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Occupational contact dermatitis

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Skin reactions are common in the workplace. The skin, which constantly is exposed to chemicals and industrial processes, develops inflammatory reactions, most commonly dermatitis, which may have an allergic or irritant basis. Occupational skin reactions are one of the most common of all occupational disorders. This article discusses allergic and irritant dermatoses, contact urticarial reactions, their incidence, causes, the industries in which they most commonly are found, diagnosis, and treatment.

Epidemiology

Data about the incidence and prevalence of occupational skin disease (OSD) and the industries in which workers are most at risk for OSD are available from a variety of national, state, and clinical sources. Such sources include the United States Bureau of Labor Statistics (BLS) annual survey, state-based worker's compensation systems, and published clinical data. OSD may be viewed primarily as occupational contact dermatitis (OCD), because most cases (86%–97%), whether irritant or allergic in nature, involve contact dermatitis [1–3]. Investigations provide new insight into the epidemiology of contact dermatitis in the workplace. Data may not be directly comparable between databases, but taken together the data provide an overview of the magnitude of work-related skin problems.

The BLS annually publishes the number of work-related illnesses and injuries reported to the Occupational Safety and Health Administration [4]. Criteria for reporting illnesses and injuries in the workplace were outlined in 1972 [5]. For most of the first 2 decades of reporting, skin diseases were the most common work-related illness. Skin disease ranks as the second most common occupational illness, ranking behind disorders associated with repeated trauma and ahead of respiratory illnesses and poisonings, in the United States, the United

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Kingdom [6], and Sweden [7]. The number of cases in the United States declined from 85,000 to 90,000 per year in the early 1970s to a nadir in 1983 of approximately 40,000 cases. This change may reflect improvements in health and safety on the job [8]. For unknown reasons, the incidence of OSD rose, peaked in 1992, and declined again to significantly lower levels. In 2000, there were 41,800 cases of work-related skin diseases, representing 11.5% of all work-related illnesses. This amount results in an incidence of 4.6 cases of OSD per 10,000 full-time workers during that year. This incidence of OSD is similar to that in Northern Bavaria [9]. The current US rate is slightly lower than the rate of 4.9 cases per 10,000 full-time workers in 1999 and is two thirds the rate of 6.9 cases per 10,000 full-time workers in 1996. Although the official data reported to the US government represent workers in all private industries, the true prevalence of OSD may be underestimated by a factor of 10 to 50 [10]. Surveys of workers in high-risk industries reveal a 1-year prevalence of skin disease of 5% to 20% [8].

Differences in job classification make direct comparison of reported data difficult, but it is clear that workers in certain industries have a higher risk for OSD. According to 2000 BLS statistics for the 10 major standard industrial classification sectors, the manufacturing sector has the highest number of cases of OSD at 18,600, and the agriculture, forestry, and fishing sector has the highest incidence of OSD of 15.5 cases per 10,000 full-time workers [1]. Service industries represent a significant source of OSD, with 14,000 cases reported. In these three sectors, the current rates represent a significant drop from the rate 5 years ago [8]. In Washington State from 1989 to 1993, the highest rate of accepted workers' compensation claims for OSD was in the agriculture, forestry, and fishing sector (2.8 claims per 1000 full-time employee-years) [11]. This sector is followed by manufacturing at 1.8, and construction at 1.3 claims per 1000 full-time employee-years. Review of specific industry subsets shows that workers in automotive glass replacement and beauty shops have high rates of accepted claims. In Northern Bavaria, where a different job classification scheme is used to track OSD cases, the highest incidence is seen in hairdressers and barbers (97.4 cases per 10,000 workers per year), followed by bakers, florists, and pastry cooks [9]. In the United Kingdom, where data also is kept according to gender, women who work as hairdressers, in laboratories, on assembly lines, in kitchens, and in healthcare have the highest rates of OSD among female workers, and men who work as printers, machinists and chemical plant operatives have the highest rates among male workers [6]. In Singapore, workers in the metal and engineering, building and construction, and electronics categories had the highest rates of OSD, each representing about 16% of total cases [1].

Work-related skin problems are overwhelmingly caused by contact dermatitis [1–3,11]. It has been reported that 80% of cases of OCD are attributable to irritant contact dermatitis (ICD) and 20% to allergic contact dermatitis (ACD). The BLS does not keep track of the causes of OCD, so data regarding the prevalence of these two forms must be derived from other sources. In support of

the commonly quoted distribution, a study of healthcare workers calculated the ratio to be 4:1 in favor of ICD [12]. Much published data that is based on experiences in contact-dermatitis specialty clinics suggest that ACD is responsible for more than the typically quoted 20% of OCD cases. Over a 10-year period in the Contact and Occupational Dermatoses Clinic at the National Skin Center in Singapore, 97% of all cases were contact dermatitis (61.2% of the cases were ICD, and 36% were ACD) [1]. In all but two industry subsets in this study, ICD was more common than ACD. In studies from other centers, the incidence of work-related ICD is equal to that of ACD [13,14]. Over a 5-year period in one of the North American Contact Dermatitis Group (NACDG) centers, 60% of patients with work-related skin disease had ACD, and 34% had ICD [3]. This center's data reflect that seen in the other 11 NACDG centers [2]. Over a 3-year period, 29% of patients evaluated in the clinics had work-related skin conditions, 54% of which were ACD and 32% of which were ICD.

