

# Immunologic Contact Urticaria

John McFadden, DM, FRCP

## KEYWORDS

• Contact urticaria • Atopic • Food • Cosmetics

## KEY POINTS

- The wheal and flare reaction from direct contact with a chemical or protein agent is known as *contact urticaria*.
- Contact urticaria can be either nonimmunologic (no sensitization required) or immunologic (sensitization required).
- There are a wide variety of agents that can cause immunologic contact urticaria, and they can be either low-molecular-weight chemicals (haptens) or proteins.
- Immunologic contact urticaria can be easily missed clinically, particularly if the presentation is an apparent worsening of a preexisting dermatitis or urticaria.
- Management requires avoidance of the implicated agent.

## NONIMMUNOLOGIC CONTACT URTICARIA

Nonimmunologic contact urticaria (NICU) is caused by a wide variety of agents. NICU reactions tend to be of less severity than immunologic contact urticaria (ICU) reactions. H-1 antihistamines do not seem to protect against NICU reactions from contact with agents such as benzoic acid or cinnamal (Lahti 1980). NICU reactions can be, however, inhibited by either oral or topical nonsteroidal antiinflammatory drugs.<sup>1</sup> Prostaglandin D2 is released during NICU reactions to agents such as benzoic acid and sorbic acid.<sup>1</sup>

Lahti and Basketter (1980) classified NICU agents into the following subheadings: animals (eg, arthropods, caterpillars, corals); foods (pepper mustard thyme); fragrances and flavorings (eg, balsam of Peru, cinnamic acid, cinnamic aldehyde); medicaments (eg, benzocaine, camphor, witch hazel); metals (cobalt); plants (nettles seaweed); and preservatives and disinfectants (eg, benzoic acid, formaldehyde).<sup>2</sup>

Skin testing for NICU can be performed using either an open or closed patch test reading at 20, 40, and 60 minutes.

---

Conflicts of interest: none.

Department of Cutaneous Allergy, St John's Institute of Dermatology, King's College, St Thomas' Hospital, Lambeth Palace Road, London SE1 7EH, UK

E-mail address: [john.mcfadden@kcl.ac.uk](mailto:john.mcfadden@kcl.ac.uk)

Immunol Allergy Clin N Am 34 (2014) 157–167

<http://dx.doi.org/10.1016/j.iac.2013.09.005>

[immunology.theclinics.com](http://immunology.theclinics.com)

0889-8561/14/\$ – see front matter © 2014 Elsevier Inc. All rights reserved.

ICU

ICU involves a type 1 hypersensitivity mediated by allergen-specific immunoglobulin E (IgE) and, therefore, requires a sensitization phase. ICU can spread beyond the localized contact point. A staging system reflects the level of spread (Box 1).<sup>3</sup>

Broadly, 2 types of agents can cause ICU (Wang and colleagues 2013)<sup>3</sup>: proteins, such as natural rubber latex (NRL), with a high molecular weight that is often more than 10 000 kDA, and hapten chemicals, which conjugate with carrier proteins (eg, albumen), and the hapten-carrier protein can cause sensitization. Atopic individuals are more predisposed to developing ICU.

Diagnostic testing can be performed several ways; but the open test, which consists of gently rubbing the agent onto an open area of clinically normal skin and observing the site for 60 minutes, is most commonly used. Edema and erythema development denotes a positive test. Prick testing (or scratch testing) is also commonly used. As a rough guide, test reactions caused by ICU usually appear within 15 to 20 minutes, whereas reactions caused by NICU usually appear within 45 to 60 minutes.<sup>4</sup> In vitro radioallergosorbent test (RAST) tests for allergen-specific IgE are also used when available.<sup>3</sup>

NRL ALLERGY

Fourteen different allergenic proteins have been identified within NRL.<sup>5</sup> Hev b1 and Hev b 3 are the major protein allergens involved in patients with spina bifida. Hev b 5 is the main allergen in different risk groups and is recognized by 92% of health care workers and 56% of patients with spina bifida. Hev b 5 shows homology with kiwi acid protein. Hev b 6 is a major allergen, more important in health care workers than patients with spina bifida. Its sequence shows more than a 50% identity with chitinases from fruits, such as banana, avocado, and chestnut, giving rise to the latex-fruit syndrome.

