

Persistent Photosensitivity After Allergic Contact Dermatitis to Epoxy Resin

Tiffany Kwok, MD,* Cheryl F. Rosen, MD, FRCPC,†‡ Frances J. Storrs, MD,§ Edmund Lobel, MD, FACD,|| and Joel G. DeKoven, MD, FRCPC†¶

Background: Persistent photosensitivity after allergic contact dermatitis (ACD) to epoxy resin has rarely been described.

Objective: The aim of this study was to create awareness that ACD to epoxy may be a trigger for persistent photosensitivity.

Methods: We present a series of 5 patients who developed ACD to epoxy resin and later a photodistributed eczematous eruption when exposed to sunlight, with a documented decrease in minimal erythema dose to UVA and UVB.

Results: The age of patients ranged from 34 to 71 years, and there were 3 men and 2 women. Each patient had occupational exposure to epoxy. Symptoms of epoxy ACD preceded photosensitivity by 5 months to 12 years in 3 cases and occurred simultaneously in 2 cases. Patch testing to epoxy resin was positive in all patients. Phototesting revealed a decreased minimal erythema dose to UVA and UVB in each of the 3 patients who were phototested. Photopatch testing was positive for epoxy resin in 1 of the 2 patients tested. All patients remained photosensitive for at least 2 years after diagnosis, with only 1 case of photosensitivity resolving with extended avoidance of epoxy.

Conclusions: There is an association between ACD to epoxy resin and development of persistent photosensitivity. Possible mechanisms to explain the relationship between the 2 phenomena are discussed.

EPOXY RESINS are commonly used in the workplace because of their physical strength, strong adhesive properties, ability to resist other chemicals, low shrinkage, and ease of curing.¹ Epoxy resin is most commonly found in both industrial and personal use adhesives and glues. It may also be found in surface coatings such as paints and primers, electrical insulation, and in polyvinyl chloride products.¹⁻⁴ It is thus important to recognize the role of occupational exposure to epoxy.

An epoxy resin is formed from the chemical reaction between uncured epoxy resin, such as bisphenol A, and epichlorohydrin (3-chloro,1,2-epoxy propane; chloromethyloxirane). This results in the attachment of 2 glycidyl groups of bisphenol A, creating a diglycidyl

ether of bisphenol A. An epoxy resin system is a polymer formed by combining the epoxy resin with curing agents such as amine and amide hardeners and additives such as plasticizers, diluents, solvents, fillers, and pigments.⁴ Choice of the curing agents and additives helps determine durability and resistance of the final epoxy resin system. Each component of the epoxy resin system may act as a potent sensitizer and cause irritant or allergic contact dermatitis (ACD) before it is cured. Epoxy resin is a common contact allergen showing positivity in up to 1.8% of patients patch tested.⁵

Persistent photosensitivity with a decrease in the minimal erythema dose (MED) to UVB and UVA after ACD to epoxy resin has rarely been reported. Allen and Kaidbey⁶ first described this phenomenon in 1979 in a group of pipefitters with occupational exposure to epoxy resin. We present a series of 5 patients with ACD to epoxy resin associated with persistent photosensitivity and discuss possible mechanisms of association between the 2 entities.

CASE 1

A 40-year-old female train car painter with longstanding well-controlled ACD to epoxy resin presented to the occupational contact dermatitis clinic in 2007 with a flare of her ACD secondary to inadvertent workplace exposure. Since her diagnosis 12 years before, she had avoided occupational exposure to epoxy-based paints at work by becoming a welder in a different area of the factory. Unfortunately, she developed a severe dermatitis on her face and

From the *Division of Dermatology and Cutaneous Sciences, University of Alberta, Edmonton, Alberta; †University of Toronto; ‡Division of Dermatology, Toronto Western Hospital, Toronto, Ontario, Canada; §Department of Dermatology, Oregon Health and Science University, Portland, OR; ||Skin and Cancer Foundation Australia, Sydney, New South Wales, Australia; and ¶James R Nethercott Occupational Disease Specialty Program, St. Michael's Hospital, Toronto, Ontario, Canada.

Address reprint requests to Joel G. DeKoven, MD, FRCPC, James R Nethercott Occupational Disease Specialty Program, St. Michael's Hospital, 30 Bond St, 4th Floor, Shuter Wing, Toronto, Ontario M5B 1W8, Canada.

E-mail: joel.dekoven@utoronto.ca.

The authors have no funding or conflicts of interest to declare.

