulose and chlorhexidine digluconate 0.5% aq. Readings at D2/D3 showed a +++/+++ reaction to the gel only.

Breakdown of gel was performed into 4 ingredients provided by the firm, diluted in water at concentrations identical to that in the product, namely hydroxyethylcellulose, glycerin, chlorhexidine digluconate and decyl glucoside. A +++ reaction to decyl glucoside 0.55% aq. (commercial product used 1% in the gel formula is a 55% decyl glucoside aqueous solution) confirmed delayed hypersensitivity to this component only.

Discussion

Decyl glucoside or decyl D-glucoside, also named decyl-beta-D-glucopyranoside, belongs to the alkyl glucosides family and is obtained by the condensation of the fatty alcohol decyl alcohol and a D-glucose polymer (Fig. 1). Industrially, the glucose

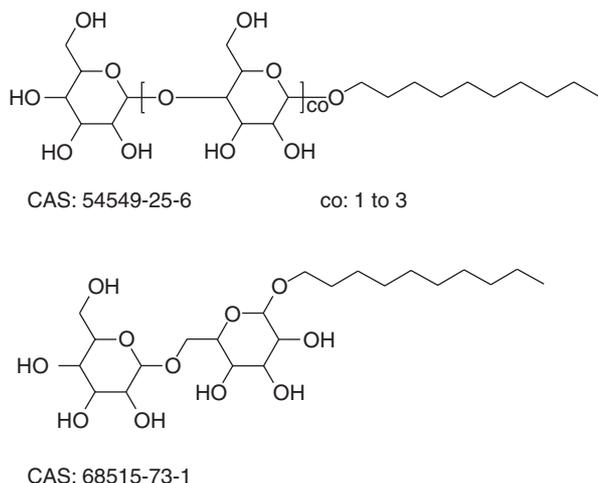


Fig. 1. Molecular structures found for decyl glucoside.

is obtained from maize or wheat and the fatty alcohol from coconut oil. Decyl glucoside is marketed under several names including AG-10LK, Atlas G-73500, Oramix NS 10 and Plantaren 2000.

This non-ionic surfactant and cleansing agent has been widely used for several years, due to its foaming power and good tolerance in rinse-off products like shampoos, hair dyes and colours, and soaps. Decyl glucoside is also employed in leave-on products such as no-rinsing cleansing milks, lotions and several sunscreen agents and is contained as a stabilizing surfactant of organic microparticles in sunscreen agent Tinosorb[®] M. Totally biodegradable, decyl glucoside is found in several 'gentle' and 'natural' cleansers. Although classified among irritants, a 55% aqueous solution of decyl glucoside tested at 10% dilution in 100 volunteers was neither irritant nor sensitizing. However, despite its chemical structure and reactivity, decyl glucoside could be a sensitizer. Other glucosides are used for similar properties (2), like coco glucoside and lauryl (dodecyl) glucoside in cosmetics, and cetearyl glucoside as a surfactant and emulsifying agent because of its higher viscosity. In practice, and probably due to their manufacturing processes, such alkyl glucosides are blends of several copolymers based on several fatty alcohols and a glucoside polymer. For example, coco glucoside, provided by Cognis in Plantacare[®] 818 UP, contains C₆ (max 0.5%), C₈ (24–30%), C₁₀ (15–22%), C₁₂

(37–42%), C₁₄ (12–18%) and C₁₆ (max 4%) fatty alcohols, while decyl glucoside in Plantacare[®] 2000 UP contains C₆ (max 1%), C₈ (33–40%), C₁₀ (21–28%), C₁₂ (27–32%), C₁₄ (9–12%) and C₁₆ (max 1%) alcohols (3).

Such variations may explain some uncertainty when searching for the precise CAS no. of decyl glucoside. 3 CAS nos. are found in the International Cosmetic Ingredient Dictionary and Handbook (2), 58846-77-8, 68515-73-1 and 141464-42-8. On the INCI website, however, the CAS no. for decyl glucoside is only 54549-25-6 (4) and concerns a C_{8–10} alkyl polyglucoside, i.e. a glucoside polymer condensed with octanol and/or decanol. CAS no. 58846-77-8 designates *n*-decyl- α -D-glucopyranoside or *n*-decyl- β -D-glucopyranoside. CAS no. 68515-73-1 refers to C_{8–10} glucoside based on octyl- and decyl glucoside too but is not mentioned on the INCI website (3). CAS no. 141464-42-8 is sometimes attached to C_{6–16} alkyl polyglycoside coco glucoside too (3), also containing decyl glucoside.