Pathophysiology

The mechanisms underlying the development of ACD and ICD have been traditionally divided into immunologic and nonimmunologic pathways, respectively. ACD is a classic example of a type-IV delayed hypersensitivity reaction that requires sensitization to an allergen before development of a clinical response. In ICD, an irritant substance elicits a perceptible eruption in the skin without any preceding sensitization. Although the initiating factors of each type of contact dermatitis differ, the mediators of skin inflammation in each clinical setting overlap significantly. Chemicals are not exclusively irritant or allergic in nature. The mechanism of contact urticaria is discussed briefly.

Allergic contact dermatitis begins with a sensitization phase in which small molecules pass through the stratum corneum and are taken up by Langerhans cells residing in the epidermis. Substances that penetrate the epidermis to cause ACD are small—usually less than 1000 Da—uncharged, and fairly hydrophobic [15]. The molecules are haptens, which must interact with a protein to induce an allergic response. The ingested hapten is processed in lysosomes and expressed on the surface of the Langerhans cell in association with an HLA-DR (MHC class II) molecule. Some cutaneous allergens, such as nickel, may interact directly with HLA-DR molecules already present on the surface of Langerhans cells [16]. Within 24 hours, Langerhans cells leave the epidermis and make their way through the afferent lymphatics to the regional lymph nodes, where antigen is presented to naïve $CD4^+$ T cells called $CD45RA^+$ cells. Appropriate binding of the HLA-DR/hapten complex to the CD3/CD4/T-cell receptor complex activates the T cells, which become memory and effector $CD45RO^+$ cells and, under the influence of interleukin 2 (IL-2), increase in number. The duration of the sensitization phase varies according to the allergen, but is typically 1 to 2 weeks long.

Rechallenge with antigen begins the elicitation phase. Langerhans cells present antigen to large numbers of circulating $CD45RO^+$ memory and effector

T cells that were created by sensitization, which become activated and expand further in number. Expression of common leukocyte antigen on the surface of activated T cells allows the cells to migrate to the skin, where they exert their effects. An extensive cascade of cytokines and other inflammatory mediators released from activated T cells, keratinocytes, and mast cells causes the clinical appearance of ACD [17]. $CD4^+$ and $CD8^+$ T cells have demonstrated an ability to induce and carry out contact hypersensitivity reactions [18]. The antigen itself and the manner in which it is recognized may dictate the role for each T-cell subtype. The elicitation of ACD typically occurs within 48 to 72 hours of rechallenge.

Irritant contact dermatitis results from skin-barrier disruption and subsequent release of inflammatory mediators [19]. Although irritant skin reactions depend on certain host factors, they do not require previous sensitization. Highly irritating chemicals are alkaline materials, strong acids, and powerful oxidizing and reducing agents [15]. The irritation potential of a substance also depends on its concentration, the vehicle, and the duration of exposure [17]. Depending on the type of irritant contacted, there is a broad range of disruptive effects on the epidermis, including denaturation of keratins, lipid-barrier removal, cell-membrane damage, and direct cytotoxicity [20]. Disruption of the stratum corneum and its barrier lipids results in the release of proinflammatory cytokines from damaged cornified cells and from viable keratinocytes that become activated. Most of the cytokines previously linked with ACD, such as intercellular adhesion molecule 1, lymphocyte function-associated antigen (LFA)-1, IL-1, tumor necrosis factor (TNF-α), and interferon γ have been found in irritant reactions [20]. Of these, TNF-α may be the major mediator of inflammation in ICD [21]. As a result of the release of these cytokines, activated T cells are present in the epidermis and dermis in irritant reactions [20].

Mild irritants, such as water, soaps, and detergents, typically cause chronic, subclinical irritation, which is cumulative and eventually leads to clinically perceptible dermatitis [19]. The loss of the stratum-corneum lipid barrier in these situations results from a loss of cohesion of corneocytes and desquamation and causes a measurable increase in transepidermal water loss (TEWL). This change stimulates epidermal turnover and hyperplasia. Although dermatitis clears clinically 2 weeks after experimentally induced acute or chronic ICD, the skin remains damaged subclinically according to TEWL and skin-color reflectance measurements [22,23]. The skin continues to be significantly more prone to subsequent irritation than unaffected skin for 4 weeks after acute ICD resolves and for at least 10 weeks after chronic ICD resolves. These data support the hypothesis that repeated low-grade insults to the skin that occur before the skin is healed completely will accumulate and cause ICD [24].

The mechanisms that lead to urticarial responses to contactants have been reviewed [25]. Immunologic contact urticaria (ICU) is a type I hypersensitivity reaction. In a previously sensitized individual, an allergen contacts and penetrates the skin, binds to immunoglobulin E (IgE) on mast cells, and causes degranulation. The release of inflammatory mediators, including histamine, prostaglan-

dins, leukotrienes and kinins, causes a range of responses, from localized urticaria to anaphylactic shock. Nonimmunologic ICU (NICU) does not require activation of the immune system to elicit a response. Contactants cause direct release of prostaglandins and other vasoactive mediators, leading to a range of clinical findings, from erythema or pruritus alone to a wheal and flare response. NICU and ICU may be mild and over time can lead to changes in the skin that are indistinguishable from ICD or ACD.

