

# Occupational causes of contact dermatitis



PREVENTION. CARE. RECOVERY.

Te Kaporeihana Āwhina Hunga Whara

»» *A distillation of best practice reflecting ACC's current position*

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- Contact dermatitis encompasses: contact allergic dermatitis, contact irritant dermatitis, contact urticaria and photocontact dermatitis.
- Given the right circumstances, an irritant can induce dermatitis in anyone. The degree, the frequency and the duration of exposure are all important factors – i.e. contact irritant dermatitis is dose dependent.
- Contact allergic dermatitis affects only a small percentage of exposed individuals. Many years of uneventful exposure may precede sensitisation, but once an individual becomes sensitised, even tiny exposures can induce dermatitis.
- Contact irritant dermatitis can be prevented by reduced exposure to irritants and the use of moisturising creams.
- The management of contact allergic dermatitis usually requires complete avoidance of the allergen.

## Background

The incidence of occupational contact dermatitis is 5-19 cases per 10,000 full-time workers per year<sup>1</sup>. These figures are likely to underestimate the problem, as not all cases are notified as an occupational disease. The industries most affected by occupational dermatitis are<sup>2</sup>:

- Food handler/chef
- Hairdresser/beautician
- Medical/dental/nurse/vet
- Agriculture/florist/gardener
- Cleaning/laundry
- Painting
- Mechanical/engineer
- Printing/lithography
- Construction.

## Clinical

Contact dermatitis encompasses: contact allergic dermatitis (CAD), contact irritant dermatitis (CID), contact urticaria (CU) and photocontact dermatitis (PCD). Different forms of dermatitis may coexist e.g. an individual may have atopic dermatitis, CID and CU. In general, morphology does not differentiate contact dermatitis from the endogenous form; the diagnosis is suggested by the distribution, severity, temporal association with certain activities, and allergy testing as appropriate.

CID results when physical or chemical damage overwhelms the skin's repair mechanisms. It may be acute (e.g. a chemical burn from a strong alkali) and it may be due to a single irritant. However, more often it is the chronic and cumulative effect of multiple irritants. Irritant dermatitis CID most commonly affects the hands, but irritant particles may cause dermatitis where they gather under clothing. Water, soaps, solvents, acids, alkalis and rough textures are common irritants. Given the right conditions, an irritant can induce dermatitis in anyone: frequency and duration of exposure are important factors – i.e. CID is dose dependent. If the individual is innately susceptible or the barrier layer is disturbed (e.g. recently healed or current dermatitis), or penetration of the barrier layer is enhanced by occlusion (e.g. clothing or gloves), the risk of CID is increased.

CAD affects only a small percentage of exposed individuals. Many years of uneventful exposure may precede sensitisation, but once an individual is sensitised, even tiny exposures can induce dermatitis. A cell-mediated immune reaction results in dermatitis 24-96 hours after contact with the allergen. CAD most commonly affects the hands, but may also involve sites of secondary contact (e.g. face) where small amounts of allergen have been transferred accidentally by contaminated fingers. Although there are thousands of potential allergens, a relatively small number account for the majority of CAD cases. Common allergens include rubber additives, chromate, epoxies, nickel, hair dyes, fragrances, biocides and plant derivatives including colophony.

CU may be IgE mediated or (more commonly) may occur through non-immunological mechanisms. It results in immediate itching, welts or aggravation of eczema at the site of exposure, and occasionally generalised urticaria (in the case of immune mediated CU). It is most commonly caused by raw meat, fish or vegetables in food handlers, fish processors and abattoir workers; it can also be caused by rubber latex.

PCD can be photoallergic or phototoxic. It follows skin contact with a chemical that is activated by ultraviolet to produce either a photoallergen (causing dermatitis through immunologic mechanisms) or a phototoxin (causing dermatitis through non immunologic mechanisms). Therefore the dermatitis affects sun-exposed sites. In New Zealand sunscreen chemicals are the principal photoallergens. Phototoxic reactions are most

commonly due to furocoumarins in plants (e.g. parsnip, celery) and as such affect vegetable harvesters and gardeners.

## Investigation

There is no diagnostic test for CID. The following questions must be used in assisting diagnosis:

- Is the patient exposed to irritants?
- Is the frequency/duration/concentration of exposure sufficient to plausibly cause dermatitis?
- Are the sites of the dermatitis consistent with the manner of exposure?
- Does the dermatitis resolve/improve with reduction/cessation of the irritant exposure?
- Are there alternative explanations that might better account for the signs and symptoms?

CAD is diagnosed by patch testing: test substances are applied under occlusion to intact skin; readings are usually taken at 48 and 96 hours. Photo-patch testing for the diagnosis of allergic PCD is the same, except allergens are photo-exposed. CU is diagnosed by scratch-patch testing (test substances are applied over a superficial scratch, occluded and left for 20 minutes) or occasionally prick tests or RAST (radioallergosorbent tests) are used.

A recent editorial commented that “most dermatologists use patch testing infrequently and a significant minority of dermatologists do not patch test at all”<sup>3</sup>. Of those who do patch testing, many limit their test to a routine screen, which adequately evaluates only 16% of patients with contact allergy<sup>3</sup>. The scarcity of facilities for patch testing, photo-patch testing and scratch-patch testing is a major impediment to the adequate investigation of contact dermatitis.

## Management

CID can be prevented and managed by reduced exposure to irritants and the use of moisturising creams. While this sounds simple enough, in practice this is a complex area. Wearing gloves for prolonged periods may prove more irritating than the exposure one was trying to avoid by wearing gloves. There is a paucity of data on barrier creams and moisturisers, particularly in respect of their benefit in the management or prevention of dermatitis in particular occupations. The management of CAD usually requires complete avoidance of the allergen.

While short periods away from work may be necessary, recommendations to change career should not be given lightly. Most workers with contact dermatitis can continue in their jobs with appropriate treatment and work modifications; those with atopic tendencies may not fare well whether they stay in or leave their jobs.

A history of occupational exposure should be taken when a patient presents with contact dermatitis. If occupational exposure is considered to be significant in the aetiology, a claim should be lodged with ACC. The relevance of the exposure can then be determined and patients may receive assistance with ongoing treatment.

## References

1. Belsito DV. Occupational contact dermatitis: etiology, prevalence, and resultant impairment/disability. *J Am Acad Dermatol* 2005; 53:303-313.
2. Judd L. A descriptive study of occupational skin disease. *NZ Med J* 1994; 976:147-149.
3. Scheman A. Patch testing. *Arch Dermatol* 2004; 140:1529-1530.

## Further reading

1. Department of Labour 1995, A Guide to Occupational Skin Disease, published by Occupational Safety and Health Service.
2. Crowe MA. Contact Dermatitis. <http://www.emedicine.com/ped/topic2569.htm>.