Contact urticaria from latex gloves is the most frequent form of presentation of latex allergy and is more common than pruritus alone.<sup>5</sup>

Although rare, latex is the second cause of intraoperative anaphylaxis (after muscle relaxants).<sup>5</sup> Reactions to latex usually occur during the maintenance phase of operation, whereas when anaphylaxis is caused by opiates or muscle relaxants, it is usually during the induction phase.<sup>5</sup> The association between latex and fruit allergy varies, with the reported prevalence ranging between 21% and 58% of cases. The severity can also vary between mild itching and anaphylaxis. The most common foods implicated are banana, avocado, chestnut, and kiwi. Less commonly reported are papaya, lychee, fig, peach, potato, chickpea, spinach, and the leafy green vegetable phuk-waan-ban.<sup>4</sup>

Latex allergy may not always be suspected from the history. Prick testing has a sensitivity of 93% and specificity of 100%.<sup>5</sup> The sensitivity of serum RAST for

**Box 1**  
**Staging system**

Stage 1: Localized urticaria

Stage 2: Generalized urticaria

Stage 3: Urticaria plus extracutaneous manifestations (rhinitis, conjunctivitis, asthma, gastrointestinal symptoms, angioedema)

Stage 4: Systemic anaphylaxis shock

allergen-specific IgE to latex is also high. Prick testing is considered generally safe, but isolated cases of anaphylaxis have been reported.<sup>6</sup>

People with a latex allergy should be aware of products besides gloves that contain latex both in the hospital and home setting. These products include (in the hospital) catheter stoppers, elastic bandages, tourniquets tubes, and masks. In the domestic setting, these include balloons, condoms, mats, bottles, and baby bottle nipples. Alternatives for latex are available and include nitrile, neoprene, and polyvinyl chloride. Nitrile gives similar protection against infection as that offered by latex; synthetic polymers, such as neoprene, can be used as an alternative for surgical procedures.<sup>5</sup>

Occasionally, health care workers can develop contact urticaria to agents other than latex, such as handling the drug **donepezil** (used for Alzheimer), or disinfecting compounds, such as **dodecyl dimethyl ammonium chloride**.<sup>3</sup>

## FOOD

Virtually any food is capable of eliciting an ICU response.<sup>7</sup> ICU within the food industry is a major occupational problem. Food handlers can develop ICU from agents such as **apple, potato, carrot, and tomato**.<sup>3</sup> Shellfish and seafood are also common sources of ICU. **Prawn and lobster** as sources of ICU were recently highlighted.<sup>8</sup> Cooks who have ICU to raw seafood can usually tolerate eating cooked seafood provided the seafood is protein denatured by cooking.<sup>9</sup> Wang and Maibach<sup>3</sup> (2013) highlight the recent problems of **wheat** allergens among bakers. A new group of wheat allergens capable of causing type I hypersensitivity (asthma and contact urticaria) have been identified.<sup>10</sup>

Certain foods are repeatedly reported as causing problems.

### Seafood

Hjorth<sup>11</sup> (1975) reported on housewives with atopic dermatitis who develop contact urticaria to **haddock** and **herring**. He also reported a similar case of contact urticaria to **prawns** in a sandwich maker.<sup>12</sup> A 12-year-old atopic boy acquired contact urticaria to codfish.<sup>13</sup> A 19-year-old man developed generalized urticaria, angioedema of the face and lips, and a dry cough 20 minutes after his cousin, a fisherman, had greeted him with an embrace.<sup>14</sup> Open tests with raw **cod** and **sardine** gave large wheal-and-flare responses at both sites after 10 minutes. After 20 minutes, he complained of generalized pruritus and nasal irritation and was tachycardic, necessitating treatment with epinephrine. His RAST test was positive to **codfish, eel, and carp**.

### Fruit

Konstantinou and Grattan<sup>15</sup> (2008) reviewed the cross-reactivity between pollens and fruit (**Table 1**). Clinically, this usually manifests as a mucosal ICU. They term this **food**

**Table 1**  
Common cross-reactions between pollen/plant allergens and fruit

| Pollen/Plant Allergen | Common Fruit Cross-reaction                                        |
|-----------------------|--------------------------------------------------------------------|
| Birch                 | Apple, pear, carrot, celery, tomato, cherry                        |
| Mugwort               | Carrot, celery, aniseed, peach                                     |
| Ragweed               | Melon                                                              |
| Goosefoot             | Banana melon, peach                                                |
| Latex                 | Avocado, banana, chestnut, kiwi, mango, melon, fig, papaya, tomato |

**contact hypersensitivity syndrome** (also known as **oral allergy syndrome**). There are oropharyngeal sensations of pruritus, tingling and burning, which are accompanied by angioedema of the lips, oral mucosa, soft palate or tongue, shortly after eating raw (uncooked) foods. Patients may also experience exacerbation of hay fever symptoms. Handling the fruit can also cause contact urticaria. Gastrointestinal symptoms, such as diarrhea and stomachache, are uncommon. Rarely, life-threatening reactions with angioedema or shock have been reported. Denaturing the protein by cooking usually stops any reaction, with the exception of **nuts** and sometimes **celery**.