Predisposing factors

An intact stratum corneum, a protective barrier layer of compact, dead, keratinized epidermal cells, has a crucial role in preventing potential allergens from initiating ACD and in preventing irritants from constantly inflaming the skin. Several factors in the workplace may have a role in altering the stratum corneum, leading to a predisposition to occupational skin reactions (Box 1). Work

Box 1. Risk factors for contact dermatitis

Exogenous factors

- Frequent contact with:
  - Water
  - Soap and water
  - Organic solvents, greases, cutting oils, chromates, rubber
- Environmental factors
  - Heat and high humidity
  - Low humidity and cold temperatures
  - Repetitive motion and frictional forces
  - Ultraviolet light
- Personal factors
  - Nonuse of protective clothing and gloves
  - Inadequate washing of skin and clothing

Endogenous factors

- Underlying skin diseases
  - Atopic dermatitis, psoriasis, icthyosis
- Coloration of skin
  - Fair skin prone to contact reactions
- Areas of skin exposed to work materials
  - Dorsum hands and feet more easily affected than thick palms and soles

that requires frequent immersion in water causes maceration, and frequent wetting and drying leaches stratum corneum proteins, which in turn causes breaks with chapping, scaling, and fissuring. These effects allow allergens to have ready access to the skin. Wet drying alone is a common cause of irritant dermatitis, and soaps and detergents accentuate these reactions.

In hot, humid work environments, workers are less likely to wear protective clothing and gloves, and increased perspiration solubilizes chemicals that are deposited on the skin, allowing them to penetrate the stratum corneum. Alternately, low-humidity environments cause chapping and desiccation with the similar breaks in the protective skin barrier. Working with organic solvents, which can damage the skin, also predisposes workers to contact dermatitis.

Diseased skin with inflamed and altered epidermis (such as atopic dermatitis or psoriasis) also increases the chances of superimposed contact dermatitis. Compared with patients with non-OCD, patients with OCD are younger; less likely to have atopy; and more likely to be male, have hand and arm dermatitis, and show positive patch test responses to metals, rubber compounds, paraphenylenediamine, and epoxy resin. Compared with patients with irritant OCD, patients with allergic OCD cases are more likely to be female; have atopy; and have dermatitis of the hands, arms, and eyelids [26].

Underlying psoriasis may complicate industrial dermatoses because of an unusual feature called the Koebner phenomenon (the evolution of psoriasis plaques in areas of normal-appearing skin that is exposed to pressure and friction). Pressure and friction from occupational jobs may induce new lesions of psoriasis on the palms and volar areas of the fingers. Scratches, abrasions, lacerations, and trauma incite the development of new lesions, but only in persons with a latent form of psoriasis. Lichen planus also produces industrial Koebnerization.

Evaluation and diagnosis

The most common occupational skin reaction is dermatitis, and more than 90% of such cases involve the hands and forearms [27]. Such reactions are allergic or irritant in nature. Acute or chronic contact eczematous reactions can occur. The acute phase of dermatitis, whether allergic or irritant, appears as erythema, edema, and vesiculation, with subsequent exudation, crusting, and increased scaling (Fig. 1). Pruritus is intense. In more chronic forms, there is erythema, but scaling and fissuring are more pronounced, and lichenification (thickening of the epidermis with marked accentuation of the skin creases) that is caused by constant rubbing and scratching is usually present (Fig. 2). It is often impossible to use appearance to differentiate between allergic and irritant contact reactions, although in both the reactions occur in the areas of occupational contact. A careful history, physical examination, and patch testing should be performed to establish that the dermatitis is an occupationally related condition, to determine if it is allergic or irritant in nature, and to identify the specific allergens or irritants that are causing the problem.

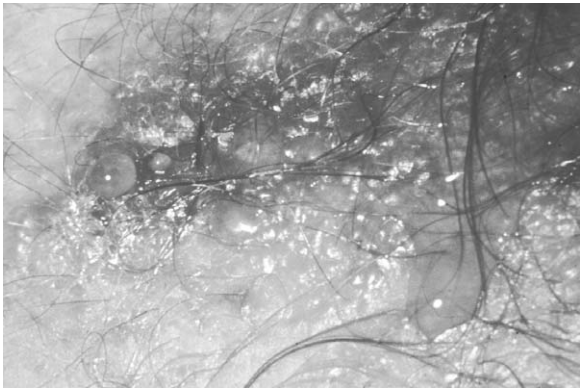

Fig. 1. Acute contact dermatitis with erythematous papules and clear vesicles and bullae. (See also Color Plate 1.)

Mathias suggested seven criteria for probable occupational causation of a skin rash [28]. Answering yes to at least four of the questions may provide probability for workplace exposure. The questions are as follows:

1. Is the appearance consistent with contact dermatitis?
2. Are there workplace exposures to potential cutaneous irritants or allergens?
3. Is the anatomic distribution of dermatitis consistent with the exposure a worker would obtain on the job?
4. Is the onset and time course of the eruption consistent with contact dermatitis?
5. Are nonoccupational exposures excluded as possible sources of dermatitis?

