

CHRONIC OCCUPATIONAL CONTACT URTICARIA IN A FOOD HANDLER

Zahida Sunday¹

Amy Burdzik²

¹ Occupational Medicine Registrar, Department of Health, Western Cape Government, Cape Town

² Senior Lecturer, Division of Occupational Medicine, School of Public Health and Family Medicine, University of Cape Town, Cape Town

Email | zahida.sunday@westerncape.gov.za

ABSTRACT

As the second most common occupational skin disease after contact dermatitis, contact urticaria is a significant skin condition. The prevalence varies according to gender, age, history of atopy, pre-existing dermatoses, occupation, and workplace exposures. Health workers, food handlers and hairdressers are high-risk occupational groups. In the food industry, multiple foods, additives (including flavourings, preservatives and enzymes) and spices may induce immediate skin reactions and IgE-mediated sensitisation. This article discusses a case of occupational contact urticaria in a food handler working in the butchery and fishery of a local supermarket chain.

Keywords: contact urticaria; contact urticaria syndrome; protein contact dermatitis; food handler

INTRODUCTION

Occupational dermatoses are predominantly caused by contact dermatitis, which may be eczematous irritant (non-immunologic) or allergic (delayed cell-mediated type-IV hypersensitivity) in nature.^{1–3} Immediate-contact skin reactions have been reported since the 1970s. They are characterised by immediate inflammatory reactions of the skin following contact with an external substance.⁴ These include a spectrum of conditions:

- Contact urticaria (CU) was first described by Fisher in 1973 and involves a localised wheal-and-flare reaction that occurs within 30–60 minutes of contact with the precipitating substance. The reaction resolves within 24 hours, leaving no skin sequelae.⁴
- Contact urticaria syndrome (CUS) was first reported by Maibach and Johnson in 1975. It involves CU reactions with systemic symptoms.⁴ Severity staging of CUS includes four stages:
 - Stage 1 involves localised cutaneous reactions with urticaria or dermatitis after contact with the causative agent.^{4,5}
 - Stage 2 occurs when there is generalised urticaria after local contact.^{4,5}
 - Stage 3 includes symptoms of oro-laryngeal dysfunction, rhino-conjunctivitis, asthma or gastrointestinal dysfunction.^{4,5}
 - Stage 4 is the most severe and involves anaphylactic or anaphylactoid reactions.^{4,5}
- Protein contact dermatitis (PCD) was first described by Hjorth and Roed-Petersen in 1976,⁶ and involves eczematous

changes and inflammatory paronychia associated with contact with proteinaceous materials.

Our understanding of these conditions is still evolving.⁵ CU may be either non-immunologic/irritant (NICU) or immunologic/allergic in nature (ICU).^{1,5} The former may occur on first exposure, is always localised and is thought to be due to a combination of vasogenic mediators, including prostaglandin release.^{1,5,7} ICU requires pre-sensitisation and may be localised (CU) or generalised, or involve extracutaneous systems (CUS).^{5,7} It is due to type-I hypersensitivity and is mediated by allergen-specific IgE.^{5,7} A third ICU category, PCD, requires pre-sensitisation^{5,7} and is thought to occur when the loss of skin integrity through irritant dermatitis (including wet work or occlusive maceration) or microtrauma allows the penetration of proteinaceous material through the skin barrier, leading to sensitisation.⁶ The link between immunologic CU and PCD remains poorly understood.⁵ It has been suggested that recurrent episodes of CU cause the development of PCD, but the history of urticaria is often missing in PCD cases.⁸

Occupational contact urticaria (OCU) is caused by exposure to skin irritants and allergens in the workplace. The most common sites affected by OCU are the hands, followed by the wrists, arms and face.^{4,7,9} Statistics on OCU are scarce globally,⁷ but more so in South Africa. Searching for symptoms of urticaria on occupational screening questionnaires may improve its reporting, diagnosis and management.⁴ The prevalence of CU in the general population is 1–3%,⁴ but it is speculated that it is

underreported and often undiagnosed, because symptoms may be minor and of short duration.⁵ Kanerva et al¹⁰ analysed data from the 'Finnish Register of Occupational Diseases' between 1990–1994 and found that CU (including PCD) accounted for 29.5% (815/2 759) of occupational allergic contact dermatoses in Finland, second after allergic contact dermatitis (70.5% (1 944/2 759)).