Although unlikely to be rare, contact allergies to topical antiseptics used after body piercing are seldom reported. This case seems to be the first of delayed-type hypersensitivity to decyl glucoside, largely employed as a safe cosmetic ingredient. Its use in a leave-on product probably encouraged the induction of contact allergy. Because alkyl glucosides are comparable mixtures, it is likely that patients sensitive to decyl glucoside

may also react to other alkyl glucosides and vice versa.

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The positivity ratio – another parameter to assess the diagnostic quality of a patch test preparation

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Key words: false-positive reactions; patch testing; positivity ratio; quality control; reaction index.

The proportion of doubtful or irritant patch test reactions, in relation to allergic positive reactions, varies between allergens. The reaction index (RI) (1) is defined, where the number of allergic reactions is a, of doubtful reactions q, and of irritant reactions i, as $(a - q - i) / (a + q + i)$, thus ranging from -1, when all observed test reactions are doubtful

or irritant, to +1, when only allergic positive test reactions occur. An IVDK analysis showed that 13 standard series allergens had a positive RI at day 3 (D3) (2).

To some allergens, e.g. Amerchol L-101 (50% petrolatum), benzalkonium chloride (0.1% pet.), benzoyl peroxide (1% pet.), cocamidopropyl betaine (1% aqueous), 1,3-diphenylguanidine (1% pet.), octyl gallate (0.3% pet.), phenyl mercuric acetate (0.05% pet.), or propylene glycol (20% aq.), the majority of positive reactions are no stronger than + according to international guidelines (3) and national criteria of the DKG (4). In most cases with a + reaction to such allergens, no clinical relevance can be found (5–9). Thus, these reactions probably have to be regarded as false-positive, irritant reactions. Whereas, if morphology alone was considered, these reactions would commonly be regarded as allergic. Similarly, contact allergy to one of its components can be detected in only 39% of patients with a + reaction to fragrance mix (8% pet.), but in 69% of those with a ++ and in 93% of those with a +++ reaction (10). Patients with an irritant reaction to sodium lauryl sulfate (SLS) 0.5% aq. had significantly more weak positive reactions to several allergens of the standard series and a vehicle and preservative series (11), lending still further support to the concept, well known among experienced patch testers (12, 13), that + reactions are not always allergic.

Against this background, we propose the positivity ratio (PR) as an additional measure of the diagnostic quality of a patch test preparation. The PR is the percentage of + reactions among the total of positive reactions (i.e. +, ++, or ++++) observed, and can be defined as:

$$(n_{(+)} * 100) / n_{(++++)}$$

Asymptotic or exact confidence limits can additionally be computed for this binomial proportion to quantify precision.

Based on patch test data of the IVDK for the years 1999–2001 ($n=28,138$ patients), we analysed the PR and RI of 7 selected allergens from the standard series, as well as of the above-mentioned problematic allergens. Patch testing was performed according to the guidelines of the DKG (4). Allergens were purchased from Hermal, Reinbek, Germany. Test reactions at D3 were included in this data analysis.

The results are shown in Table 1. With the exception of formaldehyde, the PR of the 7 standard allergens ranged from 48% to 72%. In contrast, with the exception of Amerchol L-101, the PR of the problematic allergens ranged from 82% to 94%. It therefore appears that a PR of around 80% might be regarded as the borderline between good and not so good test preparations in this analysis.

The data also show that the PR and the RI are inversely related. This correlation is statistically significant ($p < 0.0001$); the Spearman correlation coefficient was -0.920 . Except for formaldehyde, all standard allergens in this analysis have a low PR ($< 75\%$) and a high RI (≥ 0.5), while, with the exception of Amerchol L-101, all problematic allergens have a high PR ($> 80\%$) and a low RI (≤ 0). Reactions to formaldehyde and Amerchol L-101 do not quite fit into this pattern, but can be regarded as borderline cases. These test preparations are known to be moderately irritant, and in the case of formaldehyde, poor reproducibility of patch tests is well known (14).

The combination of a low RI with a high PR, such as we found with the problematic allergens, indicates

many doubtful or irritant reactions and a high proportion of + reactions. 2 interpretations are possible. If the allergen is moderately irritant with only weak sensitizing potential, and one to which few patients are exposed, it is probably being tested at too high a concentration. In this case, not only the doubtful reactions, but also some of the + reactions are presumably irritant, rather than allergic. If, conversely, the allergen is a moderately strong sensitizer, to which many patients are exposed, it is possibly being tested at too low a concentration, and not only the + reactions, but also a high proportion of the doubtful reactions are possibly weak allergic reactions (15). However, it is difficult to distinguish between such irritant reactions and genuine, albeit weak, allergic reactions without resorting to the repeated open application test (ROAT) or provocative use test (PUT). These, together with serial dilution testing, are probably the best approach to resolve uncertainty about allergens with such a reaction profile of RI and PR.