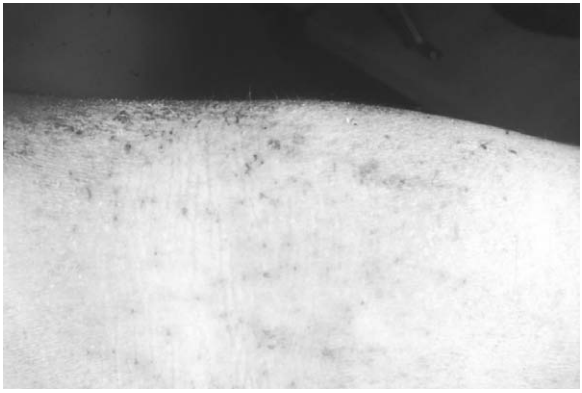

Fig. 2. Chronic dermatitis displaying erythema and numerous excoriations. Prominent lichenification is seen as leathery thickening of the skin and prominence of fine skin lines caused by constant rubbing and scratching. This reaction occurred in the antecubital fossa, a typical area of involvement in atopic dermatitis. (See also Color Plate 2.)

6. Does the dermatitis improve away from work and at home?
7. Do patch or provocative tests identify a probable causal agent?

The following suggestions for history and physical examination and the discussion of skin testing can help the examiner answer Mathias' criteria.

History

History of dermatitis

The patient's description of events can be important in the investigation. The date of onset of the rash; its rapidity of onset and description (immediate or hive-like lesions suggest a contact urticarial reaction, and burning, stinging, and redness that appear at the time of exposure suggest an acute irritant reaction). Chronic ICD can take longer to evolve, especially if the irritant is mild, such as in work involving constant water contact. Approximately 3 months of constant wetting and drying are needed before ICD develops. Allergic contact reaction can occur within weeks of exposure to a new material, or it may require years of exposure.

The anatomic site of skin lesions, especially at the onset of the dermatitis, may be crucial in the evaluation. The hands are the most common site, and which hand and what areas on the hands (eg, palm, dorsum, fingers) should be noted. The initial sites of the dermatitis should conform to skin sites exposed to suspected allergens or irritants on the job; as the skin process continues, other body areas may become involved that are not clearly related to skin exposure. Determining if the dermatitis improves away from work is another important Mathias criteria. Some chronic dermatoses may be slow to improve, even while away from work and receiving treatment.

Identifying the therapy that was used to treat the dermatitis, which may modify or worsen the condition, is important in assessing the problem. Topical treatments sometimes contain ingredients that cause irritation or allergic reactions (eg, Neomycin, topical steroids, preservatives), thereby perpetuating what might have been a self-limited problem.

Occupational history

It is important to identify the nature of the patient's current and previous jobs, exposure to materials, and physical environmental factors. Determining the time spent performing wet work and the frequency of contact with irritating chemicals are crucial. Material safety data sheets (MSDSs) can help identify materials and their side effects. Information regarding the physical environment of the work-place (temperature, humidity, airflow, exposure to ultraviolet light) may explain drying or chafing of the skin or explain any predisposing factors that increase penetration of materials into the skin.

Personal history

Determining whether the patient has a history of underlying diseases (atopic dermatitis, psoriasis, hand dermatitis) is important, because its presence often complicates the establishment of work-related reactions. The relative odds of developing work-related skin disease is 13.5 times greater for individuals with a history of atopic skin disease compared with those without this history, and the course of work-related dermatitis may be more prolonged [26]. Patients with psoriasis may develop new lesions on their hands if their jobs entail repeated friction or repeated trauma to the hands (Koebner phenomenon, discussed earlier). Patients should be queried about previous work exposure, episodes of dermatitis, hobbies, and secondary jobs to find other possible sources of allergies or irritants.

Physical examination

Although the physical examination should concentrate on the affected areas of skin that correspond to the site of the presumed workplace exposures, the examination also should include skin sites that were not directly related to the exposure. Evidence of other skin conditions, such as flexural atopic eczema (see Fig. 1); scalp, elbow, and knee psoriasis; and dermatophyte infections of the palms, soles, or nails, should be assessed.

Sometimes the configuration of the presumed contact site may be suggestive of a specific cause (ie, a linear configuration or a sharp cutoff at sites of protective equipment such as gloves or face mask). A rash on the hands that conforms to where gloves are worn suggests a dermatitis to materials involved in glove manufacture. The outline of the reaction is usually not specific, however, and not helpful in identifying the cause of the problem.

Fig. 3. ICD of the hand of a worker who has constant contact with water. Apron-like chronic dermatitis is seen, with dryness, scaling, and lichenification over the dorsum of the hand and fingers, plus intense reaction in finger webs. (See also Color Plate 3.)

Fig. 4. Chronic ICD in a food handler. Erosive lesions in finger webs and under ring are seen. (See also Color Plate 4.)

