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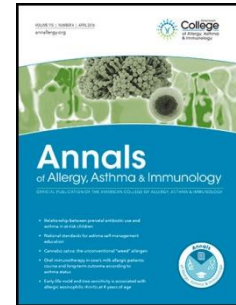
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Occupational Dermatitis: How to identify the exposures, make the diagnosis, and treat the disease.

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Key messages:

- Irritant contact dermatitis is the most common occupational skin disease.
- Wet work, and exposure to solvents that defat the skin, cause the majority of irritant contact dermatitis.
- Common sensitizing exposures across a number of occupations include gloves and rubber accelerators, preservatives, biocides, and sterilizing/cleaning agents.
- Use the history of exposure, and the physical distribution of the skin reaction, to direct the evaluation.

Introduction

Occupational skin diseases are the second most common type of occupational illnesses in the US, after musculoskeletal injuries and repetitive trauma¹. Using data from the occupational health supplements to the 2010 National Health Interview Survey, the prevalence rate of dermatitis among current workers was approximately 10%, representing about 15.2 million workers with dermatitis. Women are affected

more than men (58% vs 42%), and the highest prevalence rates were in healthcare workers². Estimates from the 2011 Behavioral Risk Factor Surveillance System (BRFSS) are higher, reporting a prevalence rate of between 12.9% and 17.6% of work-related dermatitis³.

Contact dermatitis constitutes 90% to 95% of all occupational skin diseases, and includes both allergic and irritant forms. The estimated annual costs of evaluation and treatment exceed \$1 billion. Skin cancers, skin infections, and skin injuries constitute the remaining 5 to 10%.

This review will address the different kinds of occupational contact dermatitis, how work-related skin disease may present, and its strong association with asthma and rhinosinusitis as well. Key exposures will be organized by occupation, each first characterized by a case report. Practical aspects of making the diagnosis by history, review of safety data sheets, and prick skin or patch testing will be reviewed separately. Treatment and outcomes will follow.

Kinds of occupational dermatitis

The two major subtypes of occupational dermatitis are irritant and allergic. Although irritant dermatitis is typically reported to constitute the majority, 80%, of occupational contact dermatitis (NIOSH), other studies suggest that allergic contact dermatitis is more prevalent⁴. In this report of 135 subjects with occupational skin disease who underwent patch testing, 60% were diagnosed with allergic contact dermatitis, compared to 34% with irritant contact dermatitis. As not all workers with occupational skin disease will undergo evaluation for skin sensitizers, the number sensitized to a workplace allergen may be under-reported because they were never evaluated by patch testing.

Exposures causing mechanical dermatitis at work include physical agents, such as extremely hot or cold environments, and UV and solar radiation. Mechanical trauma can cause scrapes, cuts and bruises that damage the skin, and can also increase exposure and susceptibility to allergens. Biological agents

including parasites and bacteria are the major sources of skin infections. Chemical agents are the primary cause of both irritant and sensitizer induced skin reactions, and biological agents such as plants and animal materials can act as either skin irritants or skin sensitizers as well.

Differentiating irritant from allergic contact dermatitis

The skin has only a limited number of ways it can react to chemical or biological agents, and it can be difficult to differentiate irritant from allergic skin disease on the basis of appearance ⁶.

Acute dermatitis is characterized by erythema, edema, scaling and vesicle formation, whereas chronic dermatitis typically demonstrates lichenification, excoriation, fissuring, and hyperkeratosis. Irritant contact dermatitis can be associated with stinging, burning, soreness, and pain in addition to itching. Over time, it is most commonly associated with skin thickening. In contrast, the hallmark of allergic contact dermatitis is pruritus.

The history becomes a key factor in distinguishing the two. In general, irritant dermatitis is limited to the area of direct skin contact, and symptoms may begin with initial contact; a period of immunological latency is not necessary. However, low grade irritants may not elicit symptoms until after a period of exposure, and some, such as sodium lauryl sulphate, cause a more intense inflammatory reaction at 48 hours after exposure, a time course more characteristic of allergic reactions. Allergic contact dermatitis is characterized by a period of latency, where exposure does not cause symptoms while the immune response is being developed. This can occur over several months, but sensitization after years of exposure has also been reported. Allergic responses typically develop from hours to days after exposure, and take longer to resolve than irritant reactions.