In Melbourne, Australia, a 12-year retrospective study of patients attending a tertiary occupational skin-disease clinic revealed 1 443/1 720 (83.9%) occupational dermatoses.⁷ CU was diagnosed in 151/1 720 patients overall, with OCU diagnosed in 143 of these patients, representing 9.9% (143/1443) of the occupational dermatoses cases.⁷ The most commonly affected occupations in this study were health workers, food handlers and hairdressers.⁷

In the food industry globally, a range of foods are responsible for inducing immediate skin reactions after topical exposure, these include animal proteins (commonly meats, fish/seafood and eggs), fruit and vegetables, and plants and grains (commonly various flours).¹ Additives such as enzymes (eg α -amylase), flavourings, preservatives and spices are also culprit agents.^{1,4} Immediate type-I hypersensitivity cross-reactions are also recognised between foodstuffs.¹

Insufficient data are published about the epidemiology of OCU in South Africa. Data from a tertiary occupational skin clinic at the National Institute of Occupational Health¹¹ reported that 21/216 (9.7%) of patients seen there were diagnosed with occupational-related CU, and a similar report from Groote Schuur Hospital in Cape Town reported that 1/31 (3%) of new patients seen in a two-year period had occupational urticaria.¹²

We present a case of OCU in a food handler in South Africa.

CASE REPORT

A 33-year-old assistant working in the butchery and fishery at a retail supermarket was referred to the occupational dermatology clinic at a tertiary hospital by the Occupational Health Practitioner at her workplace. She had been experiencing a possible allergic reaction whenever her skin came into contact with food (especially fish) in her work area. Erythematous, pruritic wheals and itchy, swollen eyes and lips developed immediately on starting her work shift. These resolved after seven hours, despite her still being at work and performing the same activities and not taking medication.

Her symptoms had started about one year prior to her presentation at the tertiary occupational dermatology clinic; she had been employed in her current role for one year and nine months. There were no respiratory symptoms, rhinitis or conjunctivitis. She was asymptomatic at home but she noted that during the winter months her symptoms could occur during her morning commute to work using public transport.

For severe episodes, she used oral chlorpheniramine prescribed by her general practitioner. Her symptoms resolved with this

treatment and with time off work, but they recurred whenever she returned to work. She suspected workplace contact exposure to fish, meat, chicken, ice and/or spices, although she had noted that she could develop the skin rash even before touching these items. Although she cooked at home, she rarely prepared fish. Her winter-time symptoms support the suspicion of cold as a causative or exacerbating agent.

Her skincare routine at home included the use of fragranced soap without the application of emollients afterwards. At work, she is required to sanitise her hands frequently using a disinfectant rub or a hand-sanitising gel (on average every 15 to 20 minutes) and to wash with disinfecting hand soap and water whenever she touches meat, chicken or seafood (see Table I). No emollients are provided at work to preserve the skin-barrier function.

TABLE I: SUSPECTED CAUSATIVE WORKPLACE EXPOSURES FOR CU IN OUR PATIENT

HAZARD TYPE	SUSPECTED AGENT
Biological	- Animal proteins: seafood/fish, meat (beef/mutton/pork), chicken, sausage casings - Marinades (including lemon concentrate, vinegar, garlic, sugar, salt, milk, tomato, apricot jam/puree, onion, wheat flour, spice extracts, molasses) - Spice mixes (including coriander, paprika, black pepper, mustard, onion, cumin, garlic, parsley, celery, fenugreek, thyme, oregano, wheat, maize, yeast extract, milk powder, salt, spice extracts)
Physical	- Cold - Wet work
Chemical	- Additives: whitening agent, raising agent, emulsifier, stabiliser, acidity regulators, ascorbic acid, flavouring, colourants, sodium saccharin, acesulfame K - Preservatives: sodium metabisulphite, sodium sulphite, sulphur dioxide, potassium sorbate, sodium benzoate - Disinfecting hand soap (containing dialkyl dimethyl ammonium chloride and alkyl dimethyl benzyl ammonium chloride) - Propanol-based disinfectant rub (containing 30–40% n-propanol and dialkyl dimethyl ammonium chloride) - Hand-sanitising gel (containing 50–60% ethanol) - Surface disinfectant sanitiser (chlorine-based, containing sodium dichloro-5-tazinetriene)

Her background medical history included a diagnosis of human immunodeficiency virus (HIV) infection, for which she was prescribed antiretroviral therapy (tenofovir, emtricitabine and efavirenz) since the time of her diagnosis five years previously. She had no personal or family history of atopy. She had never reported any allergic reactions when ingesting food.