DOI: 10.1097/DER.0b013e31828afadf

© 2013 by the American Contact Dermatitis Society. All Rights Reserved.

torso after a workplace airborne epoxy exposure to a train car freshly painted with epoxy paint. The patient had no other relevant medical history or family history of atopy or other skin disease and did not take any medications.

She was patch tested to the Chemotechnique Diagnostics North American Standard Series with IQ chambers (Dormer Laboratories, Inc, Rexdale, Canada; Scanpor tape, Omniderm Pharma, Vaudreuil-Dorion, Canada) to elicit other possible related allergens. She had a 2+ reaction to epoxy resin, 2+ reaction to fragrance mix, and 1+ reaction to methyl methacrylate, the latter 2 positives with unknown significance. She was not patch tested to the epoxy series because of her remote history of significantly positive patch test reactions to epoxy. Nevertheless, she developed a severe recall reaction comprised of persistent erythema involving the periorbital areas, dorsa of the hands, and right anterior shoulder in response to patch testing, which necessitated an extended period of time away from the workplace.

Upon her return to work and after a flash burn while welding, a rash first developed during the winter on photoexposed areas of the patient's head and neck, worsening when she was outdoors on days when the sun reflected off the snow. Significant exacerbations later developed in the spring and summer. The patient was noted to be Fitzpatrick skin type II before this eruption. Phototesting revealed a decreased MED to UVA and UVB, with 1+ reaction to 10 mJ/cm² UVB at 8 hours and 2+ reaction at 24 hours and a 1+ reaction to 4 J/cm² UVA at 8 hours and a 2+ reaction at 24 hours persisting at least 72 hours. Photopatch tests to the North American Standard Series (Table 1) were difficult to assess because of widespread erythema of the entire photopatch test area although the dose of UVA used was 1 J/cm². Photopatch testing to epoxy resin was not done. Anti-nuclear antibodies and extractable nuclear antigens were negative, and anti-dsDNA was weakly positive at 40 IU/mL.

We advised photoprotection with clothing, hats, sunscreen and sun avoidance, Mylar filter treatment of her car's windows, and work restrictions. The patient also underwent a trial of Plaquenil (Sanofi-Aventis Canada, Inc., Laval, Quebec) for her photosensitivity with no effect. Azathioprine led to an improvement in the photosensitivity but had to be discontinued because the medication was not tolerated. She uses clobetasol propionate cream for the eruption on her body, tacrolimus 0.1% ointment for her face, and hydroxyzine for pruritus. Unfortunately, this photosensitivity persists over 4 years after diagnosis and only improves upon spending continuous time indoors for several weeks.

CASE 2

A 35-year-old female plastics bench mechanic presented to the dermatology clinic in 1992 with a 4-year history of ACD to epoxy. Her ACD started 3 months after becoming a plastics bench mechanic and manifested as dermatitis on the sides of her fingers, persisting despite altered work duties which minimized exposure to epoxy. Five months later, she developed numerous episodes of acute facial swelling and a rash on the neck and dorsal arms ending

TABLE 1. Photopatch Series Used for Patient 1

1. Parsol MCX (octyl methoxycinnamate)
2. Benzophenone-4
3. Thiourea
4. Dichlorophene
5. Triclosan
6. Hexachlorophene
7. Chlorhexidine diacetate
8. Sandalwood oil (Indian)
9. Chlorpromazine hydrochloride
10. PABA 4-amino benzoic acid in 5% alcohol
11. Petrolatum
12. 3,4,5 Tribromo salicylanilide
13. Octyl dimethyl PABA in 5% alcohol
14. Benzophenone-3
15. Promethazine hydrochloride
16. Octyle dimethyl PABA in 5.0% pet
17. Bithionol
18. PABA 4-aminobenzoic acid in 5.0% pet
19. Fentichlor
20. Octyl salicylate
21. Butyl methoxydibenzoylmethane (Parsol 1789)
22. Homosalate
23. Methylantranilate
24. 6-methyl coumarine
25. 4-methylbenzyliden camphor
26. Octocrylene
27. Control

PABA indicates para aminobenzoic acid.

abruptly at the wrist and V of the neck, requiring 4 visits to the emergency department. Personal exposures included facial cosmetics and nail polish. Medical history was significant for hypertension treated with a thiazide diuretic.

Numerous biopsies of affected skin on the neck revealed spongiotic dermatitis with dyskeratotic keratinocytes, parakeratosis, neutrophils in the stratum corneum, prominent vacuolar change along the dermal-epidermal junction thought to represent secondary impetiginization, and a chronic inflammatory infiltrate, comprised of primarily lymphocytes and some melanophages in the dermis obscuring the dermal-epidermal junction.

