Occupational Skin Disease

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Contact dermatitis, the most common occupational skin disease, is characterized by clearly demarcated areas of rash at sites of exposure. The rash improves on removal of the offending agent. In allergic contact dermatitis, even minute exposures to antigenic substances can lead to a skin rash. Common sensitizing agents include nickel and members of the Rhus genus (e.g., poison ivy, poison oak). Severe skin irritants tend to cause immediate red blisters or burns, whereas weaker irritants produce eczematous skin changes over time. An occupational cause should be suspected when rash occurs in areas that are in contact with oil, grease, or other substances. Direct skin testing (patch or scratch) or radioallergosorbent testing may help to identify a specific trigger. Skin cancer can have an occupational link in workers with prolonged exposure to sunlight and certain chemicals, although it can take decades for lesions to develop. In workers with occupational skin disease, workplace changes and protective measures are important to prevent future exposure. (Am Fam Physician 2002;66:1025-32,1039-40. Copyright© 2002 American Academy of Family Physicians.)

• A patient information handout on work-related skin problems, written by the author of this article, is provided on page 1039.

ork-related skin diseases account for approximately 50 percent of occupational illnesses and are responsible for an estimated 25 percent of all lost workdays. These

dermatoses are often underreported because their association with the workplace is not recognized.¹

Occupational skin diseases affect workers of all ages in a wide variety of work settings. Industries in which workers are at highest risk include manufacturing, food production, construction, machine tool operation, printing, metal plating, leather work, engine service, and forestry.^{2,3}

General Principles of Diagnosis

Because of the prevalence of occupational exposures that can cause or exacerbate skin disorders, it is advisable to screen all patients with skin disease for a work-related cause. If occupational skin disease is suspected, questions should be asked about the exact time

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relationship between the skin condition (i.e., onset, improvement, and recurrence) and the work exposure, including the effects of time off and return to work. An in-depth occupational history should cover the following points:

1. General work conditions (e.g., heat, humidity) and specific activities in the patient's present job that involve skin contact with potential hazards. Note that Material Safety Data Sheets are more informative for large acute exposures than for the low-level chronic exposures that are so common with skin conditions.

2. Physical, chemical, and biologic agents (chemical and trade names) to which the patient is or may be exposed.

3. Presence of skin diseases in fellow workers.

4. Control measures to minimize or prevent exposure in the workplace, including personal and occupational hygiene (e.g., handwashing instructions and facilities, showers, laundry service) and the availability of gloves, aprons, shields, and enclosures.

5. Compensation the patient received for skin disease in a previous job.

6. Other exposures, including soaps, detergents, household cleaning agents, materials used in hobbies (e.g., resins, paints, solvents), and topical medications, especially those containing sensitizing agents such as neomycin (e.g., Neosporin). The depth of questioning should reflect the morphologic presentation of the skin disorder. The physician should look for eczema, hives, asthma, hay fever, clothing or food allergy, psoriasis, acne, oily skin, miliaria (i.e., "prickly heat," with many tiny vesicles near openings of sweat and sebaceous glands), contact allergies (e.g., reactions to metal objects, cosmetics, home cleansers), fungal infections (e.g., athlete's foot, ringworm), family history of atopy or psoriasis, and systematic diseases that may have skin manifestations (e.g., diabetes mellitus, peripheral vascular disease).

The history of the illness and the occupational history may reveal a close association between the skin condition and a specific work exposure known to produce such skin effects. The appearance of the condition may also suggest the cause. For example, a glovepattern distribution of vesicular lesions on the hands strongly indicates a contact dermatitis.

It is not unusual to discover an underlying skin disease that is exacerbated by work exposures. However, multiple occupational and nonoccupational exposures may be identified, no clear time relationship between the skin lesions and the work history may be found, or the skin lesions may be difficult to classify.

General Principles of Treatment

For therapeutic purposes, contact dermatitis can be classified as acute (weepy, edematous, vesicular, blistered) or chronic (dry, cracked, scaly, thickened). Therapeutic mea-

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sures can almost always provide some relief, but cure depends on identification of the offending agent and cessation of exposure.

ACUTE DERMATITIS

Wet Dressings. Absorbent material (e.g., cotton dressings) moistened with cool water or Burow's solution (aluminum acetate diluted 1:40 in water) should be applied to the affected area four to six times a day. The effects of this treatment include bacteriostasis, gentle debridement, debris removal, and evaporative cooling (which lessens pruritus).

Steroids. Topical steroids have no effect on acute vesicular reactions, but they may be applied once vesicles have resolved. Systematic steroid therapy is indicated when lesions are widespread, vesicular, and edematous or bullous. Short courses of prednisone, in a dosage of 40 to 60 mg per day for five to seven days, are usually satisfactory and do not require tapering. Secondary infections should be treated. Systemic steroids should be used cautiously in patients with diabetes mellitus, psychotic disorders, uncontrolled hypertension, or infections such as tuberculosis or herpes.