In the final analysis, the assessment of a weak positive reaction as allergic or irritant in each individual case has to be made according to the patient's history and exposure and according to the clinical pattern of the dermatitis. In this respect, no schematic rules can be deduced from RI and PR, though these parameters may help to optimize patch test preparations, and may be an aid in interpreting such reactions.

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Table 1. Reaction index (RI) and positivity ratio (PR) of 7 standard allergens and 8 problematic allergens. Data of the IVDK, 1999–2001

Allergen preparation	Number of patients tested	Number of patients with positive reaction	Number of patients with doubtful or irritant reaction	PR (exact 95%-confidence interval)	RI
<i>Allergens of the standard series</i>					
Nickel sulfat 5% pet.	25,369	3,563	507	48% (47–50%)	0.8
4-Phenylenediamine base 1% pet.	25,451	1,095	334	57% (53–59%)	0.5
Thiuram mix 1% pet.	25,446	682	176	57% (53–61%)	0.6
Fragrance mix 8% pet.	25,403	3,021	945	67% (65–69%)	0.5
Potassium dichromate 0.5% pet.	25,572	1,054	387	68% (65–71%)	0.5
Balsam of Peru 25% pet. (Myroxylon Pereirae)	25,442	2,725	1019	72% (71–74%)	0.5
Formaldehyde 1% aq.	25,503	438	254	81% (77–84%)	0.3
<i>Problematic allergens</i>					
Amerchol L-101 50% pet.	17,763	1,056	456	77% (74–80%)	0.4
Benzoyl peroxide 1% pet.	4,796	485	494	82% (78–85%)	0.0
Phenylmercuric acetate 0.05% pet.	16,746	1268	2301	85% (83–87%)	–0.3
Propylene glycol 20% aq.	16,832	388	373	86% (82–89%)	0.0
Benzalkonium chloride 0.1% pet.	17,862	109	118	91% (84–96%)	0.0
Octyl gallate 0.3% pet.	16,935	598	1461	92% (89–94%)	–0.4
Cocamidopropyl betaine 1% aq.	17,324	397	611	94% (92–96%)	–0.2
1,3-Diphenylguanidine 1% pet.	6,364	162	180	94% (89–97%)	–0.1

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Photocontact dermatitis due to dexketoprofen

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Key words: arylpropionic acid; cross-sensitivity; dexketoprofen; non-steroidal anti-inflammatory drugs; photocontact dermatitis.

Case Report

A 27-year-old woman presented with an itchy lesion on her right hand after applying Enangel[®] (dexketoprofen trometamol 1.25%, Laboratory Menarini, Barcelona, Spain) for joint pain and after 2 days of sun exposure. Patch test results with the Spanish standard series were negative. Patch testing without UVA irradiation with non-steroidal anti-inflammatory drugs (NSAIDs) and dexketoprofen was negative. Photopatch testing (following irradiation with UVA 7.5 J/cm²) was positive for ketoprofen 1% pet. and dexketoprofen 1% pet. at 1 and 3 days (Table 1).

Patch and photopatch tests were done with the components of Enangel[®] (dexketoprofen, trometamol, lavender essence HBE-8028, ethyl alcohol 96% and carbomer; kindly provided by Laboratory Menarini), and only dexketoprofen was positive on photopatch testing. Controls in 6 healthy controls (patch and photopatch test to dexketoprofen) were performed with negative results.

Discussion

Photocontact dermatitis due to topical dexketoprofen has only recently been described (1). It is well known that topical use of NSAIDs frequently induces photosensitivity reactions (2, 3).

Dexketoprofen is an NSAID of the arylpropionic acid group, their chemical structures sharing common elements such as the benzoyl radical and the thiophene ring (4). Cross-reactions have been reported between ketoprofen and other arylpropionic derivatives such as ibuprofen (5), tiaprofenic acid (6), suprofen (7), piketoprofen (8) and flurbiprofen (9). But, cross-reactivity has not been demonstrated in other cases of contact dermatitis due to piketoprofen (10) or ketoprofen (11).