Patterns of exposure and association with respiratory diseases

Occupational contact dermatitis typically develops on areas of direct skin contact, and a systemic response to local exposure is rare. The most commonly exposed areas include the face, neck, 'v' of the anterior chest where fabric may not cover the skin, as well as forearms and hands. Airborne exposure will affect similar areas that are uncovered and open to the air, which may include the scalp, back of the neck, and ankles. Often, the pattern and appearance of the rash will give insight to the inciting exposure. For example, sensitization to rubber accelerators will affect the hand and forearm area covered by gloves. Sensitization to chromium may present as a foot and leg dermatitis from contact with chromium cured leather. Conversely, a rash on exposed skin, along with the potential development of nasal septal perforation ('chrome holes'), chronic sinusitis and asthma may develop from airborne chromium exposure from electroplating tanks. A linear rash along an exposed arm or leg may be caused by sensitization to plant material. Hence, the pattern of the rash provides an important clue as to the nature of the inciting exposure. And many airborne exposures can cause respiratory sensitization and disease in addition to a dermal presentation. For example, of the ten most common occupational contact allergens identified by patch testing of 397 subjects from 1/1/01 to 12/31/06, seven were possible causes of occupational asthma as well. These agents included epoxy resins, nickel, cobalt, chromium, para-phenylenediamine, formaldehyde, and glutaraldehyde. The remaining common occupational contact allergens, thiuram, carba mix, and glyceryl thioglycolate, were not associated with respiratory disease⁷. The conclusion is that with the evaluation of allergic occupational skin disease, the practitioner must also keep in mind that some skin sensitizing exposures can cause respiratory disease as well.

Common occupations of concern for work-related skin disease

1) Health care workers

Patient 1 is a 34 year old nurse who previously worked in the ICU and ER, but recently changed positions to work as an endoscopy nurse in a busy GI practice. Her new job duties included assisting the specialists with procedures, placing specimens in formalin, cleaning the exam room surfaces with a quaternary ammonium compound after each procedure, and placing the scopes in an enzyme bath as part of the sterilization procedure. After about 6 months on the job, she began to notice an itchy rash on her hands and arms that developed by the end of the work day, and had typically resolved by the next morning. She has a history of seasonal allergic rhinitis and asthma in childhood, but no longer required treatment in her 20s. Several months later, she began to develop a cough and chest tightness in addition to the itchy rash.

This case illustrates the complex allergenic exposures encountered by many health care workers. These can trigger both type IV contact allergies, as well as type I IgE mediated responses, and patients should be evaluated by both prick skin testing and patch testing, depending on the history. In one study of 55 health care workers, prick skin testing revealed 27% sensitized to latex, and patch testing showed sensitization in 51%. These agents included fragrance, nickel, rubber accelerators including thiuram mix and carba mix, formaldehyde, and glutaraldehyde. The final diagnoses, with some overlap between patients, were 61% with ICD, 31% with ACD, and 27% with contact urticaria. In particular, 11% were sensitized to thiurams and also had contact urticaria to latex. The clinical presentation of these workers is also instructive. The mean duration of rash was 40 months. The rash was predominantly on hands and arms, with some reporting rash on the face, eyelids, and neck. The rash was characterized as itchy, red, and scaling in >85%, with reports of papules, blisters, and cracking in about 60%. Importantly, of the 30 workers with more detailed information, 53% also had nasal symptoms, 39% had ocular symptoms, 10% had cough and 3% had wheeze. Eye and nose symptoms were more common in those allergic to latex, but not all⁸. And a review of the results of 31,849 patients patch tested for a rash found that those employed in the health care sector (n=~2200) had significantly higher rates of

sensitization to thimerosal, glutaraldehyde, formaldehyde, Glyoxal, and thiuram mix compared to non-health care workers⁹. A more recent study included the same allergens, as well as Cocamide Diethanolamine (DEA) and bacitracin¹⁰.

Dental workers have a unique set of exposures that should be considered in those developing an occupational contact dermatitis. Aside from sensitization to glove allergens, biocides, and cleaners, common in all health care workers, sensitization to dental acrylates is important. Acrylates are heavily used as dental cements, and in the preparation of dental implants and dentures, and are highly sensitizing. Several studies indicate that patch testing to 2-HEMA (2-hydroxy ethyl methacrylate) is able to detect from 80% to 100% of sensitized workers^{11, 12}.