Systemic Antihistamines. Diphenhydramine (e.g., Benadryl), in a dosage of 25 to 50 mg three or four times daily, or hydroxyzine hydrochloride (Atarax), in a dosage of 25 mg three or four times daily, provides an antipruritic effect. Because of the sedating effects of these drugs, patients should be advised against operating machinery or vehicles. Doxepin (Sinequan), in a dosage of one to three 10-mg capsules taken at night as needed, is effective, but patients should be monitored for anticholinergic effects. A 5 percent doxepin cream (Zonalon) is also effective. Topical antihistamines should be avoided because of their potential sensitizing action.

CHRONIC DERMATITIS

Emollients. Topical agents such as petrolatum (Vaseline) provide an occlusive film over inflamed skin, decrease fissuring, and reduce evaporation. Emollients are most effective

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when they are applied after skin has been soaked or washed in water.

Topical Steroids. These agents reduce inflammation and associated pruritus. Topical steroids should be applied no more than twice a day; more frequent use provides no advantage and may induce tachyphylaxis. Treatment with higher potency fluorinated steroids, such as 0.05 percent fluocinonide (Lidex), in contrast to mid-potency 0.1 percent triamcinolone acetonide (Kenalog), is indicated in patients with more persistent dermatitis or chronic dermatitis affecting the hands. Occlusion with plastic wrap or plastic gloves enhances absorption. Fluorinated steroids should not be used around the eyes, on the face, or in the groin area because of their long-term effects, which include the development of striae.

General Principles of Prevention and Control

Most occupational skin diseases can be prevented. The following issues should be considered:

1. Predisposing factors that contribute to work-related skin disease on a particular job. For example, fair skin would be a predisposing factor in a construction worker who is chronically exposed to sunlight.

2. Avoidance of certain work environments by workers with preexisting skin disease. For example, a hairdresser with chronic eczematous eruption of the hands might be advised to change professions.

3. Preventive measures on the job. For example, the employer of a worker with occupational acne might be advised to provide the worker with gloves and aprons that are impervious to oils.

PREPLACEMENT SCREENING

A history or skin survey can be used to determine if a worker has a preexisting condition that increases the likelihood of occupational skin disease in the job under consideration. Common occupational exposures, risk groups, and skin diseases are listed in *Table 1.*^{2,3} In acute contact dermatitis, the skin is usually blistered and weepy. Chronic dermatitis typically leads to dry, scaly, thickened skin.

CONTROL MEASURES

As long as proper protective measures are in place, workers with irritant or even allergic contact dermatitis often can remain on the job. However, ordinary protective measures may be inadequate for some workers with allergic contact dermatitis. It may be necessary to recommend that these persons be given a different job or moved to another workstation.

Many dermatoses can be prevented by improved worker and workplace cleanliness. Workers should be counseled about personal hygiene, and management personnel should be advised about proper handwashing agents. Contact with organic solvents (e.g., mineral oils, paint thinner) should be scrupulously avoided. The following should be recommended: provision of effective, nonirritating, nonallergenic skin cleansers; use of emollients, hand lotions, and creams after handwashing; frequent clothing changes; daily showering; rapid removal of oil- and chemical-soaked clothing; use of company laundering facilities or separate washing of workers' clothing at home; and no eating, drinking, or smoking in the work area. Protective measures for selected exposures are listed in Table 2.2,3

Specific Occupational Skin Diseases

Contact dermatitis, which includes irritant contact dermatitis and allergic contact dermatitis, accounts for 90 percent of skin disorders acquired in the workplace.⁴

IRRITANT CONTACT DERMATITIS

Pathophysiology. Irritant contact dermatitis involves a nonimmunologic response to a skin irritant. Injury develops slowly over days to months through disturbance of cell hydration as a result of the defatting action of prolonged

TABLE 1 Common Occupational Exposures and Associated Skin Diseases

Exposure	Workers at risk	Skin diseases	
Chemicals	All workers	Irritant contact dermatitis, allergic contact dermatitis	
Abrasions, friction "burns," pressure injuries, lacerations	Construction, lumber, steel, and masonry workers	Keloids, postinflammatory pigmentary changes; can cause spread of lesions in workers with lichen planus and psoriasis (Koebner's phenomenon)	
Sunlight	Outdoor workers, including telephone-line workers, sailors, postal workers, landscapers, and construction workers (e.g., roofers)	Actinic keratosis, carcinoma (basal cell, squamous cell), melanoma, sunburn, photoallergic dermatitis, melanosis; worsens preexisting discoid and systemic lupus erythematosus, granuloma annulare, porphyria, rosacea, etc.	
Heat	Foundry workers (e.g., metal casting), outdoor workers	Miliaria ("prickly heat"), folliculitis, tinea pedis	
Cold	Sailors, fishermen, other outdoor workers	Raynaud's disease, urticaria, xerosis, frostbite	
Moisture	Food handlers, chefs, bartenders, dishwashers, hairdressers	Irritant contact dermatitis, paronychia	
Rhus genus (e.g., poison oak, poison ivy)	Outdoor workers, including surveyors, firefighters, park and highway maintenance workers, utility-line workers, and farm workers	Allergic contact dermatitis, contact urticaria	
Electricity	Electricians, telephone workers, construction workers	Burns, skin necrosis	
Fiberglass spicules	Insulation workers, workers in the manufacture of fishing poles and boat hulls	Irritant contact dermatitis, erythema; less commonly, erosion, urticaria	
lonizing radiation*	Medical personnel, welders (i.e., radiographs of welds), workers in the nuclear energy industry	Skin cancer, acute or chronic radiation dermatitis, alopecia, nail damage (destroys matrix)	

*—Uncommon risk in the United States because most persons who work with ionizing radiation are closely supervised; exposure is more likely in developing countries.