The photoallergic reaction to ketoprofen appears to be related to the benzophenone structure, thus explaining the cross-reaction between the arylpropionic derivatives, fenofibrate and oxybenzone (5).

In the case of contact photoallergy from dexketoprofen, fenofibrate and topicals containing benzophenones or ketoprofen are contraindicated (12). In the study performed with our NSAIDs series (Table 1), some aryl propionic derivatives did not show cross-reactivity with dexketoprofen (naproxen and ibuprofen). Other arylpropionic derivatives that do not contain the benzophenone moiety in their structure (such as naproxen) may thus still be used.

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Table 1. Results of patch test

Allergen	% Vehicle	Patch test		Photopatch test	
		D2	D4	D1	D3
Standard series		—	—	—	—
NSAIDs series					
Ketoprofen	1% pet.	—	—	++	++
Rest of NSAIDs		—	—	—	—
Constituents of Enangel [®]					
Dexketoprofen	1% pet.	—	—	++	++
	5% pet.	—	—	++	++
Other constituents		—	—	—	—

NSAIDs = non-steroidal anti-inflammatory drugs.

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Contact urticaria due to ketoprofen

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Key words: contact urticaria; cross-sensitivity; diclofenac; ketoprofen; loxoprofen; medicaments; non-steroidal anti-inflammatory drugs.

Case Report

A 24-year-old woman was seen in January 2003 to investigate the cause of contact urticaria, which had been diagnosed in 2000. Weal and flare with itching had developed over the shoulder 1 h after she had applied a poultice (Mohrus[®], Hisamitsu Pharmaceut. Co. Ltd, Osaka, Japan) containing 0.3% ketoprofen for muscle pain, following which skin lesions had spread to the whole body, with oropharyngeal swelling.

Patch tests were performed on the back with ketoprofen 1%, 5% and 10% pet., a constituent of the base, benzophenone-3 10% pet. and 2 other non-steroidal anti-inflammatory drugs (NSAIDs), loxoprofen sodium 20% and 10% pet. and diclofenac sodium 10% pet. At 1 h after application, urticarial responses appeared at the sites of ketoprofen 1%, 5% and 10% pet., loxoprofen sodium 20% and 10% pet. and diclofenac sodium 10% pet. that faded by 4 h. These substances showed no reactions in 5 normal control subjects.

Discussion

The common causes of contact urticaria are foodstuffs, topical medications, metals and various chemicals (1). In medicaments, some antibiotics, e.g. bacitracin, cephalosporins, chloramphenicol, gentamicin, neomycin, penicillin, rifamycin and streptomycin, tend to induce contact urticaria (1), whereas NSAIDs are very rare sensitizers. Ketoprofen frequently causes contact and photocontact dermatitis and photosensitivity (2, 3). Urticarial drug eruptions from ketoprofen (4), loxoprofen sodium (5) or diclofenac sodium (6) are rarely seen, and no cases of contact urticaria from these drugs are reported in the literature.

This case showed positive skin tests to ketoprofen, loxoprofen sodium and diclofenac sodium, which have 2-phenyl acetate in common between their structures (Fig. 1), suggesting cross-sensitivity between them.

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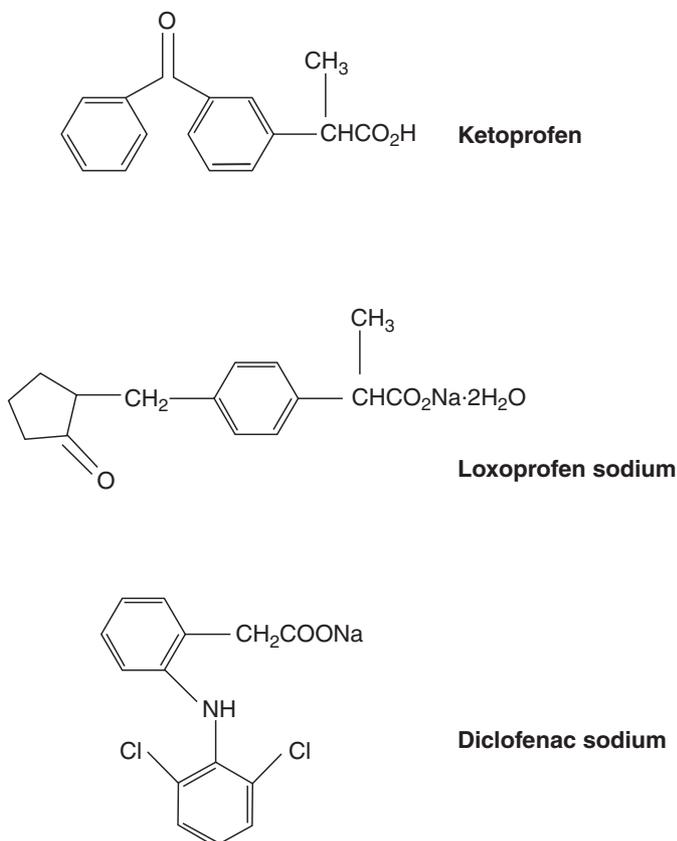


Fig. 1. Chemical structures of ketoprofen, loxoprofen sodium and diclofenac sodium.