Table 3 lists some of the more common allergens to be considered in health care workers with new onset skin disease.

Patient 1 was patch tested to formaldehyde and to glutaraldehyde with negative responses. A prick skin test of the enzyme solution, diluted 1:10 and 1:100, produced a wheal of 6 mm and a flare of 20 mm. A subsequent bronchial challenge using the enzyme solution to clean a basin caused a 35% fall in the FEV1 after a 10 minute exposure. The patient was diagnosed with contact urticaria and asthma due to sensitization to enzyme solutions and restricted from work in the clean room, where the solution was kept in open basins. Her rash and asthma subsequently resolved.

2) Cosmetologists

Patient 2 is a facial esthetician employed in a large commercial salon for over 15 years. In the past 5 years, the company has phased out use of methyl parabens in their skin care line, and substituted MCI (methyl chloro isothiazolinone) as a preservative. The patient has recently developed a persistent intense, itchy, vesicular rash on her hands. She finds it difficult to wear gloves at work as the

management prefers the beauticians do not, but the rash has worsened and makes it difficult for her to do her job.

Cosmetologists include a number of subspecialties, including make-up artists, hair stylists, estheticians who deal with skin issues, and nail technicians. Each subspecialty works with different chemicals, many of which are sensitizing. The most common site of involvement is the hand, followed by the face and scalp in these workers.

One study of 399 Danish hairdressers with contact dermatitis, who were patch tested between January 2002 and December 2011, compared their results to 1995 matched controls. Allergens significantly associated with occupation as a hair stylist included para-phenylenediamine, a black hair dye, thiuram mix, benzocaine, ammonium persulfate used as a bleaching agent, toluene-2,5-diamine sulfate used to activate hair dyes, and 3-aminophenol and 4-aminophenol used in the synthesis of hair dyes¹³. A different study of 432 hairdressers/cosmetologists in the US from 1994 to 2010 identified allergic contact dermatitis in 73% and irritant contact dermatitis in 37%. The most common occupational allergens include glyceryl thioglycolate used in permanent solutions, para phenylene diamine black hair dye, nickel, 2-hydroxyethyl methacrylate used in artificial nails, and quaternium-15 used as a preservative in lotions and other skin products¹⁴.

Gel nails are methacrylate based gel varnishes cured under UV light to form a durable nail coating that can last for several weeks. Methacrylates are highly sensitizing chemicals found not only in gel and acrylic nails, but also in wound dressings and contact lenses. One study of occupational contact dermatitis identified 52 cases of methacrylate sensitization. There were 24 cases of work-related methacrylate allergy, including 10 cases due to acrylic nails, and 7 cases caused by wound dressings.

Four individual methacrylate extracts: 2-hydroxyethyl acrylate (2-HEMA), 2-hydroxypropyl methacrylate, bisphenol A glycerolate dimethacrylate, and ethyl acrylate were able to identify 90% of the positive cases¹⁵. Additional sensitizers in nail polish include dibutyl phthalate used in polishes, topcoats and basecoats, formaldehyde, dyes including chromium, carmine, and manganese, and adhesive polymers such as tosylamide-formaldehyde resin. The hazards of nail salons were highlighted in a New York Times series by Sarah Maslin Nir, "The Price of Nice Nails" published May 7, 2015, and "Perfect Nails, Poisoned Workers," published May 8, 2015.

Make-up and skin care products contain a number of sensitizers, including fragrances, formaldehyde releasing preservatives, lanolin, parabens, propylene glycol, botanical extracts and chromium. Methyl isothiazolinone and methyl chloro isothiazolinone are both potent sensitizers, and their use is increasing. Natural ingredients, including plant extracts and herbal remedies, may also cause a severe contact dermatitis. The review article by An Goossens¹⁶ is an excellent resource.

Table 4 lists the most common allergens to be suspected in cosmetology related contact dermatitis. The patient was patch tested to preservatives used in the salon products, and was highly sensitized to methyl chloro isothiazolinone. The salon would not allow her to use products with a different preservative, and she was forced to quit her job.