Immediate hypersensitivity to **apples** is perhaps the most common fruit allergy. In a study of 262 patients who exhibited fresh fruit and vegetable allergic reactions, 139 (53.1%) were allergic to apples.<sup>16</sup> The major allergen in apples is Mal d 1, a protein structure homologous with the birch pollen major protein Bet v 1.<sup>17</sup> In Spain, 70 patients with a **peach** allergy were diagnosed through clinical history, skin prick test, and open oral challenge and rub test.<sup>18</sup> Oral allergy syndrome (86%) was the most common symptom, followed by contact urticaria (61%) and systemic symptoms (26%). Sixty-seven percent were allergic to peach pulp, and 36% reported symptoms to canned peach. Eighty-one percent of the patients with a peach allergy also had a pollen allergy, and 73% had asthma. The nonpollen allergic patients were more prone to systemic symptoms. A patient diagnosed with contact urticaria to **lychee** had positive skin prick tests to both lychee and **mango**, demonstrating the close botanic affinity between these two fruits.<sup>19</sup>

### **Vegetables**

With testing for immediate hypersensitivity on symptomatic gardeners and greenhouse workers, 2 were positive to **lettuce** and 2 to the **tomato** plant.<sup>20</sup> Sensitivity to **potatoes** can be so severe that merely rubbing the intact skin with a raw potato may cause a wheal to form.<sup>13</sup> Contact urticaria to raw potato is not uncommon in chefs and domestic cooks, and the reactions can be severe. A 4-year-old boy who bit a raw potato developed urticaria, angioedema, respiratory distress, vomiting, and diarrhea.<sup>21</sup> A skin prick test was markedly positive. Of 27 patients diagnosed with **asparagus** allergy, 10 had IgE-mediated contact urticaria after handling asparagus.<sup>22</sup> A minority developed systemic symptoms after oral ingestion. An 84-year-old man presented with a 2-year history of swelling of the face, neck, and dorsum of the hands and arms.<sup>23</sup> The eruption would always arise a few minutes after tending **runner bean plants** in his garden. A prick test to runner beans demonstrated a 5-mm wheal. This case was only the second case reported of runner bean ICU. Case reports, often occupational, have also been reported from **carrot**, **garlic**, **paprika**, and **mustard**.<sup>24–27</sup>

### **Meat**

Contact urticaria has been reported with **chicken**, **lamb**, **turkey**, **calf's liver**, **beef**, **sausage**, and **venison**, particularly in patients with preexisting hand dermatitis.<sup>13,28</sup> A 60-year-old woman complained that her hand dermatitis worsened when preparing beef burgers, and her lips would swell when eating beef burgers.<sup>29</sup> A prick test to beef was positive but negative to chicken and pork. A 21-year-old hunter had contact with game most of his life, since he hunted as a boy.<sup>28</sup> Red deer and roe deer were stored around his farm until being sold. Over 2 years, he had noticed localized urticaria after contact with raw meat and the hair of the roe deer. Prick tests and rub tests confirmed immediate hypersensitivity to roe deer meat and hair. A minority of children with meat allergy can experience oral mucosal ICU.<sup>30</sup>

### ***Milk and Egg***

Oranje and colleagues<sup>31</sup> report on 40 children presenting with immediate contact urticaria rash around the mouth after consuming egg or milk, and 34 patients out of 40 also suffered from atopic dermatitis. A 25-year-old man developed urticaria when embracing his lactating wife and when in contact with the milky vomitus of his newborn child.<sup>32</sup> A prick test to cow's milk was negative but positive to his wife's milk. Contact urticaria to cow's milk in an 8-month-old girl was reported.<sup>13</sup> After heating the milk to 80°C there was no further problem.

## **COSMETICS**

### ***Fragrances***

Fragrances can cause immediate and delayed hypersensitivity reactions. A multi-center study in Hungary found that 6.1% of patients with a contact allergy to fragrance chemicals also had immediate contact urticaria reactions.<sup>33</sup> They found that contact urticaria was provoked by the fragrance chemicals (in order of most frequent) **cinnamic aldehyde**, **cinnamic alcohol**, and **evernia prunastri**. Cinnamic chemicals may be in some foods, and evernia prunastri is found naturally as oak moss. Some of the reactions from the cinnamic agents may have been nonimmunologic. Yamamoto and colleagues<sup>34</sup> (2002) describe a case of allergic contact dermatitis to **geraniol**.

### ***Ammonium Persulfate***

Ammonium persulfate is used in hair bleaching but has been reported to cause contact urticaria. A hairdresser developed contact urticaria and angioedema after airborne exposure.<sup>35</sup> NICU can also occur with ammonium persulfate exposure.