The patient was patch tested to the North American Standard series, the corticosteroids, plastic and glue derivatives [including bisphenol A 1% in petrolatum (pet; 4,4' isopropylidenediphenol)], acrylates, and fabric finishes trays (Chemotechnique Diagnostics, Malmo, Sweden), and the patient's own products. Patch testing revealed a 1+ reaction to epoxy resin after 48 hours, increasing to 2+ after 7 days, and 2+ reaction to tosylamide formaldehyde resin after 48 hours and 7 days. Her ACD to tosylamide byproducts, often found in nail polish, possibly partially explained her eruption on her hands, sides of the face, and neck. Phototesting revealed a UVB MED of less than 10 mJ/cm² and UVA MED of 0.5 J/cm². Photopatch testing with the North American standard series (Table 2) and bisphenol A 1% in pet (4,4' isopropylidenediphenol) were irradiated with 0.5 J/cm² UVA and found to be negative. Photopatch

TABLE 2. Photopatch Series for Patient 2

1. Amyl dimethyl PABA 5% in pet
2. Sulisobenzene (BZP-4) 10% in pet
3. Cinoxate 1% in pet
4. Thiourea 0.1% in pet
5. Dichlorophen 1% in pet
6. Triclosan 2% in pet
7. Hexachlorophene 1% in pet
8. Chlorhexidine diacetate 0.5% in H₂O
9. Sandalwood oil
10. Chlorpromazine hydrochloride 0.1% in pet
11. Musk ambrette 1% in pet
12. Para-aminobenzoic acid 5% alcohol
13. Petrolatum control
14. Tribromosalicylanilide 1% in pet
15. Octyl dimethyl PABA 5% in alcohol
16. Oxybenzone (BZP-3) 3% in pet
17. Promethazine 1% in pet
18. Octyl dimethyl PABA 5% in pet
19. Bithionol 1% in pet
20. Para-aminobenzoic acid 5% in pet
21. Musk ambrette 1% in alcohol
21. Fenticlor 1% in pet
23. Amyl dimethyl PABA 5% in alcohol
24. Butyl methoxydibenzoylmethane 5% in pet
25. 6-Methylcoumarin 1% in alcohol
26. Coal tar 5% pet
27. 3,5, Dibromosalicylanilide 0.5% pet
28. 4,5, Dibromosalicylanilide 0.5% pet
29. Oak moss (synthetic) 1% pet
30. 5-Monosalicylanilide 0.5% pet
31. 3,4,4-trichlorocarbaniide 0.5% pet
32. Benzophenone 2 1% pet
33. Sulfathiazole 5% pet
34. Sulfisoxazole diolamine 5% pet
35. Potassium dichromate 0.5% pet
36. Cobalt chloride 2% aq
37. Lichen mix 16.0%
38. Octocrylene 1% pet
39. Atranorin 0.5% pet

AQ indicates aqueous; PABA, para aminobenzoic acid.

testing to epoxy resin 0.01%, 0.1%, and 1% pet showed an allergic reaction before and after irradiation, 72 hours after application. Anti-nuclear antibodies was negative, and anti-dsDNA was normal.

The patient was advised on sun safety, told to avoid thiazide diuretics, and given job restrictions to work indoors and avoid exposure to epoxy resin. Her hypertension was subsequently treated with nifedipine. Eighteen years later, her dermatitis persists in the same sun-exposed areas despite avoidance of sunlight and change in occupation to volunteer work at a food bank.

CASE 3

A 71-year-old male golf club maker presented to the dermatology clinic in 1994 with a 4-year history of a dermatitic

eruption on the face sparing the periorbital areas and mouth, posterior hairline, neck, V-shape down his anterior chest, bilateral forearms, and shins. This eruption flared when exposed to sunlight. Exposures during golf club making and repairing included epoxy, solvents, rubber grips, and glues. The patient had no relevant medical history or family history of atopy or other skin disease. He was not on medications but had taken amitriptyline in the past.