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Contact dermatitis due to eugenol used to treat oral lichen planus

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Key words: allergic contact dermatitis; dentistry; eugenol; medicaments; non-specific oral ulceration; oral lichen planus.

Case Report

A 66-year-old man, with a history of hepatitis C viral infection and dental treatment (for more than 10 years), presented with painful erosions on the lips and buccal mucosa, which he had been experiencing for 8 years. In a dental clinic nearby, he had been prescribed topical and oral corticosteroid for this as oral lichen planus. In spite of these treatments, the erosions had gradually become exacerbated over the last 2 years and formed non-specific ulceration. A biopsy taken from the upper lip showed a loss of mucous epithelium and haemorrhage. Saw-toothed acanthosis, liquefaction and band-like lymphocytic infiltration of the superficial dermis were also noted at the wound periphery. Histological findings and the clinical history corresponded with oral lichen planus, but the formation of non-specific ulceration suggested some additional disease such as contact dermatitis. He had been prescribed an ointment for the past 2 years, containing haemodialysate from calf's blood

(Solcoseryl[®]), lidocaine hydrochloride (Xylocaine[®]) and eugenol for the purposes of ulcer treatment and local anaesthesia. We stopped the ointment, and the ulcer and pain markedly improved with 2 weeks of treatment with mometasone furoate (Fulmeta[®]) ointment. He was patch tested with a series of metals and an as-is test of the ointments that he had used. He showed a positive reaction (+) only to the ointment and eugenol (2% and 0.2% pet.) at D3.

Comment

Eugenol is yellowish oil, which is extracted from cloves and cinnamon leaves (1). Its main property is anaesthetic, and it is much used in dentistry in the form of zinc oxide-eugenol (ZOE) cement. It is well documented that eugenol has the potential to cause contact dermatitis, especially amongst dental workers (2, 3). In addition, a case of oral contact dermatitis has also been reported due to mouthwash containing eugenol (4). Eugenol is also widely used as a component of perfumes and essential oils. 27% of domestic and occupational products with aroma available in Europe contain eugenol (5). About 2.5% of patients suspected of perfume allergy have a positive reaction to eugenol on patch testing (6).

Sometimes contact sensitivity on the lips causes a lichenoid reaction, which can be diagnosed as lichen planus (7). In our case, re-biopsy after epithelialization showed the characteristic pathological findings of lichen planus. Together, based on these findings, we confirmed the diagnosis of oral lichen planus, which was mixed with and exacerbated by contact dermatitis.

While eugenol is usually used as ZOE, some dentists apply it for its anaesthetic effect for certain oral disorders such as lichen planus. Dermatologists should be aware that eugenol, widely used in dentistry, may cause oral contact dermatitis.

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Allergic contact dermatitis from 1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy) ethyl]-1H-imidazole in a water-based metalworking fluid

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Water-based metalworking fluids (MWF) frequently cause irritant contact dermatitis (1, 2), and some of their constituents, especially biocides, may induce allergic contact dermatitis (3).

Key words: allergic contact dermatitis; fungicide; imazalil; water-based metalworking fluid.

Case Report

A 53-year-old metalworker with no history of skin disorders developed eczema on his hands. In the early phase of the eczema, there was almost complete recovery after 2–3 days off work, but restarting his job resulted in recurrence after 4–5 days. Over the years, despite topical corticosteroids and emollients, his eczema worsened. For complete recovery, a period of 3–4 weeks off work became necessary. When he visited our centre, there was no recovery anymore despite 1 year off work. Further questioning revealed that a few years before there had been a change in the brand of metalworking fluid (MWF). Examination showed a chronic severe dermatitis of both hands and wrists. Patch tests were performed with the European standard series, cosmetic and metalworking series, together with 2 preparations of his own MWF. We could not obtain any information from the manufacturer of his MWF and its components. There were positive reactions to colophonium, abietic acid and his MWF (Table 1). Later, we were able to obtain the cooperation of the manufacturer of the MWF in providing information. A biocide, 4,4-methylenbismorpholine and a fungicide, 1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole (Fungamet, Janssen Pharmaceutica, Beerse, Belgium), were used. Neither ingredient had been

tested previously. Further patch tests (Table 2) were performed and gave a positive reaction to Fungamet; control testing in 4 persons being negative.