Physical examination at her consultation visits revealed the thickened palmar skin of a worker chronically exposed to irritants, minor scratch marks and pigmentary changes but no eczema or urticaria (see Figure 1). A cold-provocation test and a test for dermographism were performed, both being negative.

A workplace walk-through inspection was conducted to determine potential causative exposures. In the fishery area,



Figure 1: The hands of our patient showing thickened palmar skin of a worker chronically exposed to wet work and irritants, minor scratch marks and pigmentary changes but no eczema or urticaria

different types of seafood exposure were noted, predominantly hake, salmon, snoek, tuna, kingklip and crustaceans. These were locally caught, and employees filleted and cut the fish which they served to customers. Hake was the most frequently handled, but the patient was not suspicious of any particular fish types.

Cold was a significant hazard, because employees constantly handled the crushed ice used to preserve the seafood and often entered the cold rooms with an average refrigerator temperature of 1°C and an average freezer temperature of -19°C. The ambient air temperature in the fishery was not monitored.

Work in the butchery area involved significant exposure to beef, mutton, lamb and pork, which employees had to cut, process and marinate. There was minimal exposure to chicken that had already been cleaned and cut on arrival. The employees also frequented the cold rooms in the butchery. Temperatures in this area included an average refrigerator temperature of 2°C, an average freezer temperature of -16°C and an average ambient air temperature of 16°C. In addition, work in the butchery area involved frequent exposure to various spices and marinades (see Table I).

Personal protective equipment included uniforms (white coat), safety shoes, plastic aprons and hair nets. The employees did not wear gloves in the fishery or the butchery, nor did they wear any particular personal protective equipment or garments for the cold. Each day a clean white coat was worn and this was laundered offsite. Information on the detergent chemicals used to wash uniforms is pending. The cleaning chemicals used by the employee in her work area included only a chlorine-based surface disinfectant-sanitiser.

Specific IgEs were negative for beef, mutton, pork, seafood mix (including fish, shrimp, blue mussel, tuna, salmon) and three spice mixes (including tarragon, marjoram, thyme, lovage, caraway, cardamom, clove, mace, anise, basil, fennel and

ginger). Specific IgEs for garlic and coriander were also negative. Skin-prick testing with relevant commercial preparations was not readily available and prick-by-prick testing, scratch testing and open-application testing are pending.

The patient was managed with oral cetirizine 10 mg taken in the mornings and her urticaria resolved. However, she was simultaneously accommodated by working in the butchery only and by avoiding the fishery area. Breakthrough symptoms were noted only when she was non-adherent to the cetirizine.

A diagnosis of OCU was made, based on the clinical picture. Although a specific causative contact has not been identified, it could be immunologic in nature (OICU) as it responded well to oral antihistamines and workplace accommodation. We will perform prick testing to investigate this further. Despite a negative cold-provocation test, we cannot exclude a diagnosis of physical urticaria resulting from exposure to cold exacerbated by workplace conditions, particularly as she experienced symptoms on cold mornings during winter. A further differential diagnosis includes occupational NICU. A diagnosis of drug reaction to the antiretrovirals was unlikely owing to the duration of the symptoms compared to her duration of treatment, but it cannot be ruled out as a cause or cofactor for her urticaria.

RISK FACTORS

The prevalence of OCU and PCD varies by gender, age, occupation and risk exposures, and a personal or family history of atopy.⁷ Loss of skin integrity is also thought to be a risk factor for the development of immediate-type hypersensitivity after topical exposure.⁸

Data from multiple studies show a female preponderance of allergic occupational skin disease.^{2,7} This may be due to their involvement in high-risk occupations such as healthcare and/or nursing, food handlers, hairdressers and cleaners.^{2,7} The mean age for a diagnosis of OCU is approximately 36–37 years^{2,7} and the incidence of CU declines with age.¹

A French study conducted by Bensefa-Colas et al² reported that OCU was most frequently associated with health and social workers, with natural rubber latex (NRL) as the predominant cause. OCU declined significantly after measures to prevent exposure to latex were implemented.² Other risk occupations for OCU associated with NRL and non-NRL reported in the French study were hairdressers, cleaners or machine operators.² Similar findings were reported from Australia, where, in a retrospective study, the most common occupations affected by OCU were health workers, food handlers and hairdressers.⁷ The food service industry was the most commonly affected by non-NRL OCU.⁷ Statistics from a Finnish study indicated that the highest number of cases of OCU occurred in bakers, preparers of processed food, dental assistants, veterinary surgeons, farmers and domestic animal attendants.⁷