Multiple biopsies of the scalp, neck, and hand revealed psoriasiform epidermal hyperplasia with thickening of the papillary dermis with dense collagen bundles oriented perpendicular to the surface of the specimen and a lymphohistiocytic infiltrate around the superficial vessels. There were focal areas of slight spongiosis suggesting an underlying spongiotic dermatitis but no evidence of actinic reticuloid. The patient was patch tested to the North American Standard Series and plant trays (Chemotechnique Diagnostics) and photopatch tested to the North American Standard tray (Chemotechnique Diagnostics). Patch testing revealed a 2+ reaction to epoxy, 1+ reaction to carba mix, 1+ reaction to cobalt chloride, and 1+ reaction to propylene glycol at 48 hours and 7 days. Phototesting showed a decreased MED to UVA of 0.5 J/cm² and decreased MED to UVB of 6 mJ/cm². Extended UVA testing to 0.1, 0.2, 0.3, 0.4, and 0.45 J/cm² were all negative at 24 hours. Photopatch testing used the North American Standard Series (Table 3), irradiated with 0.45 J/cm² of UVA at 48 hours. The patient's dermatitis flared significantly after irradiation. Photopatch reading 24 hours after irradiation revealed 1+ chlorpromazine hydrochloride 0.1% pet and 1+ promethazine 1% pet. Delayed photopatch reading at 5 days after irradiation revealed 1+ epoxy, 1+ carba mix, 1+ oxybenzone 3% pet, 1+ promethazine 1% pet, and 1+ chlorpromazine hydrochloride 0.1% pet. The aforementioned reactions to photopatch testing were equivocal because phenothiazines are commonly known to be phototoxic, and none were deemed relevant. Laboratory examination displayed a normal complete blood count, negative rheumatoid factor, anti-nuclear antibodies, and normal IgG and porphyrin levels.

This patient unfortunately flared significantly during patch testing. He was initiated on a psoralen plus ultraviolet light therapy and prednisone protocol (Table 4)⁷ typically used for those who have ACD to sesquiterpene lactone and photosensitivity. Adverse effects experienced from this treatment included fatigue, nausea, and skin pain on his abdomen and lateral left shoulder. However, his skin cleared after undergoing this protocol. He was able to go golfing in Hawaii with significant sun exposure and continue repairing golf clubs and contacting epoxy and his skin did not flare. A few years later, the patient developed a relapse of his photodermatitis that did not clear with prednisone and psoralen plus ultraviolet light therapy. He was subsequently lost to follow-up.

CASE 4

A 46-year-old male spray painter presented to the dermatology clinic with a 1-year history of dermatitis on his forehead and cheeks, which developed 3 months after commencing his job. The spray paint used at work was a zinc-based, anti-rust epoxy paint

TABLE 3. Photopatch Series for Patient 3

1. Octyl methoxycinnamate 7.5% in pet
2. Sulisobenzene (BZP-4) 10% in pet
3. Cinoxate 1% in pet
4. Thiourea 0.1% in pet
5. Dichlorophen 1% in pet
6. Triclosan 2% in pet
7. Hexachlorophene 1% in pet
8. Chlorhexidine diacetate 0.5% in H₂O
9. Sandalwood oil
10. Chlorpromazine hydrochloride 0.1% in pet
11. Musk ambrette 1% in pet
12. Para-aminobenzoic acid 5% alcohol
13. Petrolatum control
14. Tribromosalicylanilide 1% in pet
15. Octyl dimethyl PABA 5% in alcohol
16. Oxybenzone (BZP-3) 3% in pet
17. Promethazine 1% in pet
18. Octyl dimethyl PABA 5% in pet
19. Bithionol 1% in pet
20. Para-aminobenzoic acid 5% in pet
21. Musk ambrette 1% in alcohol
22. Fenticlor 1% in pet
23. Octyl salicylate 5% in pet
24. Butyl methoxydibenzoylmethane 5% in pet
25. Homosalate 5% in pet
26. Methyl anthranilate 5% in pet
27. 6-Methylcoumarin 1% in alcohol
28. Octocrylene 20% in pet
29. Coal tar 5% pet
30. 3,5, Dibromosalicylanilide 0.5% pet
31. 4,5, Dibromosalicylanilide 0.5% pet
32. Oak moss (synthetic) 1% pet
33. 5-Monosalicylanilide 0.5% pet
34. 3,4,4-trichlorocarbaniide 0.5% pet
35. Benzophenone 2 1% pet
36. Sulfathiazole 5% pet
37. Sulfisoxazole diolamine 5% pet
38. Potassium dichromate 0.5% pet
39. Cobalt chloride 2% aq
40. Lichen mix 16.0%
41. Octocrylene 1% pet
42. Atranorin 0.5% pet

PABA indicates para aminobenzoic acid.

used indoors. Clothing worn at work included long-sleeved overalls with a hood, a dust mask covering his nose and mouth, later switched to a full face mask, and heavy-duty vinyl gloves. This dermatitis cleared while off work but recurred within 1 to 2 days of return to work. He was no longer using epoxy paint but working in the same indoor factory environment as other spray painters using epoxy paint. The following spring (7 months after he developed his original eruption), the patient noted aggravation of his facial dermatitis when exposed to sunlight either directly or through window glass. Subsequently, he also noted irritation of his facial skin on exposure

to sunlight even when the skin had been normal at the time of sun exposure. Medical history, past medications, and family history were noncontributory.