3) Food Handlers

Patient 3 had worked at a delicatessen all through high school and in college. She had been promoted to manager, but still has to pitch in and make sandwiches when the shop is short staffed. The rules of the shop are that each worker had to wash their hands before and after each sandwich, and to wear

vinyl gloves when handling the food stuffs. After several years, the patient developed painful fissuring on her hands that was particularly irritated by the soap used in the shop.

Food handlers are exposed to a number of plant based products that can be sensitizing, capable of both type I and type IV reactions. Hence testing for skin reactions in these workers should include both prick skin testing and patch testing, depending on the history. An older report from 1987 indicated that 19/47 caterers with hand dermatitis were sensitized to a food¹⁷. The most common type I food sensitizers were fish, and the most common type IV sensitizer was garlic. A later study of 5 chefs with contact dermatitis to spices indicated that garlic, cinnamon, ginger, allspice and clove were the causative agents¹⁸. Most of the spices were patch tested as is, moistened with a drop of water, but because garlic and ginger can be irritating, patch testing was performed with a 10% water extract of these spices. Sensitization to garlic was confirmed by patch testing to the known allergen in garlic, diallyl disulfide.

In addition to almost any food, food handlers may also develop hand dermatitis from sensitization to gloves, as well as to antibacterial and disinfectant hand cleaners. Sensitizers may include Bronopol (2-Bromo-2-nitro-1,3-propanediol), Benzalkonium chloride, Chlorhexidine (Ex: Dyna-Hex, Betasept Surgical Scrub), Cetrimide (Ex: Cetavlon), Triclosan (5-Chloro-2-(2,4-dichlorophenoxy)phenol), and Triethanolamine. Wet work, and an irritant contact dermatitis are also common.

After reviewing the SDS for the soaps used in this shop, this patient was diagnosed with an irritant induced contact dermatitis from frequent hand washing and aggravated by the skin occlusion from use of vinyl gloves. Reducing the frequency of hand washing while continuing glove use improved, but did not resolve the hand dermatitis.

4) Cleaners

Patient 4 began working in a hospital as a perioperative assistant in the OR. Her job involved delivering surgical samples to the laboratory, but most of her time was spent cleaning the surgical suites after procedures. She used Virex, a quaternary ammonium compound diluted in water to clean the floors and surfaces. She used Alpha-HP multi-surface cleaner, containing phosphoric acid with a pH of 1.0 described as a strong irritant, to clean the operating room lights, and Saniwipes, also containing a quaternary ammonium compound, to wipe down different surfaces in the OR. After several months, she noted the onset of an itchy, vesicular rash present on her arms above the edge of her gloves, with a smaller rash present on her neck.

Occupational contact dermatitis is a common problem in the cleaning industry. Workers are exposed to many caustic cleaning products that are highly acidic or basic and can cause an irritant contact dermatitis, as well as to rubber and other gloves causing allergic contact dermatitis. One analysis of 655 cleaners with occupational hand dermatitis found that 31% were sensitized to a rubber accelerator, including thiurams in 12%, carbamates in 3.4%, and mercaptobenzothiazoles in ~2%. Formaldehyde was the other most common sensitizer in 3.4%¹⁹. Findings were similar in another more recent review²⁰. Disinfectants and biocides are an important cause of allergic contact dermatitis, especially in hospital cleaners. In addition to quaternary ammonium compounds, these products include formaldehyde and formaldehyde releasers, glutaraldehyde, chloroxylonol, para-hydroxybenzoic acid derivatives, and iodopropynyl butylcarbamate²¹. Eight hospital cleaners who developed an allergic contact dermatitis to a newly introduced surface-cleaning product, Mikrobac Extra™, were found to be sensitized to its ingredients PEG cocamines (polyethylene glycol ethers of the primary aliphatic amine derived from

coconut oil) and/or Benzalkonium chloride²². It is important to note that there are many publications also demonstrating an association between exposure to cleaning chemicals and asthma²³⁻²⁵.

Symptoms for patient 4 progressed to include rhinorrhea, chronic sinusitis, and severe asthma requiring daily prednisone. Symptoms abated when she was removed from her job. In this case, prick skin testing to Virex diluted 1:10 caused a late wheal of 5 mm and flare of 14 mm. Due to the severity of her asthma, she was unable to return to her job.