A history of atopy is reported to increase the risk of sensitisation, especially to NRL, foodstuffs and ammonium persulfate in an occupational setting.⁴ Irritant contact dermatitis (ICD) is also thought to favour sensitisation to allergens, especially large protein allergens, through damaging the skin barrier and allowing penetration.⁴ Approximately 30% of patients with CU have co-existent contact dermatitis.¹

CLINICAL APPROACH TO ALLERGIC OCCUPATIONAL SKIN DISEASE

The diagnosis of allergic occupational skin disease with type-I hypersensitivity is based on a suggestive clinical picture, including the history, physical examination and special investigations. Positive specific IgE or prick testing points to the occupational exposure agent.

HISTORY

A detailed history of the presenting problem should include an immediate skin-contact reaction (within approximately 30–60 minutes), which may include symptoms of itch, erythema/flare and wheal-generalised urticaria; systemic symptoms of an acute allergic reaction may also be present. CU symptoms usually resolve within 24 hours. If signs of eczema are present and PCD is suspected, asking about initial urticarial symptoms may provide a useful clue to IgE-mediated hypersensitivity, but these symptoms are often not present in the history. Current or previous therapeutic options used and the response to these must be interrogated. Work-relatedness is usually established by determining symptoms which are worse at work and better or absent at home. However, the patient may encounter similar food items when cooking at home and this must be documented.

Identifying risk factors such as a personal or a family history of atopy and pre-existing dermatoses that result in the loss of skin integrity is important, as is a detailed history of skin hygiene and skincare both at home and at work. An occupational history will identify high-risk occupations and tasks with potential exposures to high-risk agents including personal protective equipment.

PHYSICAL EXAMINATION

A meticulous physical examination is important to identify the location of all the affected areas. The hands, forearms and face are typically involved in occupational dermatoses. In the case of

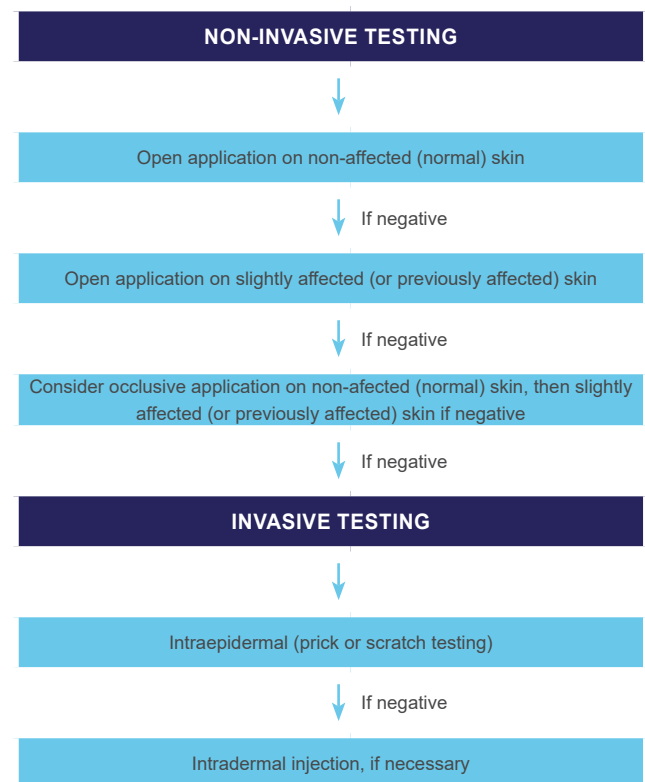


Figure 2: Protocol for investigating immediate skin-contact reactions (adapted from Gimenez et al⁴)

CU, there may be no skin lesions at the time of consultation or there may be signs of irritant dermatitis, including scratch marks and pigmentary changes. Patients with PCD have eczematous changes.

The feet should be examined in anyone presenting with hand dermatitis, because fungal infections with an id reaction (auto-eczematization in response to a primary infection) may present similarly to hand dermatitis.