Patch testing to the TRUE Test standard series (Mekos Laboratories, Hillerod, Denmark) revealed a 2+ reaction to epoxy resin mix at 48 hours and 7 days. Photo and photopatch testing were declined by the patient. Unfortunately, 1 1/2 years after the patient's last exposure to epoxy paint, he was still highly photosensitive and working at night as a taxi driver.

CASE 5

A 34-year-old male spray painter presented with a 3-month history of dermatitis on his face, neck, and dorsal surfaces of his hands and wrists with aggravation in sunlight. Occupational exposure included outdoor painting of galvanized metal towers for electricity cables with epoxy paint. The patient had worked in his present job as a spray painter for 1 month before his dermatitis developed. Clothing worn at work consisted of trousers, a long sleeved shirt, and safety helmet. Gloves consisting of fabric upper and rubber palms were supplied on an irregular basis, and he frequently worked with no hand protection. The patient had no relevant medical history or family history of atopy or other skin disease and did not take any medications. The patient did not describe a history of contact with exogenous photosensitizers.

Patch testing to the TRUE Test allergens (Mekos Laboratories, Hillerod, Denmark) and Chemotechnique epoxy series (Table 5; Chemotechnique Diagnostics) revealed a 2 to 3+ reaction to epoxy

TABLE 4. PUVA Protocol Used in Case 3⁷

- Oxsoralen, 0.3 mg/kg 1.5 hours before treatment time. If the patient begins to flare, the UVA dosage proceeds, but the prednisone dose is increased to the original controlling dose. If the patient is doing well, the UVA dosage is increased by 0.5-1 J at each UVA treatment visit, and the prednisone therapy is gradually decreased until it is no longer being used. Blood pressure and blood glucose are monitored. Protective, full-wrap plastic eye wear is worn on the day of UVA therapy and day after treatment if patient has cataracts.
- Week 1: physician determines MPD (MED while patient is taking psoralen—minimum PUVA dose). Prednisone 60 mg daily, PUVA 3×/wk, start at UVA dose 1 J less than MPD. Increase UVA by 1 J if MPD > 6 J or 0.5 J if MPD between 3 and 6 J.
- Week 2: Prednisone 40 mg, UVA 3×/wk, UVA increase by 0.5-1 J.
- Week 3: Prednisone 20mg daily. UVA 3×/wk, increase by 0.5-1 J. Stop prednisone at the end of the week if all is well.
- Week 4: PUVA 3×/wk with increase by 0.5-1 J per treatment up to a max of 10 J
- Week 5/6: begin maintenance if no flare of dermatitis—PUVA same dose as last week 4, no increasing
- Week 7/8/9: PUVA 2×/wk at last dose
- Week 10: If clearing persists, continue 1 PUVA treatment a week during the summer. Stop during winter if patient is clear in winter; otherwise, modify maintenance to maintain control of dermatitis. If there is a flare, resume prednisone and PUVA 3×/wk.

PUVA indicates psoralen plus ultraviolet light therapy.

TABLE 5. Chemotechnique Epoxy Patch Test Series Used for Patient 5

1. Hexamethylenetetramine 2.0% pet
2. Diaminodiphenyl methane 0.5% pet
3. Triethylene tetramine 0.5% pet
4. Phenylglycidyl ether 0.25% pet
5. Isophorone diamine 0.1% pet
6. Cycloaliphatic epoxy resin 0.5% pet
7. Ethylenediamine dihydrochloride 1.0% pet
8. Dimethylaminopropylamine 1.0% aq
9. Bisphenol F 0.25% pet

resin in the standard series but not to any of the Chemotechnique epoxy series. The patient declined photopatch testing because of the discomfort he experienced with the strong positive patch test reaction.

The patient ceased work, and his dermatitis gradually resolved over the subsequent 6 weeks. However, recurrent dermatitis on skin exposed to sunlight continued for a further 1 1/2 years followed by a gradual decrease in the severity of the reaction to sun exposure. Fortunately, his photosensitivity finally ceased 2 years after his last contact with epoxy.

