

Cosmetic components causing contact urticaria: a review and update†

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Summary

Immediate skin reactions are common in dermatological practice, but may often be overlooked. The main objective of this article is to provide an update of the literature concerning immediate-type reactions or contact urticaria/contact urticaria syndrome caused by cosmetic ingredients in terms of immediate clinical symptoms, positive reactions following open, scratch but, most often, prick testing, and sometimes the detection of specific IgE antibodies. To this end, a selective search in different medical literature databases was performed. This resulted in a list of cosmetic ingredients causing immediate reactions, including hair dyes and bleaches, preservatives, fragrance and aroma chemicals, sunscreens, hair glues, plant-derived and animal-derived components, permanent makeup and tattoos, glycolic acid peel, lip plumper, and alcohols. Many of the reported cases, however, lack appropriate controls and detailed investigation. In conclusion, contact urticaria may occur with or without systemic symptoms, which are sometimes life-threatening.

Key words: contact urticaria; contact urticaria syndrome; cosmetics; IgE antibodies; immediate-type hypersensitivity; proteins; skin testing.

The epidemiology of immediate skin reactions or contact urticaria (CU) is not clear; although the symptoms are easily recognizable when wheal-and-flare reactions are present, milder reactions are difficult to diagnose (1, 2). Such reactions are common in dermatological practice (3, 4), but possibly underdiagnosed, with the exception of natural rubber latex. The other trigger factors have been documented on the basis of isolated cases or small patient series (5).

The main objective of this article is to provide an update of a review, which we published earlier (6), concerning CU and CU syndrome (CUS), caused by cosmetic ingredients that have provoked immediate clinical symptoms as

well as positive skin reactions following open, scratch but, most often, prick testing. Sometimes the diagnosis was also based on the detection of specific IgE antibodies.

Immediate skin reactions comprise:

- (1) Non-immunological CU (NICU), that is, CU without previous sensitization, with skin lesions generally being restricted to the site of contact, and systemic manifestations rarely being observed (1). The eliciting substances do not cause non-specific histamine release from mast cells, as antihistamines cannot inhibit such reactions. Examples of causal agents are benzoic acid, cinnamic acid, cinnamal, methyl nicotinate, and dimethylsulfoxide (7).
- (2) Immunological CU (ICU), a type I hypersensitivity reaction in a previously sensitized individual (8). This condition is probably less often observed in clinical practice than NICU, but its mechanism is better understood: the pathogenesis is identical to that of other types of immediate hypersensitivity reaction, and involves coupling of percutaneously absorbed antigens with specific IgE molecules on

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the surfaces of mast cells (9), the symptoms resulting from the release of histamine. Pre-existing conditions, such as atopic dermatitis, may favour this condition. Generalized reactions and/or extracutaneous reactions are frequent, and this is referred to as CUS.

- (3) CUS (10), first defined in 1975 by Maibach and Johnson (11). Since then, numerous cases have been reported with a broad spectrum of clinical manifestations that can be strictly limited to the areas of cutaneous contact, or present as generalized urticaria with concurrent involvement of internal organs. The four stages of the syndrome are:

- local symptoms ranging from non-specific symptoms such as itching, tingling and a burning sensation to a wheal-and-flare response restricted to the area of contact (stage 1)
- generalized urticaria following local cutaneous contact (stage 2), which also includes angioedema
- extracutaneous manifestations, which may include the respiratory (bronchial asthma or rhinoconjunctivitis), orolaryngeal or gastrointestinal tract (stage 3)
- anaphylactic reactions (stage 4).

- (4) CU of unknown origin, which comprises reactions with mixed features, or for which mechanisms and pathophysiological features are not well understood (9).

Immediate reactions usually start within 30–60 min following skin exposure, and clear completely within 24 h; however, delayed-onset reactions may appear within 4–6 h. The mechanism of these is unknown; slower skin penetration could offer an explanation. In case of an immune-mediated reaction, the measurement of specific IgE in serum is useful, if technically possible, for both small and large molecules (proteins). The basophil activation test, which is based on the demonstration by flow cytometry of CD63 expression following exposure to allergens, is still experimental (2, 12).