Because hand reactions are common in workplaces, they are often the focus of disagreement between employers and employees about whether the reaction is occupationally related. Most reactions are an acute or a chronic dermatitis, but occasionally the reaction is urticarial. It usually is not possible to clinically determine whether the dermatitis is irritant, allergic, or endogenous (atopic, psoriasis, tinea) by examining the patterns of localizations on a patient's hands. In the early stages of the condition, however, there may be some clinical clues. Irritant reactions that occur in workers who have constant exposure to water (eg, hair dressers, medical personnel, food handlers) often involve the finger webs, concentrate under rings, and react over the dorsum of the hands in an apron-like distribution (Figs. 3,4). Allergic contact reactions may begin as vesicles on the fingers tips (Fig. 5) [29]. Some investigators state that involvement of the dorsal skin of the hands may be more indicative of allergic or contact dermatitis, whereas palmar and lateral aspects of the digits suggest endogenous hand eczema, such as

Fig. 5. ACD involving the tips of thumb and fingers in a dentist who was allergic to Benzacaine. (See also Color Plate 5.)

atopic eczema or dyshidrotic dermatitis [30]. Variations in the thickness and keratinization of the skin may confound the distribution of reactions on the hands. The thick palmar skin is harder to penetrate, so although palmar and dorsal skin of the hands is exposed to irritants and allergens, only the dorsal surfaces are affected. Likewise, eyelid skin is thin and prone to airborne materials.

Contact immediate urticarial reactions occur in workers handling fish and meat and in contact with latex (particularly gloves) (Fig. 6). The wheal and flare reaction, which involves itching, stinging, and burning within minutes of contact, can be allergic (immediate, IgE mediated) or nonimmunologic. In the nonimmunologic form, many individuals may be affected despite the lack of previous exposure to the offending agent. Allergic contact urticarial reactions are more common in patients with atopy. Fish, foods (benzoic acid preservative), animal proteins, flavorings (cinnamic acid), and perfume ingredients (Balsam of Peru) are the usual sources of this type of reaction. Life-threatening anaphylaxis may accompany contact urticaria caused by latex. Patients may be sensitive to the materials used to manufacture latex in rubber gloves (mercaptobenzothiazide); these cell-mediated immune allergic reactions cause a delayed hypersensitive dermatitis rather than urticaria. The various features of irritant, allergic, and urticarial contact reactions are compared (Table 1).

Diagnostic tests

Skin biopsy

Although skin biopsies cannot differentiate between allergic, irritant, and atopic dermatitides, this procedure is helpful in excluding psoriasis and tinea infections (although a potassium hydroxide microscopic examination or culture of skin scrapings is best in identifying fungal or *Candida* skin infections).

Fig. 6. Urticarial contact dermatitis as a result of use of latex gloves. A sharp cut-off at the wrist is seen where glove ends. (See also Color Plate 6.)

Table 1
Generalizations of contact dermatitis

Variables	ICD	ACD	Urticarial contact dermatitis
Number of people affected	Many	Few	Nonimmunologic form affects many
Where condition occurs	Localized	Spreads	Localized and with anaphylaxis
When condition occurs after exposure	Rapid with strong irritants (minutes to hours); late with weak irritants (months)	24–72 h	Minutes
Signs of occupational disease	Improves with after 3 weeks away from workplace	May improve in a few days away from workplace but 50% of cases persist	Clears within hours of stopping exposure
Atopy	Predisposition	No predisposition	Immunologic form results in predisposition
Morphology	Red, scaling fissures	Vesicles; similar to that in ICD	Urticarial reaction and edema
Effect of chronic water exposure	May cause and perpetuate condition	May perpetuate condition	No effect
Immunology	None	Type IV	Type I
Chronicity	Often	If diagnosed early, condition clears; can be chronic	Often recurs with reexposure to antigen
Diagnosis	History and physical examination	Patch test	IgE blood test and patch or prick test to latex
Examples of irritants, allergens	Hairdresser, machinist Water, shampoo, solvents	Poison ivy, oak, nickel, chrome	Latex, fish

Patch testing

Patch testing is the means of identifying the specific allergens that cause the allergic reactions. It is an imperfect bioassay, because it cannot reproduce precisely the clinical environmental factors, such as sweating, friction, and chronic daily exposures. By using prepared standard common allergens and adding relevant industrial substances, allergic reactions usually can be identified. The most commonly used patch-testing method is the Finn Chamber, in which a small amount of each allergen is placed on the individual aluminum wells that are affixed to a strip of paper tape and placed on the back. The patches are removed in 48 hours, and the positions of the patches are outlined with marker pen. Reactions may occur after 2 days (ie, the earliest time that delayed hypersensitivity manifests itself on the skin); a second reading at 96 hours or 1 week after the initial application increases the yield of the positive patch test.

Erythema, edema, and vesicles that persist after patch removal or that appear 2 to 7 days after removal is typical of positive allergic patch-test reactions. Irritant reactions can appear to be similar, although they are more likely to appear as glazed, red, follicular, pustular, bullous, or ulcerative inflammatory responses (depending on how strongly irritating the substance is) that fade rapidly after initial patch removal.

Interpretation of patch tests must be done by trained and experienced clinicians, because even chemicals on Finn Chamber standardized trays can give irritant reactions, and because testing for allergens taken from the workplace, where the precise concentration of allergen is not known, may cause irritant reactions. Compounds taken from the workplace should be placed in petrolatum in approximately the same concentration that it is found in the work environment. Another approach to testing new materials is to reproduce the dermatitis with a use test. The material in question, unless it is irritating, is applied in the same concentration that the patient encounters in the workplace and is applied to the same small area of skin (usually on the inner aspect of the arm above the elbow) twice daily for 7 days. The patient must have stopped taking oral steroids for a week, and use of topical steroids should be stopped 1 day before. Intramuscular Kenalog (triaminolone acetonide, Bristol-Myers Squibb Co.) should not be used for a month before patch testing.