Contact urticaria can occur from exposure to **hydrolyzed wheat protein** in cosmetic creams and shampoos.<sup>36</sup> Three patients reported reactions to a hair conditioner containing hydrolyzed wheat protein, one on the hands and 2 acute urticaria on the head and neck. All were atopic patients.

## **HAIR DYES**

Temesvari<sup>37</sup> (1984) reported a case of contact urticaria from aromatic amine hair dye; since then, there have been a small number of cases continuing to be reported but some of an apparently serious nature.<sup>37</sup> However, before attributing a case of severe contact urticaria from hair dye to the aromatic amine contained in the hair dye, such as **p-phenylenediamine**, 3 points should be considered. Firstly, other agents within the product (such as hydrolyzed protein) may be the culprit. Secondly, other dyes, such as **basic blue 99**, **henna**, and **reactive dyes**, may be implicated.<sup>38</sup> Thirdly, a severe allergic contact dermatitis to aromatic amine hair dye with severe facial swelling may mimic angioedema even to the point of difficulty in breathing, which is caused by the edema constricting the airways.<sup>39</sup>

## **TOPICAL MEDICAMENTS**

ICU may occur from the active agent or from the preservative, base, or additives. A wide range of antibiotics (**bacitracin**, **cephalosporin**, **chloramphenicol**, **gentamicin**, **neomycin**, **penicillin**, **rifampicin**, and **streptomycin**) have been reported as causing contact urticaria and most also have the ability to cause (rarely) anaphylaxis.<sup>13</sup> Topical **local anesthetics** also have the ability to cause ICU. Ryan and colleagues<sup>40</sup> (1980) reported on mucosal contact urticaria to **benzocaine**, whereas Waton and colleagues<sup>41</sup>

(2004) reported on contact urticaria to **lidocaine** from the use of a local anesthetic cream used for venesection. It is claimed that most cases of contact urticaria to local anesthetic agents are nonimmunologic.<sup>42</sup>

A 36-year-old woman and a 16-year-old boy developed contact urticaria to **nitrogen mustard** used for treating mycoses fungoides.<sup>43</sup> Both individuals had urticaria and anaphylactoid reactions, and both had previous exposure to nitrogen mustard.

**Sunscreens** have been an occasional source of contact urticaria. Landers and colleagues<sup>44</sup> (2003) reported a patient with both immediate and delayed type hypersensitivity to **oxybenzone**.

Additives to topical medicaments can also cause problems. A severe urticarial reaction with systemic involvement resembling an anaphylactic reaction occurred after the use of a topical steroid.<sup>45</sup> It was found that this reaction was caused by **hexylene glycol**, an excipient in the formulation. Glycols are widely used in cosmetics, foods, and drugs, particularly topical medications. A 42-year-old woman presented with a clear history of pruritus and redness within 30 minutes of applying certain topical preparations, including emollients and corticosteroid creams.<sup>46</sup> There were marked urticarial reactions to the preservative **p-chloro-m-cresol** and to the steroid creams and emollient, which contained p-chloro-m-cresol. Codreanu and colleagues<sup>47</sup> (2006) emphasized the importance of possible allergic reactions to food proteins in topical medicaments and cosmetic products.

## ANIMAL HANDLERS

Animal handlers can develop ICU to animal hair and secretions.<sup>3</sup> In Finland, cows are kept indoors most of the year so dander exposure is increased. ICU to **cow dander** is one of the most common occupational diseases in Finland.<sup>3</sup> **Dog and rat saliva, animal hair, cow placenta, rat tails, and guinea pigs** can all be causes of ICU on handling animals.<sup>13</sup> Agrup and Sjostedt<sup>48</sup> (1985) investigated 101 laboratory technicians for a possible allergy to laboratory animals. Fourteen cases of contact urticaria caused by rats were found. Other animals causing contact urticaria were mouse (7), guinea pig (4), and cat (2).<sup>48</sup>

## PLANT HANDLERS

Exposure to several plants can cause contact urticaria, so it is not surprising that occupational contact urticaria is seen. Some examples are **Compositae** and **ivy**.<sup>3</sup> Other common causes of ICU to plants in gardeners include **yucca, spathe flower, and Chinese rose**.<sup>3</sup> Paulsen colleagues<sup>20</sup> report (1998) on gardeners and greenhouse workers implicated **Christmas cactus, Barberton daisy, and Madagascar jasmine** as the most common causes of occupational plant ICU. An IgE-binding protein known as profilin is present in several plant species and has been suggested as a common causative agent for ICU.<sup>49</sup>