Dermographism may be evoked by stroking the skin and waiting 5–7 minutes for a wheal-and-flare reaction. A cold-stimulation test may similarly be performed with topical ice exposure and looking for an urticarial reaction.

INVESTIGATION

A workplace walk-through inspection is useful to identify potential causative agents. Where immediate-type hypersensitivity is suspected, specific IgE tests or prick testing should be conducted for the suspected allergens, but they are not always available. In the absence of severe systemic symptoms, a pragmatic and cost-effective approach for immediate skin reactions can be used (see Figure 2). An 'open test' is performed first by rubbing the suspected substance on a 3 × 3 cm area of non-affected skin (upper back or the extensor side of the upper arm). The duration of exposure should not exceed 20 minutes, after which a contact reaction is usually evident and the skin must be cleaned. If negative, 'open testing' progresses to slightly affected skin, then occlusive application on normal and slightly affected skin with each negative reaction. Prick testing

with commercially prepared allergens or prick-by-prick testing is the method of choice if open testing is negative.

Scratch testing is less standardised but useful for studying non-standard allergens. Positive (histamine hydrochloride) and negative controls (aqueous sodium chloride solution) must be used with both prick and scratch tests. Ideally, when testing with non-standardised allergens, the test should be conducted on 20 controls to exclude false-positive reactions.⁴ Resuscitation equipment and trained personnel must be available in case of severe reactions.

Patch testing may be useful to identify co-existent allergic contact dermatitis but should not be done in isolation if only immediate skin symptoms are present.

DIFFERENTIAL DIAGNOSIS

The diagnosis of OCU (including CUS and PCD) may be difficult despite a thorough history and physical examination, because identification of the causal agent may remain elusive. Co-existing occupational dermatoses may also be present.

Differential diagnoses for chronic urticaria (urticaria lasting >6 weeks) must be considered. This includes chronic spontaneous urticaria and chronic inducible urticaria, as outlined in Table II, along with their sub-types and appropriate diagnostic tests.

If the history of urticaria is atypical (ie the patient has recurrent unexplained fever, arthralgia and/or malaise, or urticarial plaques lasting longer than 24 hours), additional diagnoses should be

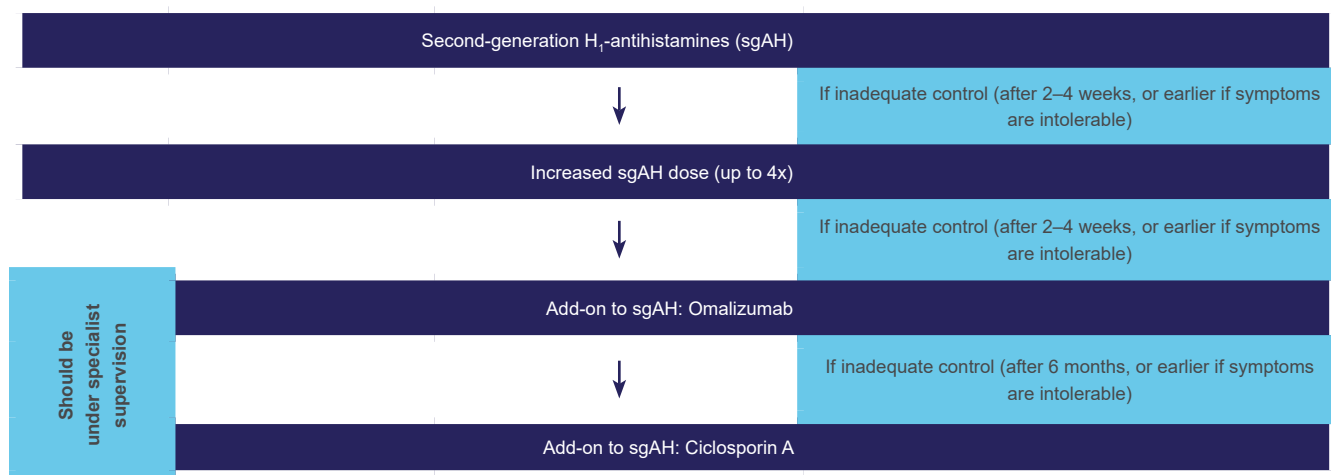


Figure 3: Recommended algorithm for the treatment of CU (adapted from Zuberbier et al¹³)