Prick or scratch tests

Prick or scratch tests may be used to diagnose suspected latex-related, immediate hypersensitivity urticarial reactions. Such tests may result in an anaphylactic reaction, so IgE RAST tests are safer, as is applying the substance in question (eg, for latex, using saline in which a piece of latex glove is soaked) to normal skin. Signs of itching, redness, and swelling in 0.5 to 2 hours indicate a positive reaction.

Common antigens and irritants

Common sensitizers and their locations found are listed (Table 2). Potential cases of occupational dermatitis are listed by industry and common antigens found in each occupation (Table 3). Agriculture, forestry, fishing, manufacturing, and construction have the greatest incidence of occupational skin reactions. Common industrial materials that may cause irritant dermatitis are detailed (Box 2), and the occupations at high risk for irritant dermatitis are noted (Box 3).

Other occupational skin diseases

Other forms of OSD can occur, include lichen-planus–like eruptions in photography developers and leukoderma (vitiligo) caused by hydroquinone, which is used in the photography development process [31]. Pigmented and

Table 2
Common allergens that cause ACD

Allergen	Source	Cross reactions/comments
Rubber (mercaptobenzothiazole, thiuram, latex)	Surgical gloves, dental dams, medical tubing, endoscopic instruments, anesthesia masks, balloons	Latex causes immediate contact urticarial responses, anaphylaxis; other materials cause delayed ACD
Paraphenylenediamine	Hair and fur dyes, leather processing, rubber vulcanization, printer ink, photography and lithography, radiograph fluid	Benzocaine, procaine, paraminobenzoic acid, diuril, sulphonamides (cross reactions)
Nickel sulfate	Instruments, tools, shoes, hair dyes, bleaches, electric wiring, jewelry, insecticides, fungicides, nickel plating	Ubiquitous throughout environment
Chromium	Cement, textile inks, paints, varnishes, leather processes	Causes recalcitrant dermatitis
Ethylenediamine	Dyes, rubber, waxes, resins, asphalt, preservative in cream medications	Pyribenzamine, aminophylline, and piperazine cross react
Thimerosol	Cosmetics, eyedrops, vaccines	—

purpuric reaction can occur in textile workers as a result of exposure to formaldehyde resin [32].

Visits to industrial plants

Although MSDSs are useful for identifying chemical hazards of a workplace, additional information can be obtained from visiting an industrial plant. Familiarity with work materials, job processes, and the industrial-plant environment enables physicians to more readily identify factors that were obtained from the patient's history.

Prognosis

Occupational contact dermatitis (OCD) may occur as a self-limited event or become a chronic, disabling problem. It is important to understand what contributes to the development of chronic skin disease in order to help patients manage their condition well and maintain their employment. The main factors that determine the prognosis of OCD are the type of contactant encountered, the

Table 3
OCD categorized by industry and common allergens

Industry	Allergen/chemical	Industry source	Other sources
Painting industry	Merthiolate, chloracetamide	Paint, wallpaper glues	Cosmetic creams, lotion
	Acrylates	Paints	—
	Chromates, nickel	Paints, pigments	Cement
	Dichlorofluamide	Fungicide	—
Printing industry	Acrylate compounds	Inks	—
	Epoxy resins, potassium dichromate	—	—
Agriculture (cattle breeders, dairy workers farmers)	Malathione	Pesticides	—
	Thiuram	Fungicides	Antabuse
	Dithiocarbamates	Fungicides	—
Forestry	Usnic acid	—	—
	Sesquiterpenes	Frullania, chrysanthemum, tansy, ragweed	Florist industry
Bakers	Ascorbinic acid, bleaches, enzymes, emulsifying agents, wet dough, yeast[a]	—	—
	Flour, sorbic acid, enzymes in flour[b]	—	—
	Chromates, benzoyl peroxide in flour[c]	—	—
Hairdressing[d]	Hair dyes (paraphenylenediamine), fragrances, nickel, Balsam of Peru	Dyes, perfumes	—
	Rubber materials (thiuram, mercaptobenzothiazole)	Rubber materials, shampoos	—
	Glycerol thioglycolate	Hair-waving chemicals	—
	Ammonium persulfate	Bleaches	Bleaches in textile industry
Electronics	Epoxy resins, ammonium bichromate, cobalt, platinum	Magnets, connectors, glue	—
	Solvents (xylene, acetone, freon, isopropanol), solder (rosiin, hydrazine)	—	—
Construction	Chromate	Cement	—
	Cobalt, nickel, epoxy resin, rubber (thiuram)	—	—
Healthcare workers: surgeons	Surgical scrubs (hexachlorophene)	irritant most often	—
	Acrylic bone cement, rubber gloves	Orthopedic surgery	—

(continued on next page)

Table 3 (*continued*)

Industry	Allergen/chemical	Industry source	Other sources
Healthcare workers: dentists	Topical anesthetics, acrylic monomer, amalgams (mercury)	—	—
	Formaldehyde	Sterilizing purposes	—
Machinist and industries using cutting oils, cooling and drilling fluids	Ethylenediamine, triethanolamine	Cutting oil	Preservative; cross-reaction between aminophylline and rubber
	Grotan	Bacteriocide	Insecticides, fungicides
	Mercaptobenzothiazole	Cutting oil	Rubber

[a] Associated with ICD, paronychia, dyshidrotic eczema.
[b] Associated with urticarial contact dermatitis.
[c] Associated with ACD.
[d] ICD and ACD often occur simultaneously.

patient's occupation and whether a change from that job was made, and the patient's history of atopy [33]. It is possible that gender and age play a role as well.