## ENZYMES

After 1 year in his job, a 27-year-old atopic process man in a chemical enzyme factory developed whealing lasting 1 to 2 hours after contact with **detergent protease** enzymes.<sup>50</sup> Prick testing to a *Bacillus subtilis*-derived protease enzyme was strongly positive as was a RAST assay. Other enzymes reported as causing ICU include **amylase** (especially in baker's),<sup>13,51</sup> **cellulose enzymes**,<sup>52</sup> **xylene** (an aromatic hydrocarbon rather than an enzyme) in laboratory workers,<sup>53</sup> and **papain** (wide pharmacologic, domestic, and industrial use).<sup>54</sup>

## RECENT CASE REPORTS

A 3-year-old atopic boy was referred after developing urticaria and angioedema immediately after he had applied his mother's moisturizing cream onto her skin.<sup>55</sup> A prick test to the moisturizing cream was positive as was prick tests to **macadamia nuts and hydrolyzed wheat protein**, both contained in the moisturizing cream. Because this was the first time the boy handled the cream sensitization must have been through previous proxy exposure to the cream on the mother's skin. The authors note that complete avoidance of hydrolyzed wheat exposure is hard to achieve through its common use in cosmetics and processed foods.

A 33-year-old man had worked for the last 5 years in a company processing meals containing seafood, mostly **squid**.<sup>56</sup> His professional duties included managing a crusher machine and cleaning these machines after use. After 1 year, he developed asthma, rhinitis, conjunctivitis, and contact urticaria. He had further symptoms 3 years later when cleaning the crusher machines with pressurized water. On prick testing to squid, there was a positive reaction with  $12 \times 30$  mm wheal reaction after 15 minutes, and then 3 more wheals developed. Occupational allergy to squid has rarely been reported before.

A 19-year-old woman with bleeding gums was advised to use oral prophylaxis and was prescribed **chlorhexidine** mouthwash to be used twice daily for 3 weeks.<sup>57</sup> The next day, she presented with urticaria on her forehead and face, the front of her elbows and forearms, the side and upper back region of the neck, and on the lower abdomen. On questioning, she had used the mouthwash for 1 minute. Prick testing to chlorhexidine confirmed type 1 hypersensitivity. In a previous study, 6 patients were reported to develop urticaria, dyspnea and anaphylactic shock caused by topical application of chlorhexidine, with testing confirming type 1 hypersensitivity.<sup>58</sup> Chlorhexidine can also be a rare contact allergen, usually from its use in the health care setting, the resulting dermatitis marked by the severity of the reaction.<sup>59</sup>

ICU to another antiseptic agent has also recently been highlighted. A 44-year-old nonatopic woman presented with a 5-year history of pruritic reddish wheals on her skin/mucosa immediately after contact with some soaps and topical products.<sup>60</sup> Recently, the use of a certain toothpaste had caused immediate swelling of her lips and tongue, leading to breathing difficulties. Kissing her husband, who was using the same toothpaste, caused swelling of her lips within minutes. The suspected products that she reacted to, namely, a toothpaste, moisturizer, metronidazole cream, and antibacterial liquid soap, all contained **triclosan** at concentrations of 0.2% to 0.5%. Early reading of patch tests showed severe urticarial reactions to the metronidazole cream and triclosan (2.0% pet.) within 10 to 15 minutes. No reactions were observed with other ingredients that could potentially cause ICU. Delayed type reactions to triclosan were negative. Triclosan is a broad-spectrum antiseptic used in a variety of household and personal care products, such as deodorants, toothpastes, hand soaps, dish soaps, facial cleansers, bath oils, emollients, and topical medicaments. It is also used as an antiseptic in hospitals. The researchers concluded that the diagnosis was ICU rather than NICU because the reaction did not stay localized, with angioedema leading to breathing difficulties. Also, the immediate skin test appeared within 15 minutes, favoring ICU, whereas NICU reactions often occur within 45 to 60 minutes.

A 27-year-old woman applied 0.5% **levofloxacin** eyedrops.<sup>61</sup> Immediately, she developed conjunctival hyperemia, nasal discharge, and sneezing. Subsequently, facial edema, pruritic rash on the trunk and extremities, and mild dyspnea developed. Prick testing to 0.5% levofloxacin hydrate solution was positive. The researchers

noted that levofloxacin is the most widely used antibiotic eyedrop in Japan. Although contact urticaria syndrome to these eyedrops is rare, 2 cases of anaphylaxis had been reported directly to the pharmaceutical company.