5) Outdoor workers, landscapers, florists

Patient 5 began working for a greenhouse as a floral designer 2 years prior, and notes that her fingers began cracking and bleeding within 10 days of starting work. She initially used cedar, pine, and fir boughs to create bases for Christmas arrangements, and then with Valentine's Day, worked with chrysanthemums, lilies, roses, and tulips. With increased exposure to tulips and Peruvian lilies while making floral arrangements for Mother's Day and weddings, she subsequently awoke one morning with blistered oozing fingers. She was patch tested to a number of different chemicals, as well as to fresh Peruvian lily (*Alstroemeria*) petals, leaves, and stems. She developed a blistering reaction to the lily parts, which took about two months to finally resolve.

Plant exposure can cause a variety of cutaneous effects, roughly classified into four basic reactions: urticarial, irritant, phototoxic (phytophotodermatitis) and allergic. Contact urticaria may be caused by a specific, IgE mediated response to the plant or food, typically fresh fruits and vegetables. Urticaria can also be caused by the direct release of allergic mediators such as histamine, acetyl choline,

prostaglandins and serotonin, caused by contact with the sharp hairs on certain plant leaves with the skin. This does not require prior sensitization, and is more common. A good example is exposure to the stinging nettle. Mechanical irritant dermatitis is caused by skin maceration by small, sharp parts of the plant, such as small spines from a cactus. Plants can also cause a chemical irritant dermatitis, such as the intense burning caused by capsicum from handling peppers. Phytophotodermatitis is a phototoxic reaction caused by exposure to plant furocoumarins that are activated by UVA light. These then can damage the dermis and epidermis, causing painful erythema, edema, bullae, and vesicles occurring roughly 24 hours after the initial exposure. Resultant hyperpigmentation can take years to resolve. Common plants causing phytophotodermatitis include carrot and celery, and many citrus fruits such as lemon, lime, grapefruit, and bergamot. A classic route of exposure is to citrus contained in after shave lotions, applied to the skin of the face, and then exposed to sunlight.

However, type IV contact dermatitis is the most common kind of plant-induced skin disease. Two large categories of plants are the typical culprits. The best known are members of the genus *Toxicodendron*, which includes poison ivy, poison oak and poison sumac, and containing urushiol as the allergen in common. "Leaves of three, let them be" refers to the 3 leaflets of poison ivy, although poison oak can have from 3 to 5 leaflets, and poison sumac from 7 to 13 leaflets. A plant must be damaged in order to release urushiol, and is considered to be especially toxic if its leaves have black spots on them – as these appear after urushiol is exposed to air and oxidized²⁶. Burning will release urushiol into the air, where it can be inhaled and cause both a contact dermatitis as well as respiratory injury and airways disease. Firefighters in certain areas of the US have developed asthma from inhaling the urushiol released from burning hillsides of poison oak or sumac²⁷. Several plants, including mango and cashew, are known to cross-react with members of the *Toxicodendron* genus. Patch testing is not recommended to make the

diagnosis of a Rhus allergy, as patch testing itself can be sensitizing. The diagnosis is made principally on the basis of history and physical exam.

Members of the daisy family, Compositae or Asteraceae, are the second most common sources of plant contact dermatitis. These include sunflowers, dandelions, and chrysanthemums, and are related to ragweed as well as to edible plants such as lettuce and artichokes. The active sensitizer is sesquiterpene lactone, which is located both within the flowers as well as the leaves and the stem. Plant dermatitis can also be caused by botanical extracts added to lotions and topical preparations. The-
Dermatologist.com²⁸ is a useful source of information. Allergic contact dermatitis has also been reported in 2 florists working with Alstroemeria, or Peruvian lily²⁹. Patch testing was positive to the plant itself, as well as to the allergen, tuliposide A. A study of 153/220 Dutch Alstroemeria bulb workers included evaluation by questionnaire and patch testing. Of these, 48 were determined to have major or minor hand dermatitis, and 25 (52%) were sensitized to Tuliposide A by patch testing³⁰.

Patient 5 subsequently developed nasal congestion and throat tightness along with sore burning fingers when working with both daisy mums (chrysanthemums) and Alstroemeria. She was atopic, with positive prick skin test reactions to trees, grasses, weeds including ragweed, along with cat, dog, and Alternaria. She developed both a contact dermatitis and a fall in peak flows when working with chrysanthemums, and also with Peruvian lilies and tulips. After the latter exposure, her methacholine challenge PC20 FEV1 fell by three doubling doses, indicative of a positive asthmatic response to Alstroemeria. The patient was restricted from exposure to chrysanthemums and Alstroemeria, and had to seek a job in a non-floral setting.