Discussion

As far as we know, 1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole (CAS no. 73790-28-0) has never before been reported as a potential allergen in MWF (2–4). The trademark name is Fungamet or Imazalil, which is a solution of 70% 1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl]-1H-imidazole in 30% monoethylene glycol. For veterinary or agriculture purposes, this is better known as enilconazole and used for dermatophytic infections of horses, cows and dogs as well as in the control of plant-pathogenic fungi (6). There is 1 previous report of contact sensitivity to enilconazole (5, 6).

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Contact allergy to Disperse Blue 106/124 mix in consecutive German, Austrian and Swiss patients

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Key words: clinical epidemiology, contact allergy; Disperse Blue 106/124; textile dye dermatitis.

Recently, we reported an increasing frequency of contact allergy to Disperse Blue (DB) 106/124 in selectively tested patients in Germany and Austria (1). Since May 2001, a mix of DB 106 and 124 (1% pet., Hermal/Trolab, Reinbek, FRG) was patch tested in consecutive patients in the centres of the German Contact Dermatitis Group (DKG) and the Information Network of Departments of Dermatology (IVDK) (<http://www.ivdk.org>) listed in the footnote. The mix was added to the standard series in a so-called 'monitor series'. Results obtained up to the end of July 2002 are presented.

Table 1. Initial positive patch test results

Test substance	D2	D4
Colophonium (20% pet.)	++	+++
Abietic Acid (10% pet.)	+++	+++
Own MWF concentrate (10% MEK)	+	++
Own MWF as used (undiluted)	++	++

MWF = metalworking fluid.

Table 2. Further positive patch test results in patient and controls

Test substance	Subject	D2	D4
Fungamet (1% eth.)	Patient	?+	+
	Control 1	–	–
	Control 2	–	–
	Control 3	–	–
	Control 4	–	–
4,4-methylenbismorpholine (0.3% aq.)	Patient	–	–
	Control 1	–	–
	Control 2	–	–
	Control 3	–	–
	Control 4	–	–

Table 1. Comparative demographic characteristics of patients reacting positively to Disperse Blue (DB) 106/124 mix (1% pet.), May 2001 to July 2002, supplemented with results of a logistic regression analysis, outcome 'DB positive' versus 'DB negative'

		Total tested (n = 3041)	Positive (n = 40)	OR (95% CI)
Male	M	35.0	20.0	0.45* (0.19–0.95)
Occupational dermatitis	O	12.2	7.5	0.80 (0.18–2.60)
Atopic dermatitis	A	17.6	17.5	1.23 (0.48–2.75)
Hand dermatitis†	H	22.9	17.5	0.44 (0.10–3.08)
Leg dermatitis†	L	10.6	10.0	0.35 (0.07–2.55)
Face dermatitis†	F	14.6	12.5	0.35 (0.07–2.52)
Age 40+	A	64.2	82.5	2.69* (1.21–6.82)

*Significant ($P < 0.05$).

†Additional sites contained in model to allow for full coding: arm, feet, head (excluding face) or neck, trunk with axillae, anogenital (as reference), 'other' and missing site – none of these sites being a significant explanatory factor.

3041 patients were patch tested with DB 106/124 mix in 13 centres. Overall, 40 patients reacted positively, the grading of reactions being similar to that in the previous study (1). The national and regional variation differed remarkably: in 3 centres, less than 0.5% positives; in 2, no reactions at all; and in the remaining 8 centres, at least 1% positives, with Basel ranking top (6.2%, $P < 0.05$ for Basel versus the remaining centres). Concomitant reactivity to *p*-phenylenediamine (PPD) hardly exceeded chance (Cohen's $\kappa = 0.051$, 95% CI = 0.00–0.11), with marked asymmetry (i.e. many more positive reactions to PPD among the discordant pairs, $P < 0.0001$, exact McNemar's test) very similar to the previous analysis.

Logistic regression analysis of important patient characteristics (MOAHLFA index (2)) as potential risk factors for positive reactivity to DB 106/124 showed significantly increased risk in females and patients aged 40 or older ($P < 0.05$), although the power of the study was limited by its small size (Table 1). Model fit, as assessed with the Hosmer & Lemeshow test, was excellent ($P = 0.903$).