A 26-year-old woman experienced several episodes of fish allergy since she was 2 years old, developing respiratory distress and widespread wheals within 15 minutes after eating fish.<sup>62</sup> She subsequently strictly avoided eating fish. She also had a history of asthma, atopic dermatitis, allergic rhinitis, and egg allergy. When she was 24 years old, she developed an urticarial eruption on the site where she had applied a cosmetic cream containing **codfish-derived elastin**. The RAST test was positive for codfish, tuna, salmon, mackerel, flatfish, sardine, and salmon. Immunoblot analysis confirmed the presence of IgE antibodies to codfish elastin. Elastin, which is commonly used in cosmetic topical formulations, is traditionally isolated from bovine tendon. Because of the perceived risk of bovine spongiform encephalopathy, there has been a trend to use elastin extracted from fish, particularly the aortic arch of codfish, bonito, yellowtail, and tuna.

A woman (age undisclosed) ingested **sesame**-coated sticks.<sup>63</sup> She suffered intense swelling of the oral mucosa after 15 minutes. She had previously been asymptomatic when ingesting sesame, and personal medical history was unremarkable except for Graves disease. Dermatologic examination revealed edema and erythema on the buccal mucosa, the hard palate, and tongue. The reaction faded within 6 hours after the use of antihistamine and local corticosteroid administration. Prick testing revealed a positive reaction of a 5-mm wheal to sesame. An oral provocation test 1 month later replicated the patient's symptoms. Sesame allergy has increased in the last few years because of the ubiquitous use of oil-rich sesame seeds in cooked and processed food, such as buns, bread, cakes, cereals, salad dressings, and humus.<sup>64</sup> Sesame may also be present in oriental and Middle Eastern foods. Sesame allergy may either coexist or cross-react with peanut allergy. An atopic background is noted with most patients with a sesame allergy. Despite the high population exposure to sesame, there is a paucity of case reports involving sesame allergy.

A 29-year-old woman was referred with recurrent urticarial wheals on the forearms.<sup>65</sup> She was employed as a forensic technician and had regular exposure to *Cannabis* plants at work for the last 2 years. More recently, she noticed this rash developing within a few minutes of contact with the plant, particularly the *Cannabis* flowers. Prick testing with dried extract of *Cannabis* produced a 4-mm wheal; but a 13-mm wheal and flare was observed with an extract of immature flowering plant, and a 15-mm wheal and flare was observed with an extract of flowering material, implicating the flowering version as more allergenic. The researchers note that 4 people in the same workplace reported similar symptoms, so ICU to *Cannabis* may be more common than reported.

## REFERENCES

1. Lahti A. Non-immunologic contact urticaria. *Acta Derm Venereol* 1980;60:1–49.
2. Lahti A, Basketter DA. Immediate contact reactions. Chapter 5. In: Rycroft R, editor. *Contact dermatitis*. 3rd edition. Berlin: Springer-Verlag; 2001. p. 111–32.
3. Wang CY, Maibach HI. Immunologic contact urticaria- the human touch. *Cutan Ocul Toxicol* 2013;32(2):154–60.
4. Amin S, Lahti A, Maibach HI. Contact urticaria and contact urticaria syndrome (immediate contact reactions). In: Zhai H, Wilhelm KP, Maibach HI, editors. *Marzulli and Maibach's dermatotoxicology*. 7th edition. Boca Raton (FL): CRC Press; 2008. p. 525–36.