DISCUSSION

These 5 cases demonstrate an association between ACD to epoxy resin and persistent photosensitivity. The ages of patients affected in this case series range from 34 to 71 years, with 3 men and 2 women involved. Epoxy exposure occurred through the patient's occupation in all patients rather than through personal exposures. Three patients were painters, 1 made and repaired sports equipment, and 1 was a mechanic. Symptoms of epoxy ACD preceded photosensitivity by 5 months to 12 years in 3 cases and occurred simultaneously in 2 cases. All patients remained photosensitive for at least 2 years after diagnosis, with only 1 case of photosensitivity resolving with extended avoidance of epoxy. Table 2 shows compiled demographic data and patch test results. Patch testing was positive to epoxy resin in all patients. Photopatch testing was positive to epoxy resin in 1 of the 2 patients who were tested.

Minimal erythema dose is the smallest amount of UV radiation required to produce erythema at an exposed site 24 hours after irradiation. The dose of UVA selected for photopatch test irradiation is usually 1 J/cm² below the UVA MED. Because the UVA MED was already so low in our patients, testing was cautiously performed with the next dose below the MED.

The relationship between ACD to epoxy and persistent photosensitivity has rarely been described. Allen and Kaidbey⁶ in 1979 described 8 pipefitters working at the same gas company that used heated epoxy to 60°C, releasing fumes in the area on 1 occasion, with these exposed workers developing ACD to epoxy shortly thereafter. Two months later, 7 of the 8 workers developed a decreased MED to UVA, which clinically persisted for at least a year.⁶

TABLE 6. Demographic and Patch Test Characteristics of Cases⁷

Case	Age, y	M/F	Occupation	Patch Test Results	Duration of Epoxy ACD Before		Phototest, Photopatch Test Results	Persistence of Photosensitivity	Treatment
1	40	F	Train car painter	2+ epoxy 2+ fragrance 1+ MMA	12 y		MED UVA 4 mJ/cm ² MED UVB 10 mJ/cm ² Photopatch indeterminate	>3 y, ongoing	Avoidance of epoxy, sunlight. Failed Plaquenil.
2	35	F	Plastics bench mechanic	2+ epoxy 2+ tosylamide formaldehyde resin	15 mo		MED UVA 0.5 J/cm ² MED UVB <10 mJ/cm ² Photopatch—ve bisphenol A 0.01%, 0.1%, 1% in pet—ve	>22 y, ongoing	Avoidance of thiazide diuretics, epoxy, sunlight.
3	71	M	Golf club maker	2+ epoxy 1+ carba mix 1+ cobalt chloride 1+ propylene glycol	Concurrent		MED UVA 0.25 J/cm ² MED UVB 6 J/cm ² Numerous equivocal or suspected phototoxic reactions present. Not completed	Improved on PUVA, prednisone for >2 y but flared when stopped. He died 2 y later of unknown cause.	PUVA/prednisone protocol. (Burke)
4	46	M	Spray painter	2+ epoxy	7 mo		Declined	>1.5 y, ongoing	Avoidance of epoxy, sunlight.
5	34	M	Spray painter	2-3+ epoxy in standard series but not in Chemotechnique epoxy series	Concurrent			Resolved 2 y after last contact with epoxy	Avoidance of epoxy, sunlight.

MMA, methylmethacrylate; PUVA, psoralen plus ultraviolet light therapy; ve, positive.

Since then, the association between ACD to epoxy and persistent photosensitivity has rarely been reported.⁸ None of the 41 workers in a large case series of individuals with ACD to epoxy resin displayed symptoms suggesting photosensitivity.⁹

The diverse presentations of our patients call into question the possible mechanisms surrounding the relationship between epoxy ACD and photosensitivity. Theories postulated include epoxy resin itself directly triggering photosensitivity; however, if this were the case, we would expect the association between ACD to epoxy and persistent photosensitivity to be more commonly reported given the high prevalence of ACD to epoxy. An alternative explanation could involve ACD to a specific component of the epoxy resin system or a cross-reactant with epoxy resin acting to trigger photosensitivity. This hypothesis is favored by Allen and Kaidbey⁶ because all 8 of their patients were photopatch test positive to bisphenol A but only 4 of 8 were photopatch test positive to epoxy resin.⁶ In our case series, case 2 was the only patient photopatch tested to bisphenol A (0.01%, 0.1%, and 1% pet) but was not positive. Rose et al¹⁰ postulated that ACD may serve to up-regulate the immune response such that photoallergy or photosensitivity is more easily produced or recognized.