Methods

Different databases were searched: MEDLINE and CDESKPRO (an in-house literature database). A literature review was performed with the search terms (contact urticaria syndrome) AND (cosmetics), (contact urticaria syndrome) AND (fragrances), (persulfate)

OR (ammonium persulfate) OR (potassium peroxy-monosulfate), (contact urticaria OR systemic) AND hair dyes, (IgE OR immediate) AND cosmetics, (contact urticaria) OR (type 1), and (contact urticarial) OR (immediate).

Cosmetic components causing CU and/or CUS

Adverse reactions to cosmetics include irritant, allergic and photo-allergic contact dermatitis, and CU (13); the last of these is addressed in this review. Many of the reported cases, however, lack appropriate controls and detailed investigation. Table 1 provides a list of cosmetic ingredients causing ICU, NICU, and/or CUS, along with their CAS number, if known, and the respective literature references.

Hair care products

There are a few reports of hair dyes causing immediate-type hypersensitivity, some with anaphylaxis or respiratory symptoms: *p*-phenylenediamine (PPD) and its derivatives, such as *p*-aminophenol and *p*-methylaminophenol (26), and toluene-2,5-diamine (28). The reactions seem to occur only after oxidation by H₂O₂, and are attenuated when the antioxidant sodium sulfite is added to the mix (28). Goldberg et al. (18) identified Brandowski base (CAS no. 20048-27-5), an oxidation product of PPD, as a culprit. The natural permanent hair dye henna, derived from the leaves of the shrubs *Lawsonia alba* or *Lawsonia inermis*, is also a rare cause of rhinitis, conjunctivitis, sneezing, urticaria, and asthmatic symptoms (2). Sensitization seems to occur mainly through inhalation of henna powder dispersed in the air (34). Temporary tattoos, especially those that contain black dyes, have become extremely popular among teenagers in recent years. Most of these tattoos, in addition to hair dyes, contain PPD (156). Haluk Akar et al. (156) reported a case of a 15-year-old adolescent female who had been unaware of being previously sensitized to PPD from a black henna tattoo, with an angioedema-like reaction that occurred after her first exposure to hair dye. Delayed allergic reactions to PPD are known to have the potential to elicit severe facial oedema - thus mimicking type I immediate reactions (157).

Moreover, also direct hair dyes, for example Basic Blue 99 and Basic Brown 17 (39), and patent blue dye (36), have been causes of contact urticaria, mainly acting through occupational exposure.

Persulfate salts have a strong oxidizing action that accelerates the bleaching process and also makes the hair easier to dye (9). Currently, potassium persulfate is more frequently used than the ammonium salt, because the latter has an unpleasant odour (53). It is a potential cause