6) Metals

Patient 6 is a research chemist working for a pharmaceutical company that makes generic injectable drugs. The patient had no symptoms in his job for the first 7 years, when he first noticed an itchy rash on his arms. Symptoms progressed to include eye swelling and watering, a runny nose, and round, raised, itchy hives that lasted for 2-3 hours around his neck area. He initially noted that his rash appeared to be better at home and worse at work. The rash began to spread onto both hands and wrists, extending onto the chest area, and responded to treatment with steroid creams.

The patient had spent the past four and a half years developing and testing a chemical process to manufacture carboplatinum and oxaliplatin. Given his success, these chemotherapeutic agents were now being commercially produced, and the patient had oversight for the process.

The patient was patch tested to several different platinum compounds, including commercially available 0.25% ammonium tetrachloroplatinate in petrolatum, as well as two extracts prepared by the patient in his lab: 0.25% iodocis-platin in petrolatum, and 0.25% potassium tetrachloroplatinate in petrolatum. He reacted strongly with a blistering vesicular rash to all platinum compounds after two days of application, demonstrating sensitization to platinum. The location of the rash, present on the arms and chest, corresponded to airborne exposure to the platinum solutions.

A number of metals are known to be potent contact allergens. The most common sensitizers include nickel, cobalt, and chromium. Sensitization to indium, iridium, mercury, platinum, and titanium is less frequently encountered³¹. Palladium allergy is becoming more frequent, in part because it cross-reacts with nickel³². Although most allergy is caused by personal exposure to jewelry and other metal objects, occupational sensitization also occurs. Exposure can be airborne via electroplating baths, metal smelting, welding, in pigment and batteries production, galvanization, or by grinding, polishing, or machining metal³¹. Additive manufacturing is a method of creating three dimensional objects by

layering different metals onto a framework, and can involve exposure to airborne nickel, chromium and cobalt nano particles³³. Patch test panels to common metals are available from a number of different companies, although not all extracts have been well validated as either sensitive or specific.

Concurrent with the placement of the platinum patches, the patient developed rhinoconjunctivitis and wheezing. Spirometry showed a clinically significant bronchodilator response, with a 12% improvement in the FEV1. These results show bronchospasm, most likely in response to platinum exposure via the patch tests. The patient was treated with a prednisone burst and taper, an albuterol inhaler, topical nasal steroids and Lidex 0.05% external gel to be applied to the chest twice daily until the rash resolved. This outcome makes clear that platinum can act through both a type I and type IV mechanism.

Patient 7 had worked as a firefighter for 18 years. One year ago, his department received new boots that were similar to previous boots, but different in that the top two inches of the inner boot consisted of bare leather and was unlined. The rest of the boot was lined with Nomex, a nonleather fire retardant material. After about a month, the patient noticed a nickel sized itchy spot located on the back of his left calf that he attributed to his boot rubbing, and so began wearing higher knee socks that prevented direct contact of the boot with his skin. However, the rash became worse over the next few months, and spread to both legs. While on vacation that summer, his rash significantly improved, but became much worse when he returned to work. In late September, after wearing his fire gear for 3 hours, his legs became completely irritated. He was provided with new boots, but his rash spread to both palms of his hands and his upper arms, after wearing leather fire gloves, and also to his waist after wearing a leather belt. Patch testing revealed large positive reactions to chromium. Most leather is tanned with chromium, which was the source of his rash. His dermatitis resolved when he changed to wearing leather tanned with vegetable extracts. The two most common occupational exposures to chromium are through tanned leather, such as in work boots, and cement.