In conclusion, contact allergy to DB 106 and 124 appears frequent enough in consecutive patients to warrant further investigation. In some countries, high proportions of sensitized patients have been reported (3, 4), in line with our own observations from Basel, Switzerland. However, the degree of exposure is largely unknown; possibly, imported clothing poses a particular risk (5). Taken together, these facts sufficiently support the current inclusion of the DB mix as a valuable screening agent (6) in the European standard series.

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Nickel-induced angular cheilitis due to orthodontic braces

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Key words: angular cheilitis; nickel; orthodontic brace.

Case Report

A 12-year-old boy presented with a 10-month history of persistent soreness and splitting in the angles of the mouth. He denied lip licking or use of any cosmetic preparations on his face or lips and had not changed his toothpaste recently. The only relevant past medical history was the insertion of metallic orthodontic braces, 2 months prior to the onset of the rash. There was no personal or family history of atopy. On examination, an angular cheilitis was evident, with some fissuring associated with it. There was no eczema

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around the mouth and no evidence of gingivostomatitis or erosions of the oral mucosa. Bacterial and mycological swabs from the angles of the mouth were negative. The condition had failed to respond to a variety of emollients, topical corticosteroids and antifungals prescribed by the general practitioner (GP). A clinical diagnosis of nickel-induced angular cheilitis due to orthodontic braces was made.

He was patch tested with the European standard series and his toothpaste. There was a strong positive reaction to nickel sulfate on day 2 (D2) ++ and D4 +++. No reactions were seen with the remaining patch tests. This confirmed our clinical suspicion of nickel-induced angular cheilitis due to orthodontic braces. Removal of the braces resulted in complete remission of the angular cheilitis. Follow-up over a period of 18 months has shown no recurrence of the condition.

Discussion

Angular cheilitis is an eczematous eruption of the skin and contiguous labial mucous membrane at the angle of the mouth. A variety of aetiological factors have been described, including candidosis, bacterial infections, mechanical irritation, nutritional deficiencies and conditions associated with hypersalivation, which causes maceration and inflammation. Atopic and seborrhoeic eczema can also be associated with this condition (1). Our patient had none of these other aetiological factors, and swabs ruled out the possibility of an infective cause.

A variety of metals are used in orthodontic braces and wires. Stainless steel is the most frequently used alloy and consists of chromium, nickel, molybdenum, iron, carbon, silicon and manganese (2). Even though nickel and other metals used in dentistry have been known to cause oral lichenoid reactions (3), nickel sensitivity is more commonly associated with a generalized skin eruption including urticaria and eczema (4). Contact cheilitis and stomatitis have been reported from nickel (5, 6). However, the occurrence of angular cheilitis following contact sensitivity to nickel in braces has not been reported. Nickel is released from such metallic devices by contact between saliva (5) and orthodontic braces. Subsequent

stagnation of saliva in the angles of the mouth, a factor more likely in patients wearing braces, makes this a probable site of sensitization, resulting in angular cheilitis. We suggest patch testing in patients wearing braces presenting with angular cheilitis. Removal of the braces or substitution of nickel with vitallium can be suggested in those found to be allergic to the metal.

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Allergic contact reactions to dental gold

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Key words: allergic contact; dental patients; dermatitis; gold alloys; lichenoid eruptions.

Case Report

A 62-year-old, non-smoking, full-time housewife, with no family or personal history of atopy, presented with a lichenoid intra-oral reaction to an 11-unit fixed gold-acrylic restoration inserted 7 years earlier, in 1993. Within a short time after insertion, she developed angular cheilitis and complained of temporomandibular joint pain, oral muscular pain, oral dryness and perioral dermatitis. In 1994, this progressed to eyelid dermatitis. Patch testing with the European standard series was negative. The patient's discomfort continued, and in 1997 the symptoms resembled those of temporomandibular disorders (TMDs). The patient had white lesions bilaterally on the buccal mucosa. A biopsy confirmed lichen planus.

In 1998, patch testing with a dental screening series (Chemotechnique Diagnostics, Tygelsjö, Sweden) showed positive reactions to gold sodium thiosulfate 2% (pet.) (++) and potassium dicyanoaurate 0.1% aq. (++) at day 3. However, the patient had, at that time, no signs of contact stomatitis, and the clinical relevance of the positive tests was questioned. She had no reactions to her gold ring.