Table 1. Cosmetic components causing contact urticaria and/or its syndrome

Products and components	CAS no.	Reactions	Authors
Hair care products			
<i>p</i> -Phenylenediamine	106-50-3	ICU, asthma, angioedema, anaphylaxis	Mavroleon, 1998 (14); Calnan, 1963 (15); Edwards, 1984 (16); Temesvari, 1984 (17); Goldberg, 1987 (18); Fukunaga, 1996 (19); Belton, 1997 (20); Sahoo, 2000 (21); Wong, 2003 (22); Birnie, 2007 (23); Helaskoski, 2014 (24)
<i>p</i> -Aminophenol/ <i>p</i> -methylaminophenol	123-30-8/55-55-0	Anaphylaxis	Nagano, 1991 (25); Oshima, 2001 (26); Uehara, 2014 (27)
Toluene-2,5-diamine sulfate	615-50-9	Anaphylaxis	Pasche-Koo, 1998 (28); Taniguchi, 2000 (29)
<i>N,N'</i> -(2,5-diamino-2,5-cyclohexadiene-1,4-diylidene) bis-1,4-benzenediamine (Bandrowski's base)	20048-27-5	Anaphylaxis	Goldberg, 1987 (18)
Henna	83-72-7/84988-66-9	ICU, conjunctivitis, rhinitis, asthmatic symptoms	Pepys, 1976 (30); Starr, 1982 (31); Cronin, 1996 (32); Majoje, 1996 (33); Bolhaar, 2001 (34); Oztas, 2001 (35); Davari, 2011 (36)
Basic Blue 99	68123-13-7	ICU, oral and auricular swelling and itching	Wigger, 1996 (37); Jagtman, 1996 (38); Davari, 2011 (36); Vanden Broecke, 2014 (39)
Basic Brown 17	68391-32-2	ICU	Vanden Broecke, 2014 (39)
Ammonium persulfate and potassium persulfate	7727-54-0/7727-21-1	NICU, asthma, anaphylaxis	Calnan, 1963 (15); Brubaker, 1972 (40); Fisher, 1976 (41); Pepys, 1979 (30); White, 1982 (42); Kellett, 1985 (43); Blainey, 1986 (44); Pankow, 1989 (45); Schwaiblmair, 1990 (46); Van Joost, 1991 (47); Parra, 1992 (48); Wrbitzky, 1995 (49); Yawalker, 1999 (50); Borelli, 1999 (51); Perfetti, 2000 (52); Roderiguez, 2001 (53); Aalto-Korte, 2003 (9); Munoz, 2003 (54); Babilas, 2005 (55); Moscato, 2005 (56); Harth, 2006 (57); Munoz, 2008 (58); Bregnhøj, 2009 (59); Becker, 2010 (60); Hoekstra, 2012 (61); Hougaard, 2012 (62)
<i>m</i> -Aminophenol	591-27-5	CUS	Tsunoda, 1993 (63)
<i>o</i> -Aminophenol	95-55-6	CUS	Tsunoda, 1993 (63)
2,4-Diaminophenoxyethanol-HCl	66422-95-5	Generalized pruritus and erythema, anaphylaxis	Nosbaum, 2012 (64)
Glyceryl monothioglycolate	30618-84-9	Type I and IV	Engasser 2000 (65)
Antimicrobial agents and preservatives			
Chlorhexidine	55-56-1	ICU, Quincke's oedema, dyspnoea, anaphylaxis	Ohtoshi, 1986 (66); Okano, 1989 (67); Evans, 1992 (68); Torricelli, 1996 (69); Thune, 1998 (70); Snellman, 1999 (71); Krautheim, 2004 (72); Garvey, 2007 (73); Nagendran, 2009 (74); Sharma, 2009 (75); Silvestri, 2013 (76); Wittczak, 2013 (77)
2-Phenoxyethanol	122-99-6	ICU, anaphylaxis	Bohn, 2001 (78); Hernandez, 2002 (79); Birnie, 2006 (80); Lujan, 2009 (81); Núñez Orjales, 2010 (82)
Polyaminopropyl biguanide	32289-58-0/27083-27-8/28757-47-3/133029-32-0	Anaphylaxis	Kautz, 2012 (83); Creytens, 2014 (84)
Sodium benzoate	532-32-1	CUNS	Munoz, 1996 (85)
<i>p</i> -Chloro- <i>m</i> -cresol	59-50-7	Angioedema, hives	Walker, 2004 (86)
Sorbic acid	110-44-1	CUNS	Rietschel, 1978 (87)
Triclosan	3380-34-5	ICU, angioedema	Özkaya, 2013 (88)
Sunscreens			
Benzophenone-3	131-57-7	Anaphylaxis	Ramsay, 1972 (89); Berne, 1998 (90); Emonet, 2001 (91); Yesudian, 2002 (92); Landers, 2003 (93); Bourrain, 2003 (94); Spijker, 2008 (95)
Fragrance components			
<i>Myroxylon pereirae</i> (balsam of Peru)	8007-00-9	CUNS	Rudzki, 1976 (96); Forsbeck, 1977 (97); Temesvári, 1978 (98); Katsarou, 1999 (99); Cancian, 1999 (100); Tanaka, 2004 (101)

Table 1. Continued.