Not only has the overall incidence in reported cases of OCD improved over recent years [4], but so too has the prognosis [14,26,34]. Whereas studies carried out prior to the last decade quoted response rates of somewhere between 30% to 50% of patients [35], more recent reviews show that 78% to 84% of patients with OCD improve [14,34]. Still troubling is that roughly 15% to 20% of patients have not improved, or have worsened when evaluated two to four years after diagnosis. Goh [33] suggests that the improved prognosis of OCD may be due

Box 2. Industrial materials that may cause ICD

Petroleum distillates
Alkalis, acids
Fuels
Soaps, detergents, cleaning agents
Organic solvents
Ketones
Turpentine
Water (with frequent use)
Alcohols
Cutting fluids
Chlorinated hydrocarbons
Glycols

Box 3. Occupations at high risk for ICD

Housekeepers
Doctors, dentists
Food service workers
Machinists
Hairdressers
Printers
Mechanics
Agricultural workers, gardeners

to better diagnostic procedures, more accurate identification of contactants, and increased education and preventive efforts.

Studies comparing the prognosis of occupational irritant contact dermatitis (ICD) to occupational allergic contact dermatitis (ACD) have had varied outcomes, showing that ICD has a better, equal to, or worse prognosis than ACD [35]. A direct comparison of the two forms of OCD is difficult to make. ACD broadly encompasses sensitivity to numerous different compounds, which vary in their ability to cause chronic dermatitis and in their prevalence outside of the workplace. Nickel, chromate, and glutaraldehyde contact allergies carry with them a poor prognosis, whereas sensitivity to epoxy resins has a favorable prognosis [35]. The avoidance of ubiquitous allergens such as nickel, chromate, and rubber chemicals is a problem that may contribute to chronicity in contact allergies to them [33]. ICD may result from similarly varied irritants. ICD to cutting fluids and solvents tends to cause persistent dermatitis despite avoidance, whereas ICD to acids and alkalis most often clears after exposure is avoided [36]. Other work has shown that ICD may have a worse prognosis than ACD [14]. In long-term follow-up, symptoms of patients with ACD are more likely either to have improved significantly or have completely resolved than those of patients with ICD. Longer duration of exposure to both irritants and allergens appears to increase the likelihood of developing chronic dermatitis [34].

Patient factors contribute to the overall prognosis of OCD as well. Atopic patients are more susceptible to ICD and generally fare worse than non-atopic patients [35]. When assessed by questionnaire an average of four years after diagnosis, workers with atopy were more likely to have persistent symptoms. Women reported better outcomes than men in the same study [14]. In a separate study, gender was not found to be a factor in persistence of ICD, but 90% of men with ACD cleared compared with only 50% of women [36]. A third study failed to find a difference in prognosis between men and women [34]. Age does not appear to be related to chronicity of symptoms [14,34], but older patients seem to be more likely to spend time away from work as a result of their skin disease [34].

Although one would intuitively expect for a patient's work-related contact dermatitis to improve with a change in jobs, many studies have not found this to be

the case [35]. The factors responsible for persistent dermatitis in those situations reviewed are not clear, but those who eliminate exposure to relevant contactants through a job change have a better prognosis than those who do not [33]. Construction workers with ACD appear to have the worst prognosis compared to patients with other occupations [33]. Some workers will continue to work despite persistent dermatitis, and symptoms may actually resolve in the setting of continued exposure to offending agents [33,35,36].

Data retrieved from a physician-based surveillance system for occupational skin disease in the UK (EPIDERM) were evaluated to better define the risk factors for spending time away from work due to OCD [34]. Two years after their diagnosis, information regarding cases was obtained via questionnaires sent to diagnosing physicians. Twenty-one percent of patients with OCD had spent time away from work due to either unemployment or sick leave. Of these patients, 35% were off work less than three weeks, and 54% were off from three weeks to one year. The main risk factors for spending time away from work were older age, an allergic etiology, and whether a medicolegal claim had been made. Those patients filing claims were four times as likely to have taken time off from work. Factors that increase the likelihood of a person returning to work include a worker's pre-injury job satisfaction, skill level, and education regarding avoidance of the contactant [35].

Management

Patients with OCD can develop acute or chronic symptoms, each of which requires different therapy [37]. Widespread acute allergic contact dermatitis (ACD) readily responds to systemic corticosteroid treatment providing the patient with prompt relief of symptoms. Adequate doses of prednisone, typically 40 to 60 mg per day, may be given, and then should be followed by a taper over the course of about two weeks. Involvement of limited areas of skin is treated easily with potent topical steroids two to three times daily. Examples of these include triamcinolone acetonide, betamethasone dipropionate, and clobetasol propionate. An ointment base should be used to minimize the risk of sensitization or cross-reaction to preservatives present in cream bases. Topical drying agents may speed the healing of lesions that weep significant amounts of serous exudate. Clean dressings soaked in aluminum and calcium acetate solutions are applied to affected areas for 20 to 30 minutes several times daily. Lukewarm, soapy water is an effective alternative to the previously mentioned solutions. Bathing in colloidal oatmeal (eg, Aveeno, Johnson & Johnson, New Brunswick, NJ) also helps relieve the symptoms of widespread involvement. The pruritus that accompanies acute contact dermatitis does not respond well to oral antihistamines; however, their sedating effects are useful when taken at night. Evidence of bacterial superinfection, such as honey-colored crusts or pustules, should be treated with oral antibiotics that cover staphylococci and streptococci.