5. Cabanes N, Igea JM, de la Hoz B, et al. Latex allergy: position paper. *J Investig Allergol Clin Immunol* 2012;22(5):313–30.
6. Nicolau N, Johnston GA. Anaphylaxis following prick testing with natural rubber latex. *Contact Dermatitis* 2002;47(2):251–2.
7. Brancacio RR, Alvarez MS. Contact allergy to food. *Dermatol Ther* 2004;17:302–13.
8. Teo S, Teik-Jin Goon A, Siang LH, et al. Occupational dermatoses in restaurant, catering, and fast-food outlets in Singapore. *Occup Med* 2009;59:466–71.
9. Onesimo R, Giorgio G, Pill S, et al. Isolated contact urticaria caused by immunoglobulin E-mediated fish allergy. *Isr Med Assoc J* 2012;14:11–3.
10. Cirila AM. Asthma and baker's allergy: experience with health programs. *G Ital Med Lav Ergon* 2011;7:152–5.
11. Hjorth N, Reed-Petersen J. Berufsekszeme durch proteine. *Z Hautkr* 1975;50:85–7.
12. Hjorth N, Weissmann K. Occupational dermatitis in chefs and sandwich makers. *Contact Dermatitis Newsletters* 1972;11:300.
13. Rietschel RL, Fowler JS. Contact urticaria. Chapter 34. In: Rietschel RL, Fowler FS, editors. *From fisher's contact dermatitis*. Baltimore (MD): Wilkins and Wilkins; 1995. p. 778–807.
14. Kalogeromitros D, Armenaka M, Katsarou A. Contact urticaria and systemic anaphylaxis from codfish. *Contact Dermatitis* 1999;41:170–1.
15. Konstantinou GN, Grattan CEH. Food contact hypersensitivity syndrome: the mucosal contact urticaria paradigm. *Clin Exp Dermatol* 2008;33:383–9.
16. Ortolani C, Ispano M, Pastorello E, et al. The oral allergy syndrome. *Ann Allergy* 1988;61:47–52.
17. Holm J, Baerentzen G, Gajhede M, et al. Molecular basis of allergic cross-reactivity between group 1 major allergens from birch and apple. *J Chromatogr B Biomed Sci Appl* 2001;756:307–13.
18. Cuesta-Herranz J, Lazaro M, de las Heras M, et al. Peach allergy pattern: experience in 70 patients. *Allergy* 1998;53:78–82.
19. Giannattasio M, Serafini M, Guarrera P, et al. Contact urticaria from litchi fruit (*Litchi chinensis* Sonn). *Contact Dermatitis* 1995;33:67.
20. Paulsen E, Stahl Skov P, Andersen KE. Immediate skin and mucosal symptoms from pot plants and vegetables in gardeners and greenhouse workers. *Contact Dermatitis* 1998;39:166–70.
21. Beausoleil J, Spengel JM, Pawlowski NA. Anaphylaxis to raw potato. *Ann Allergy Asthma Immunol* 2001;86:68–70.
22. Tabar AL, Alvarez-Puebla MJ, Gomez B, et al. Diversity of asparagus allergy: clinical and immunological features. *Clin Exp Allergy* 2004;34:131–6.
23. Narayan S, Sansom JE. Contact urticaria from runner bean. *Contact Dermatitis* 2002;47:243–4.
24. Moreno-Ancillo A, Gil-Adrados AC, Dominiguez-Noche C, et al. Occupational asthma due to carrot in a cook. *Allergol Immunopathol* 2005;33(5):288–90.
25. Jappe U, Bonnekoh B, Hausen BM, et al. Garlic-related dermatoses: case report and review of the literature. *Am J Contact Dermatitis* 1999;10:37–9.
26. Foti C, Carino M, Cassano N, et al. Occupational contact urticaria from paprika. *Contact Dermatitis* 1997;37:135.
27. Valsecchi R, Leghissa P, Cortinovis R, et al. Contact urticaria syndrome from mustard in anchovy fillet sauce. *Contact Dermatitis* 2000;42:114.
28. Geyer E, Kranke B, Derhaschnig J, et al. Contact urticaria from roe deer meat and hair. *Contact Dermatitis* 1998;39:34.