Products and components	CAS no.	Reactions	Authors
Fragrance mix	–	CUNS	Cancian, 1999 (100); Temesvari, 2002 (102); Tanaka, 2004 (101)
Cinnamal	104-55-2	CU type C, CUNS	Diba, 2003 (103); Mathias, 1980 (104)
<i>Cinnamomum cassia</i> oil	84961-46-6	CUNS	Rudzki, 1976 (96); Rietschel, 1978 (87)
Geraniol	106-24-1	Oedema	Yamamoto, 2002 (105)
Toothpaste flavours			
Menthol	1490-04-6/2216-51-5/	ICU, rhinitis, asthma, anaphylaxis	Holmes, 2001 (106); Andersson, 2007 (107); Paiva, 2010 (108)
Mentha piperita oil	89-78-1/15356-70-4		
	8006-90-4/84082-70-2		
Carvone	6485-40-1/99-49-0/2244-16-8	Angioedema	Hansson, 2011 (109)
Plant-derived and animal derived cosmetic ingredients			
Rubber latex and musa paradisiaca fruit (banana)	9006-04-6	Anaphylaxis	Cogen, 2002 (110); Smith, 1998 (111)
<i>Aesculus hippocastanum</i> extract (chestnut)	8053-39-2	Anaphylaxis	Seitz, 2011 (112)
<i>Avena sativa</i> (oat)	84012-26-0	ICU, oral allergy syndrome	De Paz Arranz, 2002 (113); Boussault, 2007 (114); Vansina, 2010 (115)
Wheat and hydrolysates	94350-06-8/222400-28-4/70084-87-6	ICU, anaphylaxis, WDEIA CUNS, conjunctivitis, rhinitis, bronchospasm	Kousa, 1990 (116); Freeman, 1996 (117); Pasche-Koo, 1996 (118); Niinimäki, 1998 (119); Varjonen, 2000 (120); Pecquet, 2002 (121); Laurière, 2006 (122); Olaiwan, 2010 (123); Fukutomi, 2011 (124); Chinuki, 2011 (125); Hiragun, 2011 (126); Ota, 2012 (127); Chiniku, 2012 (128); Barrientos, 2012 (129); Ishii, 2012 (130); Airaksinen, 2013 (131); Leheron, 2013 (132); Yokooji, 2013 (133); Kobayashi, 2015 (134); unpublished case (Fig. 2); McFadden, 2000 (135); Varjonen, 2000 (120); Pasche-Koo, 1996 (118); Pecquet, 2002 (121); Laurière, 2006 (122); Olaiwan, 2010 (123)
Glycine soja oil	8001-22-7	Facial erythema and swelling	Shaffrali, 2001 (136)
<i>Sesamum indicum</i> seed oil (sesame oil)	8008-74-0	Generalized CUNS, anaphylaxis	Birnbaum, 1997 (137); Smith, 1998 (111); Pecquet, 1998 (138)
Fish-derived elastin	–	ICU	Nishida, 2012 (139)
Hydroxypropyltrimonium-hydrolysed collagen (Crotein Q®)	83138-06-1	ICU, angioedema, bronchospasm	Kousa, 1990 (116); Pasche-Koo, 1996 (118); Freeman, 1996 (117); Niinimäki, 1998 (119)
Citrus seed/citrus limon fruit oil	1180-71-8/8008-56-8	Anaphylaxis	Glaspole, 2007 (140)
<i>Chamomilla recutita</i> extract and <i>Mangifera indica</i> fruit (mango)	84082-60-0/90063-86-8	Oral allergy syndrome, angioedema	Rudzki, 1999 (141); Rudzki, 2003 (142)
<i>Chamomilla recutita</i> extract and <i>Tilia cordata</i>	84929-52-2	Itching and erythema, rhinoconjunctivitis	Subiza, 1990 (143); Foti, 2000 (144); Krakowiak, 2004 (145); Smith, 1998 (111)
Equae lac (mare's milk)	–	ICU	Verhulst, 2016 (146)
Hen egg	–	CUNS	Antonicelli, 2011 (147)
Honey	8028-66-8	–	Katayama, 2016 (148)
Other			
Tattoo ink	–	NICU	Lee-Wong, 2009 (149)
Glycolic acid peel	–	Anaphylaxis	Vishal, 2012 (150)
Lip plumper	–	NICU, irritant contact dermatitis	Firoz, 2009 (151)
Alcohol contact urticaria syndrome	–	Urticarial papules	Rilliet, 1980 (152); Wong, 2011 (153)
Panthenol	81-13-0/16485-10-2	CUNS	Schalock, 2000 (154)
Rouge	–	CUNS	de Groot, 1983 (155)

CU, contact urticaria; CUNS, non-specified; CUS, contact urticaria syndrome; ICU, immunological contact urticaria; NICU, non-immunological contact urticaria; WDEIA, wheat-dependent exercise-induced anaphylaxis.

of contact dermatitis, urticaria, rhinitis, and asthma, the last of these mainly by inhalation in an occupational context (6). Asthmatics seem to be particularly susceptible to developing such reactions (52). Some studies have shown specific binding of IgE to persulfates by two methods, namely, the immunospot test and radioallergosorbent test (RAST); hence, the mechanism of immediate hypersensitivity seems to be IgE-mediated, at least in some patients (9). Yawalker et al. (50) provided evidence that T lymphocytes specific for such low molecular weight compounds may be directly involved in mediating inflammatory processes in the airways, rather than only acting through induction of IgE synthesis in persulfate-triggered occupational asthma.