In February 2000, the patient showed lichenoid reactions bilaterally on the buccal mucosa (Fig. 1). She complained of a burning sensation, metallic taste and dryness in the oral cavity and throat. As a tentative treatment, the fixed gold/acrylic partial denture was removed, as were also 3 single gold crowns. They were replaced with fixed restorations in titanium/ceramic. Intra-radicular gold posts were not removed because of the risk of complications (e.g. root fracture). After removal of the gold restorations, the lichenoid reaction healed within the next few weeks. The oral dryness was reduced, and the patient had no complaints of oral discomfort (Fig. 2).

Discussion

During recent years, there has been increasing interest in allergic reactions

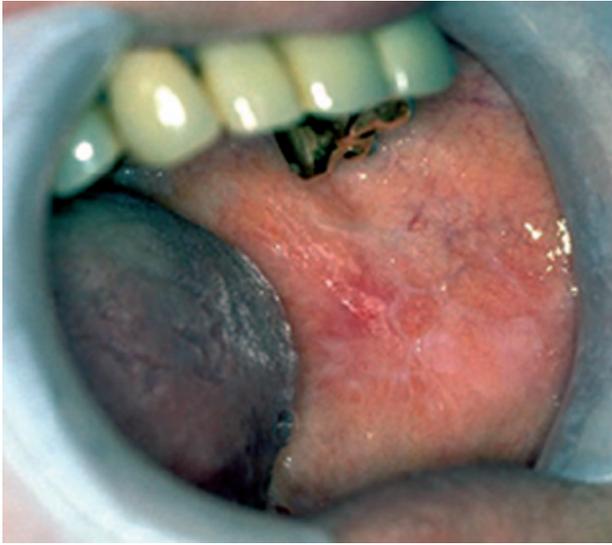


Fig. 1. Reactions to gold restorations on the buccal mucosa.



Fig. 2. Buccal mucosa 8 months after removal of gold restorations.

to gold compounds (1–3). An overrepresentation of gold allergies has been found among patients with dental restorations containing gold (4, 5). However, in many cases, the clinical relevance of a positive patch test has been uncertain. Thus, it has been suggested that gold sodium thiosulfate should not be included in the standard series but applied only

when allergic contact dermatitis from gold is suspected or for research purposes (6). Ahlgren et al. (7) found a statistically significant positive correlation between the amount of dental gold restorations and contact allergy to gold.

In the present case, the patient was exposed to a relatively large number of tooth surfaces restored with gold.

The patient's recovery is an indication of gold allergy as the cause of her discomfort and of the lichenoid contact reaction of the oral mucosa. The patient acquired the diagnosis of TMD, even though she had normal occlusion. It could be hypothesized that the oral pain and discomfort was in part related to the gold allergy. However, as insertion of fixed restorations is an extensive mode of treatment, removal of gold restorations is recommended only when objective signs or clinically relevant contact allergy (i.e. local reactions or dermatitis) are present.

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Allergic contact dermatitis due to propylene glycol and parabens in an ultrasonic gel

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Key words: allergic contact dermatitis; parabens; preservatives; propylene glycol; ultrasonic gel.

Case Report

A 62-year-old man developed dermatitis 24 h after an ultrasonic gel had been applied on his right leg. He had a previous history of circulatory problems and stasis dermatitis on both legs. Also, he had had occasional intolerance to some facial cosmetics. The ultrasound gel used was Aquasonic 100 transmission gel (Parker Laboratory Inc., NJ, USA). The eruption subsided in 4 days with topical corticosteroids.

Patch testing with our standard series (GEIDC) and the ultrasonic gel was positive to fragrance mix, Myroxylon Pereirae resin, paraben mix and the gel. We asked the manufacturer for the components of the gel, and they sent

us the different ingredients blinded to us, in the same concentrations as in the gel, listed as humectant and preservatives M and P. Patch tests were performed with these and were positive to all 3 at D4. 14 controls were negative. The manufacturer informed us that these components were propylene glycol, methyl paraben and propylparaben, though they refused to give us their exact concentrations. Later, it was confirmed that, in the case of the parabens, both were presented at bacteriostatic concentrations (<3%). Further patch tests with propylene glycol at several known concentrations were declined by the patient.

Discussion

Allergic contact dermatitis from ultrasonic gels is rare, considering their daily use all over the world. Most of the previously reported cases of allergic contact dermatitis were due to propylene glycol (1–4) and Euxyl K-400 (5–7). Positive patch tests with the other preservatives (methyl and propylparabens) have not been previously described in association with allergic contact dermatitis from ultrasonic gels, though imidazolidinyl urea (8) has been so reported.

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