29. Fisher AA. Allergic contact urticaria to raw beef: histopathology of the two specific wheal and flare reaction at the scratch test site. *Contact Dermatitis* 1982; 8(6):425.
30. Theler B, Brockow K, Dutta M, et al. Clinical presentation and diagnosis of meat allergy in Switzerland and Southern Germany. *Swiss Med Wkly* 2009; 139(17–18):264–70.
31. Oranje AP, Aarsen RS, Mulder PG, et al. Immediate contact reaction's to cow's milk and egg in atopic children. *Acta Derm Venereol* 1991;71(3):263–6.
32. Makinen-Kiljunen S, Plosila M. A father's IgE-mediated contact urticaria from mother's milk. *J Allergy Clin Immunol* 2004;113:353–4.
33. Temesvari E, Nemeth I, Balo-Banga M, et al. Multicentre study of fragrance allergy in Hungary. *Contact Dermatitis* 2002;46:325–30.
34. Yamamoto A, Morita A, Tsuji T, et al. Contact urticaria from geraniol. *Contact Dermatitis* 2002;46(1):52.
35. Poltronieri A, Patrini L, Pigatto P, et al. Occupational allergic 'march'. *Med Lav* 2010;101:403–8.
36. Niinimäki A, Niinimäki M, Makinen-Kiljunen S, et al. Contact urticaria from protein hydrolysates in hair conditioners. *Allergy* 1998;53:1078–82.
37. Temesvari E. Contact urticaria from paraphenylenediamine. *Contact Dermatitis* 1984;11(2):125.
38. Davari P, Maibach HI. Contact urticaria to cosmetic and industrial dyes. *Clin Exp Dermatol* 2010;36:1–5.
39. Tukenmez Demerci G, Kivanic Altunay I, Atis G, et al. Allergic contact dermatitis mimicking angioedema due to paraphenylenediamine hypersensitivity: a case report. *Cutan Ocul Toxicol* 2012;31(3):250–2.
40. Ryan ME, Davis BM, Marks JG. Contact urticaria and allergic contact dermatitis to benzocaine gel. *J Am Acad Dermatol* 1980;2:221–2.
41. Waton J, Boulanger A, Trechot PH, et al. Contact urticaria from EMLA cream. *Contact Dermatitis* 2004;51:284–7.
42. Phillips JF, Yates AB, Deshazo RD. Approach to patients with suspected hypersensitivity to local anaesthetics. *Am J Med Sci* 2007;334(3):190–6.
43. Grunnet E. Contact urticaria and anaphylactoid reaction induced by topical application of nitrogen mustard. *Br J Dermatol* 1976;94:101–3.
44. Landers M, Laws S, Storrs FJ. Contact urticaria, allergic contact dermatitis, and photoallergic contact dermatitis from oxybenzone. *Am J Contact Dermatitis* 2003;14:33–4.
45. Spoerl D, Schere K, Bircher AJ. Contact urticaria with systemic symptoms due to hexylene glycol in a topical corticosteroid: case report and review of hypersensitivity to glycols. *Dermatology* 2010;220(3):238–42.
46. Walker SM, Chalmers RJ, Beck MH. Contact urticaria due to p-chloro-m-cresol. *Br J Dermatol* 2004;151:936–7.
47. Codreanu F, Morisset M, Cordebar V, et al. Risk of allergy to food proteins in topical medicinal agents and cosmetics. *Eur Ann Allergy Clin Immunol* 2006; 38(4):126–30.
48. Agrup G, Sjøstedt L. Contact urticaria in laboratory technicians working with animals. *Acta Derm Venereol* 1985;65(2):111–5.
49. Amaro C, Goossens AN. Immunological occupational contact urticaria and contact dermatitis from proteins: a review. *Contact Dermatitis* 2008;58: 67–75.
50. Kanerva L, Vanhanen M. Occupational allergic contact urticaria and rhinoconjunctivitis from a detergent protease. *Contact Dermatitis* 2001;45:49–51.

51. Kanerva L, Vanhanen M, Tupasela O. Occupational allergic contact dermatitis from fungal but not bacterial alpha-amylase. *Contact Dermatitis* 1997;36(3):306–7.
52. Kanerva L, Vanhanen M, Tupasela O. Occupational contact urticaria from cellulase enzyme. *Contact Dermatitis* 1998;38:176–7.
53. Weiss RR, Mowad C. Contact urticaria from xylene. *Am J Contact Dermatitis* 1998;9(2):125–7.
54. Quinones D, Alonso S, Lopez R, et al. Contact urticaria, rhinoconjunctivitis and bronchial asthma from occupational use of papain. *Allergol Immunopathol* 1998;27:273–5.
55. Leheron C, Bourrier T, Albertini M, et al. Immediate contact urticaria caused by hydrolysed wheat proteins in a child via maternal skin contact sensitisation. *Contact Dermatitis* 2013;68:376–83.
56. Wiszniewska M, Tymoszek D, Pas-Wyroslak A, et al. Occupational allergy to squid (*Loligo vulgaris*). *Occup Med* 2013;63:298–300.
57. Chopra V, Chopra H, Sharma A. Allergic urticaria: a case report of rare skin allergy with a common mouthwash. *Indian J Dermatol* 2013;58(1):298–301.
58. Okano M, Nomura M, Hata S, et al. Anaphylactic symptoms due to chlorhexidine gluconate. *Arch Dermatol* 1989;125:50–2.
59. Goon AT, White IR, Rycroft RJ, et al. Allergic contact dermatitis from chlorhexidine. *Dermatitis* 2004;15(1):45–7.
60. Ozkaya E, Bozkurt PK. An unusual case of triclosan-induced immunological contact urticaria. *Contact Dermatitis* 2013;68:117–28.
61. Saito M, Nakada T. Contact urticaria syndrome from eye drops: levofloxacin hydrate ophthalmic solution. *J Dermatol* 2013;40(2):130–1.
62. Nishida K, Tateishi C, Tsurata D, et al. Contact urticaria caused by a fish-derived elastin-containing cosmetic cream. *Contact Dermatitis* 2012;67:171–7.
63. Erkek E, Sahin S, Ince U, et al. Mucosal contact urticaria to sesame seeds. *J Eur Acad Dermatol Venereol* 2012;26:790–1.
64. Perkins MS. Raising awareness of sesame allergy. *Pharm J* 2001;267:757–8.
65. Williams C, Thompson J, Wilkinson M. Work-related contact urticaria to *Cannabis sativa*. *Contact Dermatitis* 2008;58:62–3.