Antimicrobial agents and preservatives

Chlorhexidine is a biguanide topical antiseptic and disinfectant with broad antimicrobial efficacy. It is increasingly being used in instillation gels for urinary catheters, and in contact lens solutions, but also in many cosmetic products (72, 76, 158, 159), in which it may be used as a preservative or an antimicrobial agent at a concentration up to 0.3%, as set by the European Cosmetics Directive (now Regulation) (159). Urticaria following application to intact skin or mucosae, in some cases accompanied by dyspnoea, angioedema, syncope, or anaphylaxis, has been described (76), via the mucosal route at much lower concentrations than elsewhere, generally as low as 0.05% (67, 72). Polyaminopropyl biguanide (INCI; syn. polyhexanide, polyhexamethylene biguanide) is a widely used antiseptic, for example in contact lens solutions and wound dressings, but also in cosmetics (83). It has been shown to elicit IgE-mediated reactions when it is present in wound-care products and wet wipes (83, 84), and may partly cross-react with chlorhexidine (83). A positive basophil activation test result has been described (84).

Phenoxyethanol is commonly used in cosmetics, most often in combination with other preservatives such as parabens and formaldehyde releasers (82). Lujan et al. (81) reported one case of CU in a male patient, resulting from the use of an aftershave product containing phenoxyethanol. Three other cases of contact urticaria caused by cosmetics have been reported (78–80), but the presence of immunological IgE-mediated reactions could not be confirmed, as specific antibodies could not be identified (79).

Sodium benzoate decreases the amount of dental plaque at concentrations between 1% and 4%, and is a well-known cause of NICU in toothpaste (85). Figure 1 shows positive reactions occurring a few minutes following the application on a test chamber to the preservative agents benzoic acid, sodium benzoate, and sorbic acid (as



Fig. 1. Non-immunological contact urticaria caused by the preservatives benzoic acid, sodium benzoate, and sorbic acid (sodium metabisulfite remaining negative).

well as to *Myroxylon pereirae* and cinnamal), all of which are known to cause NICU (7).

p-Chloro-*m*-cresol is present in a large number of topical preparations, and is a rare cause of allergic contact dermatitis and also CU; the mechanism of this remains uncertain (86). Triclosan, which is used in cosmetics, such as soaps, shampoos, mouthwashes, and deodorants, has provoked a case of severe immunological contact/consort CU and angioedema, owing to its presence in a metronidazole cream (88).

Sunscreens

Skin reactions to sunscreen chemicals include CU, and allergic and photo-allergic contact dermatitis (93); reactions have been found to, for example, benzophenone-3 (INCI; syn. 2-hydroxy 4-methoxy benzophenone, oxybenzone), a common ultraviolet (UV) A/UVB sunscreen, the presence of which also needs to be labeled separately on the cosmetic packaging (92). CU and even contact-mediated anaphylaxis caused by benzophenones are, however, rare (92, 94). The severity of the clinical reaction depends partly on the area of exposed skin; therefore, patch testing does not necessarily elicit anaphylaxis (95). Benzophenones are also added to protect against discolouration of cosmetics (textiles and plastics) that are potentially exposed to sunlight (92).