The association of ACD to epoxy and photosensitivity may potentially also be explained by these 2 separate phenomena coincidentally occurring together in some or all patients in this case series. This may explain the variability between timing of ACD and photosensitivity. Another explanation for this variable timing may be that the photosensitizing agent stays in the skin for a prolonged period.

Some of the other patients in our series may have had alternate reasons for photosensitivity. The patient in case 1 worked as a welder, working with equipment that emits UVC radiation. There have been a few case reports suggesting an association between UVC exposure in welders and the development of broadband photosensitivity.^{11,12} Our patient sustained a flash burn before developing photosensitivity. This likely exposed her to a very short duration of higher-than-usual UVC radiation, which could have had a small chance of contributing to her photosensitivity. In these case reports, 1 patient's photosensitivity immediately subsided after stopping work as a welder,¹¹ and 1 patient's photosensitivity began after chronic exposure to welding arcs and persisted for at least a year despite avoidance of triggers.¹² The patient in case 2 was taking a thiazide diuretic, known to be associated with photosensitivity. In this case, photosensitivity secondary to the use of thiazide diuretics cannot be ruled out. However, it would be extremely uncommon to see a decreased MED to UVA and UVB as low as this patient's with the use of a thiazide diuretic alone. In addition, thiazide-induced photosensitivity would be unlikely to persist for more than 20 years as it has with our patient.

Most patients had positive patch tests to other allergens that, in and of themselves instead of the epoxy resin, may have contributed to or caused photosensitivity. However, this phenomenon has not been reported in association with any of the patients' other positive reactions. Furthermore, none of the patients had positive patch tests

to allergens commonly associated with photosensitivity such as *compositae* mix (tested in case 1), sesquiterpene lactone (tested in cases 1 and 3) and chrysanthemum, tansy, and alantolactone (tested in case 3).

Chronic actinic dermatitis (CAD) is a photodermatosis characterized by a persistent eczematous eruption affecting sun-exposed areas, histopathology suggestive of chronic eczema, and a reduction in the MED to UVB and UVA.^{13,14} In particular, many patients have a history of other types of dermatitis such as ACD for many years preceding development of photosensitivity. This phenomenon is called *persistent light reaction*. It is most commonly described with ACD to sesquiterpene lactone^{15,16} and photo-ACD to musk ambrette.^{16,17} Allergic contact dermatitis to sesquiterpene lactone was not found in the 2 of 5 patients in our series patch tested to this allergen, nor was photo-ACD found in cases 2 and 3 patch tested to musk ambrette. Allergic contact dermatitis to epoxy has been uncommonly implicated in the development of CAD.¹⁸ Given our patients' clinical presentations along with suggestive phototesting results, some of these patients' photosensitivity may be due to the persistent light reaction phenomenon seen in CAD. Biopsies performed in cases 2 and 3 revealed spongiotic dermatitis, which may be consistent with this diagnosis. Unfortunately, biopsy specimens were not always available to support the diagnosis for our patients. A diagnosis of CAD may explain the response to psoralen plus ultraviolet light therapy in case 3 because this treatment is often used for desensitization in the treatment of CAD.¹⁹ The photosensitivity seen with CAD may resolve over time, leaving an ongoing ACD. Case 5 was the only individual whose photosensitivity resolved with avoidance of the allergen, which is not consistent with the pattern of resolution that would be expected in CAD.

This phenomenon of ACD in association with persistent light reactivity has previously been described after exposure to alantolactone,²⁰ B6,²¹ colophony,²² olaquinox,²³ sesquiterpene lactone,²⁴ halogenated salicylanilides, bithionol fenticlor, quinoxin, and sulfanilamide (the latter seen in guinea pigs).⁶ Most of the patients described in these cases concurrently tested positive on photopatch testing to the same allergen implicated in their ACD.

Because these 5 cases were drawn from 3 separate dermatology practices, a major limitation to our case series is the lack of consistency in testing chosen by each of their clinicians. Patients did not share the same selection of patch test allergens, especially to epoxy components such as bisphenol A. All patients were not phototested and photopatch tested and did not all have similar bloodwork done to rule out other causes of photosensitivity. In addition, skin biopsies were not obtained in all patients. Having these results for all patients in the case series would potentially lend strength to the explanations for the association between ACD to epoxy resin and photosensitivity suggested in this article.