Making the diagnosis of occupational contact dermatitis

The evaluation is directed by the history. Details critical to deducing the diagnosis include:

- Time course of the rash – for how long had the patient worked in that particular job, and with which particular agents before noticing skin changes.
- What are the details of the job – start by asking what the patient does over the course of the day, with what products, and in what part of the plant.
- Get the SDS to find out product components. Not all SDSs for the same product are equivalent, so keep looking until you find one that lists all or most of the ingredients. I search on the internet a lot.
- Ask the patient what he or she thinks is the cause of the rash – patients are often very insightful. Does anyone else have the same problem at work?
- Consult lists of patch test extracts specific to an occupation or exposure – these may give you an idea of where to look as regards your patient. Such lists are available on the websites of all major suppliers of patch test extracts.
- Use the history to guide the skin testing. You don't have to patch test to every material used in the patient's workplace if he or she only implicates one or a few products, or isn't exposed to most of the agents used in the workplace. If the rash is transient, lasts a few hours, but is usually resolved by morning, then use prick skin testing to assess a probable IgE mediated clinical course. If the rash comes on slowly, and takes days to weeks to resolve, then use patch testing to evaluate a probable type IV contact dermatitis type reaction.
- You can patch test to the actual product, provided you follow some safety rules: read the SDS, make sure the agent is not caustic or irritating, and use protocols from reports of how such testing has been done and reported elsewhere in the medical literature.

Treating occupational contact dermatitis

The treatment of work-related skin disease is the same as for environmentally triggered skin disease. The key is identifying, and then removing the specific exposure from the patient. Most workers prefer to remain in their chosen profession – they are often skilled experts who do not want to change careers or occupations. The best approach is to remove the exposure, by restriction, e.g. cannot enter the room with the enzyme solutions, or substitution, e.g. replace with rubber accelerator free gloves, or engineering controls, e.g. use a down draft table for nail work. Sometimes it is not possible to do so, either for administrative reasons or because the disease is severe, and the worker will be forced to leave his or her job. But this is the least agreeable solution, and often results in significant economic and social losses for the affected person. Treatment with topical steroids and steroid sparing agents, barrier creams, and immunosuppressive agents is the same as for other dermatitis, although most patients do not improve if they remain in exposure.

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Table 1. List of common irritants ⁷

Category		Examples
Cleaners		Water/wet work, soaps, detergents, hand cleaners, degreasing agents, bleach, isopropyl alcohol, benzoyl peroxide.
	Hand cleaners	Active ingredients = ethanol or isopropanol, also include humectants (glycerin), thickening agents (polyacrylic acid), fragrance oils
	Degreasers	Petroleum, alcohol, or chlorine based. Ex: sodium tripolyphosphate, sodium meta silicate, sodium carbonate
Acids/bases		Hydrofluoric acid, chromic acid, phosphorus, ethylene oxide, phenol, fluorohydrogenic acid, nonanoic acid, metal salts
Industrial solvents	Petroleum based	Benzene, toluene, xylene, ethyl benzene, butyl acetate, gasoline, kerosene, turpentine, Stoddard solvent, hexane, carbon tetrachloride, methylene chloride, methyl alcohol, ethyl alcohol, glycidyl ethers, dimethyl sulfoxide, etc.
	Acids	Oxalic acid, formic acid, salicylic acid,
	Bases	Sodium hypochlorite, alkalis, anhydrides
Chemicals	Metals	Sodium, potassium, lithium, phosphorus, bromine, iodine, aluminum chloride
	Others	Ethylene oxide, propylene glycol, sodium lauryl sulfate, butanediol diacrylate, hexanediol diacrylate, benzalkonium chloride, epichlorhydrin, octyl gallate
Plants	Euphorbiaceae	Spurges, croton (house plants), poinsettias, machineel tree
	Racunculaceae	Buttercup
	Cruciferae	Black mustard
	Urticaceae	Stinging nettles
	Solanaceae	Pepper, capsaicin
	Opuntia	Prickly pear
	Furocoumarins	Celery, citrus: bergamot, bitter orange, grapefruit, lemon, lime
Materials	Contact	Fiberglass, wool, fire-retardant fabrics, noncarbon copy paper
	Airborne	Acids, alkalis, ammonia, arsenic, calcium silicate, cement, cleaning products, epoxy resins, formaldehyde, paper, phenol formaldehyde resins, sawdust, urea formaldehyde insulating foam, tear gas
Others	Skin Products	Cosmetics, oils, greases,
	Medications	Benzoyl peroxide, tretinoin, diclofenac, podophyllin

From Cashman M, Reutemann P, Ehrlich A. Contact Dermatitis in the US: epidemiology, economic impact, and workplace prevention. *Dermatol Clin* 2012; 30: 87–98.