Fragrance components and aroma chemicals

M. pereirae (balsam of Peru) and fragrance mix (FM) I (a mixture of eight fragrance components in the

baseline series) may elicit CU by both immunological and non-immunological mechanisms (100). Cinnamal, an ingredient common to both, is probably the most important causal ingredient in this context (103). Mathias et al. (104) reported a case of lip swelling following its use in a mouthwash by a patient suffering from allergic rhinitis and asthma. We observed a similar case (data on file) caused by a cinnamal-containing toothpaste and also by cinnamon in pastries. Rietschel (87) described a case of immediate hypersensitivity to *Cinnamomum cassia* oil in toothpaste (as well as to sorbic acid, a preservative present in a shampoo). Facial oedema following the application of cosmetic products containing geraniol, another constituent of FMI, was also described (105): a 20-min closed test showed a wheal response, but no delayed hypersensitivity after 24–72 h. An immunological mechanism has been suggested, because the patient had developed widespread urticaria and flare-up reactions on the face and neck by the day 3 reading of the patch test. Glaspole et al. (140) described a case of anaphylaxis after an individual had used lemon-scented soap when showering; this patient also reported laryngeal oedema, generalized urticaria and asthma within minutes after ingestion of juice prepared from whole crushed oranges, citrus seeds, peanuts, and tree nuts, which seems to be an unusual clinical phenomenon. The antibodies reacting with citrus seeds were suggested to have caused the cross-reactive immune response.

As reported by Holmes et al. (106) regarding type I allergy to mint-flavoured toothpaste, CU should be considered in cases of persistent undiagnosed cheilitis, and both prick tests and patch tests should be carried out with suspected products and allergens. The results of IgE-mediated allergy to mint or menthol include urticaria, rhinitis, asthma, and/or anaphylaxis (107, 108). *R*-carvone, the main ingredient in spearmint oil, is also present in toothpastes; it may cause delayed-type allergy resulting in cheilitis, but occasionally also immediate reactions such as angioedema of the lips appearing within minutes (109), with an open test resulting in an immediate and strong reaction to this compound.

Plant-derived and animal-derived cosmetic ingredients

It is becoming increasingly popular to apply matching hair to the scalp, thereby changing both the length and style of the hair, by the use of bonding glues that contain high concentrations of soluble (natural rubber) latex antigen. Repeated glue exposure may potentially sensitize consumers (110). Pumphrey et al. (160) recently described the anaphylactic death of a 28-year-old British fashion designer immediately following a hair

extension procedure. The patient had a history of nut allergy and inhalant atopy, and a known strongly positive prick test reaction to natural rubber latex (160). Moreover, this type of bonding glue may also be used in the application of artificial eyelashes (110).

Emollients and moisturizers are widely used in the treatment of atopic dermatitis, and a recent trend in the cosmetics industry is the use of plant protein derivatives (e.g. from soy, wheat, oat, or sesame). Oat proteins, in particular, are used because of their alleged anti-inflammatory, antioxidant and antipruritic properties. Both allergic contact dermatitis and ICU (114, 115) resulting from the use of emollient creams containing oat extract have occurred. In the latter case the patient later experienced an oral allergy syndrome when eating oatmeal-containing biscuits and bread. The diagnosis was confirmed by prick tests and enzyme-linked immunosorbent assay testing.

Generally, the route of sensitization to proteins can be gastrointestinal, respiratory, and percutaneous, although the penetration of proteins through intact stratum corneum is very limited. However, an impaired skin barrier, skin inflammation and the potential for elevated IgE levels to occur in atopic individuals are predisposing factors.

Most cases of IgE-mediated hypersensitivity to chestnut (*Castanea sativa*), a member of the Fagaceae family, have been attributed to the so-called latex-fruit syndrome, in which ingestion of, for example, avocado, kiwi, banana (the latter a potential culprit in hair conditioners, 111) and, less often, chestnut leads to urticaria and anaphylaxis in natural rubber latex-sensitized individuals. This syndrome is caused by cross-reactivity between class I chitinases with a hevein-like domain, such as Mus a 1 (banana), Pers a 1 (avocado), Cas s 5 (chestnut), and Hev b6.02 (late hevein). However, chestnut allergy may occur independently, with Cas s 8, a lipid transfer protein, as the offending allergen. With an increasing number of food proteins being included in so-called natural cosmetics, new cases may appear in the literature, such as contact anaphylaxis induced by cosmetic facial peel containing chestnut (112).

Shaffrali et al. (136) reported on a patient reacting to a cosmetic cream with delayed-type contact allergy to all dilutions of pure soybean extract, and also an immediate response to its 20% dilution, suggesting a possible type I hypersensitivity reaction. However, no allergen-specific IgE for soybean was found, and the patient had previously eaten soy without adverse reactions. It has been shown, however, that the RAST may yield a false-negative result in 27% of cases. Hence, a negative test result for IgE for

soybean does not preclude a diagnosis of type I hypersensitivity. Reports of immediate allergy and anaphylaxis caused by ingested sesame seed or sesame oil have been published. The latter is a known contact allergen in topical pharmaceutical products and cosmetics (138). Despite its widespread use, to our knowledge, only 2 cases of immediate-type reactions induced by cosmetic products have been reported (137, 138).