The treatment of irritant contact dermatitis (ICD) often begins after symptoms have become chronic, and differs somewhat from the treatment of acute ACD. The mainstay of treating ICD is frequent moisturization and avoidance of irritants. Moisturizers prevent the development of experimentally induced ICD and enhance the rate of healing on damaged skin [38]. The best agents for this purpose are ointments or creams that contain the fewest number of sensitizing agents possible. Examples include plain petrolatum (Vaseline), Neutrogena Norwegian Formula Hand Cream, and Cetaphil Cream (Neutrogena Corp, Los Angeles, CA). Lotions that contain primarily water, potential sensitizing preservatives, and fragrances, are not effective emollients for patients with abnormal skin, and should be avoided. Application should occur frequently and especially after contact with water or other irritants. Hands should be washed as infrequently as possible using a mild soap and lukewarm water, taking care to rinse thoroughly. Alternatively, one can use a moisturizing waterless cleanser such as Cetaphil Cleanser (Galderma Laboratories, Fort Worth, TX), which does not need to be rinsed off with water. Data regarding the efficacy of topical steroids on ICD has been mixed in the past, but the results of a recent study support the traditional use of topical steroids to treat the inflammation associated with ICD [39]. Application of a potent topical steroid, betamethasone-17-valerate, to experimentally induced irritant skin reactions resulted in improved healing compared to treatment with vehicle alone at one week. The topical steroid should be used in an ointment base to avoid sensitization, especially considering that patients with ICD are at increased risk of developing ACD.

Appropriate management of patients with OCD involves both treatment of symptoms and avoidance of further encounters with contactants. Patients should be given information regarding the allergens to which they are sensitive, including where they might be found, the trade names under which they might be listed, and all cross-reacting substances. Care should be taken to avoid allergens both in the workplace and at home. Test kits are available to identify the presence of some allergens, such as nickel (dimethylglyoxime test), chromate, and epoxy resins. Patients with both ICD and ACD need to avoid irritants, which can exacerbate both forms of contact dermatitis. Educating patients extensively about exposures they need to avoid results in a better outcome [35].

The use of protective agents such as gloves and barrier creams may allow a worker to continue in the same job without the further exacerbation of skin disease. Gloves are a highly effective means of preventing contact dermatitis, but careful attention to certain factors must be addressed when using them in the workplace [40]. Some sensitizers, such as nickel, glyceryl monothioglycolate, epoxy resins, acrylic monomers, and tuliposide A can penetrate through gloves and cause ACD. Patients may develop a type I hypersensitivity reaction to natural rubber latex, or a type IV reaction to accelerators such as thiurams, carbamates or mercaptobenzothiazole, or antioxidants such as para-phenylenediamine derivatives. Gloves can also contribute to the development of ICD. Powders present on some gloves can be irritating or sensitizing. In general, only gloves made of plastic or synthetic rubber materials should be worn, and for as little time as

necessary. When substitution of a contactant is not possible, and gloves impair a worker's performance, protection may be provided through the use of barrier creams. Data regarding the efficacy of barrier creams in clinical practice are inconsistent [41]. In one study of health care workers, a barrier cream was no better than its moisturizing vehicle at improving irritant hand dermatitis [42]. Similar to the caveat with gloves, it is important to recognize that barrier creams themselves can induce ICD or ACD [40].

If an offending agent cannot be avoided or substituted, a job change and relevant re-training may be recommended. All prospective jobs should be carefully reviewed regarding possible sensitizers and irritants prior to making a switch.

Primary prevention of OCD is preferable to managing the disease once it has developed. Primary prevention depends upon efforts made by manufacturers of chemicals, employers, and the workers themselves [41]. The key factors in successful prevention of OCD involve recognition and regulation of potential irritants and allergens, creation of safe workplaces, prevention of skin exposure through the use of appropriate barriers, proper personal hygiene, and education and motivation of workers [43]. It may be that efforts at primary prevention thus far are responsible for the reduction in the incidence of OCD seen over the past decade [8].

Appendix

Resources for information on occupational dermatitis

General information

National Institute for Occupational Safety and Clearing House Information, 4676 Columbia Parkway, Cincinnati, OH 45226, USA

Consumer product safety hotline

Injury Information Clearing House, 1111 18th Street NW, Washington, DC 20210, USA

Environmental Protection Agency

Public Information Center, 401 M Street SW, Washington, DC 20460, USA

Manufacturers of occupational protective materials, protective ointments, and barrier creams

Mine Safety Appliances, 400 Penn Center Boulevard, Pittsburg, PA 15235, USA

Ayerst Labs, 685 Third Avenue, New York, NY 10017, USA

E.I. Dupont de Nemours & Company, 1007 Market Street, Wilmington, DE 19898, USA

Zee Medical Products, 11800 Woodruff Avenue, Downey, GA 90241, USA

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