TABLE II: DIFFERENTIAL DIAGNOSES FOR CU (ADAPTED FROM ZUBERBIER ET AL ¹³)			
TYPE	SUB-TYPE	RECOMMENDED ROUTINE DIAGNOSTIC TESTS	EXTENDED DIAGNOSTIC PROGRAMME, BASED ON SUSPECTED CAUSE
Spontaneous urticaria	Chronic spontaneous urticaria	Differential blood count, ESR and/or CRP	Avoid suspected triggers. Conduct diagnostic tests for: - infectious diseases; - functional autoantibodies (eg autologous skin serum test); - thyroid gland disorders; - allergy; - concomitant chronic inducible urticaria; - severe systemic diseases (eg tryptase); - other (eg lesional skin biopsy)
Inducible urticaria	Cold urticaria	Cold-provocation and threshold test	Differential blood count and ESR or CRP. Rule out other diseases, especially infections
	Delayed pressure urticaria	Pressure test and threshold test	None
	Heat urticaria	Heat-provocation and threshold test	None
	Solar urticaria	UV and visible light of different wavelengths, and threshold test	Rule out light-induced dermatoses
	Symptomatic dermographism	Elicit dermographism and threshold test	Differential blood count, ESR or CRP
	Vibratory angioedema	Test with vibration	None
	Aquagenic urticaria	Provocation testing	None
	Cholinergic urticaria	Provocation and threshold testing	None
	Contact urticaria	Prick testing, prick-by-prick testing, scratch testing and open application testing	Specific IgE testing

considered, such as acquired or hereditary autoimmune inflammatory and autoimmune disease, or urticarial vasculitis.¹³

MANAGEMENT

The mainstay of treatment is the avoidance of the causative agent. Second-generation H₁-antihistamines (sgAH) given daily may help with symptomatic relief in CU (including ICU).¹³ Patients must be counselled on the use of emollients and skincare at home and at work in order to repair the skin barrier and prevent further damage and allergen exposure.

If there is inadequate control with sgAH after 2–4 weeks of regular daily use (earlier if intolerable symptoms), the dosage may be increased up to four-fold (see Figure 3).¹³ When treatment fails to achieve control despite regular daily high-dose sgAH after 2–4 weeks, newer agents such as omalizumab (anti IgE) can be considered as add-on treatment.¹³ Other treatment options have been used in different healthcare settings, including leucotriene receptor antagonists (eg montelukast), H₂-antihistamines (eg cimetidine) and pseudoallergen-free diets. But these all have low-level evidence and weak recommendations and are not included in the most recent guideline of the European Academy of Allergology and Clinical Immunology/Global Allergy and Asthma European Network/European Dermatology Forum/World Allergy Organisation (EAACI/GA²LEN/EDF/WAO) on the recommended treatment algorithm for urticaria.^{13,14}

Ciclosporin A is recommended only for those with severe disease when it is refractory to combination therapy with sgAH and omalizumab.¹³ Ciclosporin has a higher incidence of adverse effects compared to sgAH and omalizumab, but the risk–benefit ratio is much better than that of long-term steroids.¹³

Occupational dermatoses may have significant consequences, including frequent sick leave, possible job changes and disability. Ideally, they should be managed following a multidisciplinary approach that includes the occupational health nurse at the workplace, supervisors and treating doctors.

In South Africa, the Compensation for Occupational Injuries and Diseases Act ((COIDA) 130 of 1993) provides for compensation for occupational skin diseases. The worker is entitled to medical treatment, paid time off work (independently of the usual paid sick leave under the Basic Conditions of Employment Act 75 of 1997) and compensation should there be any permanent impairment. However, the system functions poorly and many workers end up paying for their own treatment and are lost to follow-up before their claims have been processed.

CONCLUSION

Food handlers are exposed to a wide variety of potential topical allergens in the workplace. Among occupational dermatoses, ICD is the most common followed by delayed hypersensitivity occupational allergic contact dermatitis, and then by immediate hypersensitivity CU and PCD. However, mindfulness of ICU and PCD must be maintained, especially in the food industry, where immediate hypersensitivity through cutaneous sensitisation occurs more commonly than in other occupations. Accurate diagnosis need not be challenging where specific IgE tests are negative or unavailable, and commercial allergen dilutions for specific prick testing are limited. Instead, a cost-effective pragmatic approach with open/closed application tests and prick-by-prick testing can be used. A meticulous history, a high index of suspicion and detailed workplace evaluation are therefore important to investigating appropriately and to diagnosing and managing the condition in individual employees.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest.

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