These 5 cases support an association between ACD to epoxy and the development of persistent photosensitivity, although the mechanism of association remains unclear. Based on our case series, persistent light reaction after ACD appears to be the most likely

explanation. This photosensitivity may be severe and persist despite allergen avoidance and sun protection behaviors.

REFERENCES

- Amado A, Taylor JS. Contact allergy to epoxy resins. *Contact Dermatitis* 2008;58:186–187.
- Bray PG. Epoxy resins. *Occup Med* 1999;14:743–758.
- Canelas MM, Goncalo M, Figueiredo A. Contact allergy to epoxy resins—a 10-year study. *Contact Dermatitis* 2010;62:55.
- Rietschel RL, Fowler JF, eds. *Fisher's Contact Dermatitis*. Philadelphia: Lippincott Williams & Wilkins; 2001.
- Zug KA, Warshaw EM, Fowler JF Jr, et al. Patch-test results of the North American Contact Dermatitis Group 2005–2006. *Dermatitis* 2009;20:149–160.
- Allen H, Kaidbey K. Persistent photosensitivity following occupational exposure to epoxy resin. *Arch Dermatol* 1979;115:1307–1310.
- Burke DA, Corey G, Storrs FJ. Psoralen plus UVA protocol for compositae photosensitivity. *Am J Contact Dermat* 1986;7:171–176.
- Bachurzewska B, Borucka I, Ogownowski E. Occupationally induced allergic and photoallergic reactions to epoxide resins in railroad employees. *Derm Beruf Umwelt* 1988;36:125–127.
- Lee HN, Pokorny CD, Law S, et al. Cross-reactivity among epoxy acrylates and bisphenol F epoxy resins in patients with bisphenol A epoxy resin sensitivity. *Am J Contact Dermat* 2002;13:108–115.
- Rose RE, Goulden V, Wilkinson SM. The spontaneous resolution of photosensitivity and contact allergy in a patient with chronic actinic dermatitis. *Photodermatol Photoimmunol Photomed* 2009;25:114–116.
- Young E, Van Weelden H, Van Duren JA, et al. Photodermatitis due to welding. *Contact Dermatitis* 1990;23:282.
- Roelandts R, Huys I. Broad-band and persistent photosensitivity following accidental ultraviolet C overexposure. *Photodermatol Photoimmunol Photomed* 1993;9:144–146.
- Hawk JL, Magnus IA. Chronic actinic dermatitis: an idiopathic photosensitivity syndrome including actinic reticuloid and photosensitive eczema. *Br J Dermatol* 1979;17:24.
- Somani VK. Chronic actinic dermatitis—a study of clinical features. *Indian J Dermatol Venereol Leprol* 2005;71:409–413.
- Chew AL, Bashir SJ, Hawk JL, et al. Contact and photocontact sensitization in chronic actinic dermatitis: a changing picture. *Contact Dermatitis* 2010;62:42–46.
- Menagé H, Ross JS, Norris PG, et al. Contact and photocontact sensitization in chronic actinic dermatitis: sesquiterpene lactone mix is an important allergen. *Br J Dermatol* 1995;132:543–547.
- Barber KA, Cronin E. Patch and photopatch testing in chronic actinic dermatitis. *Contact Dermatitis* 1984;10:69–73.
- Beach RA, Pratt MD. Chronic actinic dermatitis: clinical cases, diagnostic workup, and therapeutic management. *J Cutan Med Surg* 2009;13:121–128.
- Roelandts R. Chronic actinic dermatitis. *J Am Acad Dermatol* 1993;28:240–249.
- Kuno Y, Kawabe Y, Sakakibara S. Allergic contact dermatitis associated with photosensitivity, from alantolactone in a chrysanthemum farmer. *Contact Dermatitis* 1999;40:224–225.
- Bajaj AK, Rastogi S, Misra A, et al. Occupational and systemic contact dermatitis with photosensitivity due to vitamin B6. *Contact Dermatitis* 2001;44:184.
- Kuno Y, Kato M. Photosensitivity from colophony in a case of chronic actinic dermatitis associated with contact allergy from colophony. *Acta Derm Venereol* 2001;81:442–443.
- Belhadjali H, Marguery MC, Journé F, et al. Allergic and photoallergic contact dermatitis to Olaquinox in a pig breeder with prolonged photosensitivity. *Photodermatol Photoimmunol Photomed* 2002;18:52–53.
- Ross JS, du Peloux Menagé H, Hawk JL, et al. Sesquiterpene lactone contact sensitivity: clinical patterns of Compositae dermatitis and relationship to chronic actinic dermatitis. *Contact Dermatitis* 1993;29:84–87.