Table 2. Differentiating irritant from allergic contact dermatitis⁽⁵⁾

Factor	<i>Irritant Contact Dermatitis</i>	<i>Allergic Contact Dermatitis</i>
Appearance		
Acute	Erythema, edema, occasional vesicles, oozing, pustules Necrosis, ulceration if corrosive material	Edema, vesicles, oozing
Chronic	Dryness, roughness, glazing May have hyperkeratosis, desquamation lichenification, fissuring	May have hyperkeratosis, desquamation lichenification, fissuring
Location	Sharp edge, limited to contact area	More intense in contact area, but may extend beyond, limits are poorly defined
Symptoms	Burning, stinging, prickling, pain, and soreness; pruritus may be present	Pruritus is the main symptom
Clinical Course	Usually appear rapidly after 1 st exposure Delayed reactions can be seen	Sensitizing exposure, with period of no symptoms, is required Lesions appear 24 – 72 h after exposure, but may occur within 5 h or up to 7 d after
Time Course	Decrescendo time course: reach peak quickly, then start to heal	Crescendo time course: reach peak slowly with slower resolution

From Ale I, Maibach H. Diagnostic approach in allergic and irritant contact dermatitis. Expert Rev Clin Immunol 2010; 6(2): 291-310.

Table 3. Common allergens of health care workers

Example/derivatives	
Type I: evaluate by prick skin testing	
Latex	In US: glove usage test, prick-prick method; RAST
Enzymes (B. subtilis derived)	Biozyme, Endozyme
Type IV: evaluate by patch testing	
Quaternary ammonium compounds	Benzalkonium chloride
Rubber accelerators	Thiuram mix, Carba mix
Biocides	Bronopol, Thimerosal, Glyoxal
Sterilizers	Glutaraldehyde, Cidex (orthophthalaldehyde)
Formaldehyde and releasers	Quaternium-15, DMDM hydantoin, imidazolidinyl urea, diazolidinyl urea, Bronopol

Table 4. Common contact allergens in cosmetology

	Use/examples
<i>Name of allergen</i>	<i>Uses</i>
Para-phenylene diamine	Black hair dye
Ammonium persulfate	Bleaching agent
2-hydroxyethyl methacrylate	Acrylic nails
Tosylamide-formaldehyde resin	Nail polish
Fragrance	Lotions, shampoos
Formaldehyde and formaldehyde releasers	Quaternium-15
Lanolin	Lotions
Parabens	Preservative
Propylene Glycol	Preservative
Methyl isothiazolinone and Methyl chloro isothiazolinone	Preservatives
Plant derived extracts	Tea tree oil, Balsam of Peru

Table 5. Common Sensitizers in the Cleaning Industry

	Examples
Glove components/rubber accelerators	Thiuram mix, carba mix, mercaptobenzothiazole
Disinfectants: quaternary ammonium compounds	Benzalkonium chloride
Biocides: formaldehyde and releasers	Quaternium-15, DMDM hydantoin, imidazolidinyl urea, diazolidinyl urea, Bronopol
Biocides: glutaraldehyde	
Other biocides	Chloroxylenol, parabens, iodopropynyl butylcarbamate

483 **Table 6. Common metal extracts for occupational contact dermatitis**

Nickel sulfate hexahydrate	2.5% and 5% petrolatum
Cobalt (II) sulphate	2.5% petrolatum
Cobalt (II) chloride hexahydrate	1% petrolatum
Potassium dichromate	0.25%, 0.5% petrolatum
Titanium dioxide	10% petrolatum
Titanium (III) nitride	5 %petrolatum
Titanium (IV) oxalate hydrate	5% petrolatum
Iridium(III)chloride trihydrate	1% petrolatum
Indium (III) chloride	10% aqueous/1% petrolatum
Indium (II) sulfate	10% aqueous
Mercury	0.5% petrolatum
Mercury (II) chloride	0.1% petrolatum
Palladium (II) chloride	1% or 2% petrolatum
Sodium tetrachloropalladate (II) hydrate	3.0% petrolatum
Ammonium hexachloroplatinate (IV)	0.1% aqueous
Ammonium tetrachloroplatinate (II)	0.25% aqueous

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487 **Photos for MS No 18-03-0167.**

488 Patient 4. Prick skin test to QACs



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490 Patient 5. Floral worker hand rash



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492 Patient 6. Platinum induced rash and patch tests



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497 Patient 7. Firefighter with boots and chromium rash



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