Protein hydrolysates of collagen, keratin, elastin, milk, wheat, almond, and silk, added to hair conditioners to 'repair' broken hair and to provide a more voluminous appearance, are also causes of CU (119), and are capable of producing reactions through a type 1 mechanism in atopic dermatitis patients in particular (135). Hydrolysed wheat proteins are also widely used in many other cosmetic products, for which several cases have been reported in the literature, including the induction of wheat-dependent exercise-induced anaphylaxis (125, 161). Wheat contains a variety of proteins, which can be divided into salt-soluble proteins of the albumin and globulin type and salt-insoluble proteins, the latter being referred to as gluten (gliadins and glutenins) (122, 125). Their widespread use in food and non-food products aggravates the risk of sensitization. New epitopes may appear during hydrolysis, or the additives used may act as allergens (120). In the case report of Pecquet et al. (121), gluten-derived products were responsible for immediate hypersensitivity through both cutaneous and oral contact. Although the primary route of sensitization is uncertain, the order of reactions in this case favours the cutaneous route. It has been shown that the hydrolysed wheat proteins composed of large polypeptide aggregates possibly induce sensitization to a greater degree than the lower molecular weight compounds (125, 162). Leheron et al. (132) even described IgE-mediated CU in a child caused by hydrolysed wheat proteins and macadamia nuts – both of which were contained in a moisturizing cream used by the mother – with probable sensitization by proxy via maternal skin contact facilitated by atopic dermatitis (132).

Figure 2 shows a 24-year-old non-atopic beautician who developed urticarial lesions on the hands (a) shortly after application of an anti-ageing solution to a client's face. A prick test (b) with the product resulted in an urticarial reaction, but prick tests gave negative results with commercial extracts of cereals, grasses, and wheat flour diluted in water; RASTs (Unicap Pharmacia, Upsala, Sweden) with wheat, grasses and gluten also gave negative results. The cosmetic manufacturer provided the ingredients of the cosmetic products, and positive prick test reactions were observed to hydrolysed wheat proteins only. Additionally, immunoblots were carried out, and

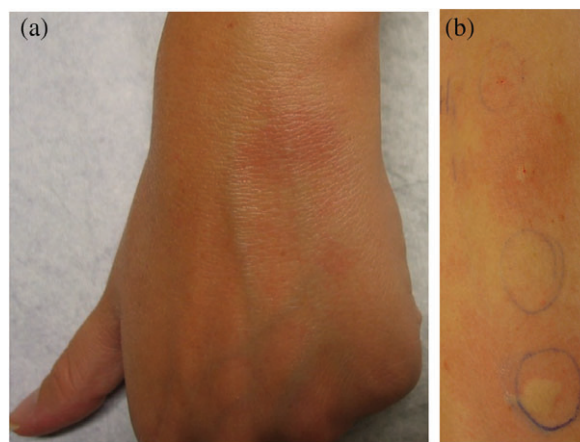


Fig. 2. Immunological contact urticaria caused by hydrolysed wheat proteins in a beautician. (a) Immediate reaction on the hands. (b) Positive prick test reaction to the hydrolysed wheat proteins. (Courtesy: case Dr S. Kerre, Aarschot, Belgium)

showed IgE antibodies in the patient's serum against various fractions of hydrolysed wheat protein.

Beside plant-, also animal-derived protein allergens in cosmetics have been reported as allergenic culprits: for example, an immediate-type reaction to a fish-derived elastin-containing cosmetic cream in a patient with a history of respiratory distress when inhaling smoke from grilled fish (139), and a case of rhinitis, choking and systemic urticaria (syndrome) following the ingestion of egg in a patient who treated her hair weekly with a homemade egg-white based mask (147). Recently, a case of ICU caused by α -lactalbumin from mare's milk-containing cosmetic cream has also been reported (146). Katayama et al. (148) reported the first case of CUS stage 3 caused by honey, induced by sensitization during skin care treatment.

A study by Niinimäki et al. (119) showed hydroxypropyl trimonium-hydrolysed collagen (stearyltrimonium hydroxyethyl-hydrolysed collagen; Crotein Q) to be an especially potent cause of immediate skin reactions, with positive prick test reactions to very low concentrations and specific IgE antibodies against Crotein Q (119).

Chamomile and mango were reported as culprits in a patient with a personal history of childhood eczema and oral allergy syndrome, with hypersensitivity to different kinds of fruit (141). Subiza et al. (143) presented 7 hay fever patients who suffered from conjunctivitis; 2 of them also had angioedema of the lids after washing their eyes with chamomile tea, a folk remedy used to treat conjunctivitis and other ocular reactions. All presented with positive prick test reactions to the tea extract; it is thought that *Matricaria chamomilla* L. [syn: *Chamomilla recutita* (L.)

Rauschert] pollens contained in these infusions were the responsible allergens (143). A similar case was reported by Foti et al. (144), also with a positive prick test reaction to German chamomile (*Matricaria chamomilla*). Moreover, a cosmetician with recurrent itching and erythematous lesions on the backs of her hands and rhinoconjunctivitis resulting from contact with depilatory wax containing *Tilia cordata* and *Matricaria chamomilla*, with positive prick test reactions to these flowers, has been described (145). One case of CUS caused by multiple components in a cosmetic skin mask was reported by West and Maibach (163); immediate open testing showed an extensive wheal-and-flare reaction to whole egg, and to *Melissa* sp. extract 1% in physiological saline. Neither passive transfer nor a RAST was performed to clarify which of the above was the most likely culprit.

Permanent makeup and tattoos

Tattoos and permanent makeup are becoming increasingly prevalent in Western society. Lee-Wong et al. (149) reported a case of anaphylaxis with an immediate skin reaction to purple and blue ink. Unfortunately, many tattoo ink manufacturers are not required to print ingredients on their labels, making it difficult to identify the actual culprit.

Glycolic acid peel

The various complications of chemical peeling that can occur are post-inflammatory hyperpigmentation, infections, scarring, allergic reactions, milia, persistent erythema, and textural changes. Vishal et al. (150) reported a case of CU limited to the area of contact with the glycolic acid peel in a patient with a history of severe acne vulgaris. Apparently, there had been no prior contact with such products in the past.

Lip plumper

New topical agents on the market are designed to increase lip volume within minutes to days following their application; the mechanisms by which they act include vasodilatation secondary to either irritant contact dermatitis or NICU. Common ingredients are essential oils of cinnamon and cayenne pepper (*Capsicum frutescens*), that is, spices that are classified as both irritants and urticants. Firoz et al. (151) reported a case of a young boy who developed an urticarial plaque of rapid onset on the right cheek following a kiss from his mother

after she had applied a lip plumper 1 h earlier; the active ingredients of this included benzyl nicotinate and *C. frutescens* resin.

Alcohol urticaria syndrome (AUS)

Angioedema following ingestion of alcohol may be caused by different agents contained in the beverages, such as yeast. AUS is a rarely reported and poorly understood entity that may be triggered either by primary alcohols or alcohol metabolites, that is, aldehydes and acetic acid (153, 164), and that seems to be more common among persons of East Asian descent who have aldehyde dehydrogenase deficiency, leading to increased serum aldehyde levels in the course of alcohol metabolism (153). Case reports of CU caused by local application of alcohol seem to be very rare, and include the following examples: a patient who noted a diffuse pruritic rash after drinking alcoholic beverages, with ethanol applied to the skin provoking an erythematous reaction in ~20 min (165); allergic CU caused by ethanol and isopropyl alcohol (166); urticaria-like lesions provoked by ethanol and stearyl alcohol, associated with delayed dermatitis (167); and a nurse with ethanol-induced CU following the application of perfume and a hand sanitizer, who developed generalized reactions after drinking ethanol (153). Rilliet et al. (152) reported a case in which immediate reactions with most of the primary alcohols were positive, and there was a positive passive transfer test result, with a method corresponding to that of Prausnitz-Küstner (152, 153).

Conclusion

This updated review confirms that various cosmetic components can cause CU with or without systemic symptoms, the latter sometimes being life-threatening. However, such cases might be more common, because patients probably lack awareness. Physicians should therefore continue to look out for possible new causes. Anaphylactic reactions provoked by patch testing with the allergen are rare, but, in patients with a severe history of CUS, emergency measures remain necessary.

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