Information from references 2 and 3.

exposure to weaker irritants such as water, solvents, or soaps. Xerosis dominates. Under excessively moist working conditions, however, these skin irritants can cause excessive cell hydration and result in maceration, most often in the feet and groin.

Severe skin irritants are less common but more serious hazards. On contact, strong acids, alkalis, and heavy metals cause chemical burns.² The skin reacts immediately, and painful lesions appear. The lesions may progress to erosion and necrosis.

Clinical Features. In irritant contact dermatitis, the rash appears in exposed or contact areas, in thin skin more often than thick skin (e.g., dorsum of the hands rather than the palms), and in the area around the belt or collar. The rash may be difficult to differentiate from the rash of allergic contact dermatitis.

Acute lesions are painful, weepy, and vesicular, whereas chronic lesions are dry, erythematous, cracked, and lichenified. The lesions assume a clearly demarcated pattern and are often asymmetric and unilateral. Hardening or adaptation of the skin may occur as a result of repeated contact with moderate irritants (e.g., rubbing alcohol, nail polish remover).

Diagnosis. The diagnosis is based on the presence of rash in exposed areas and clinical improvement of the rash on removal of the offending agent (*Table 3*).^{2,3}

Treatment. The previously described general principles of treatment should be followed. For exposure to severe irritants, the extent of injury can be reduced by immediate, continuous, and prolonged water irrigation (up to three hours), with contaminated clothing removed while the affected area is under water. Alkali burns should not be treated with acid (vinegar), and acid burns should not be treated with alkali (baking soda), because these measures will result in additional damage through an exothermic reaction.

With large or full-thickness burns, hospitalization may be necessary, especially if the patient has circumferential burns of the neck or extremity, a medical condition such as diabetes mellitus, or poor hygiene.⁵

ALLERGIC CONTACT DERMATITIS

Pathophysiology. Allergic contact dermatitis is an immunologic cell-mediated response to even trivial exposure to an antigenic substance. In photoallergic contact dermatitis, a subcategory of allergic contact dermatitis, ultraviolet light initiates an allergic response.

The most common sensitizing agents are listed in *Table 4.^{2,3}* The percentage of persons who react to these agents varies widely. For example, only 6 percent of persons react to nickel³ (found in almost all alloys), whereas as many as 70 percent react to members of the Rhus genus (e.g., poison oak, poison ivy).⁶

Skin burns caused by severe irritants such as strong acids or alkali should be treated with immediate, continuous, and prolonged water irrigation, with contaminated clothing removed while the affected area is under water.

From 10 to 17 percent of health workers react to latex.⁷ Sensitization to one chemical may induce sensitivity to related chemicals.

Clinical Features. Rash appears in areas exposed to the sensitizing agent, usually with an asymmetric or unilateral distribution. Sensitizing agent on the hands or clothes is often transferred to other body parts. The rash is characterized by erythema, vesicles, and severe edema. Pruritus is the overriding symptom.

Latex allergic reactions range from pruritus to erythematous, weeping lesions.⁸ A concurrent irritant contact dermatitis from nonlatex chemicals used in glove production may also develop. Exposure to latex, as well as some other substances, can proceed to anaphylaxis.

In photoallergic dermatitis, sunlight and exposure to the offending substance usually

TABLE 2

Selected Occupational Exposures and Protective Measures

Exposure	Protective measures
Dust, fiberglass spicules, irritating solids (e.g., cement)	Clothing made of tightly woven material, preapplication of mild dusting powder, leather gloves with smooth finish, steel-tipped shoes
Liquids, vapors, fumes	Face shields, plastic or synthetic rubber* gloves and aprons, adequate ventilation
Moderate alkalis, solvents	Synthetic rubber,* neoprene plastic, or hypoallergenic gloves with replaceable soft cotton liners
Trauma	Leather gloves with smooth finish, steel-tipped shoes
Sunlight, ultraviolet light	Sunscreen, protective clothing (hat, long-sleeved shirt or jacket)

*—Natural rubber deteriorates.

Information from references 2 and 3.

affect the face and arms, but the sun-shaded area under the chin is generally spared. Although sunlight is essential for the initiation of photosensitization, the dermatitis may continue long after the photoallergy is eliminated (a condition known as a "persistent light eruption").⁹ Common photoallergic substances include fruits and vegetables (e.g., limes, celery, parsnips), hydrocarbons (e.g., coal tar, pitch, asphalt, anthracene), and drugs (e.g., tetracycline, thiazide diuretics, phenothiazines, sulfonamides) and fluorescein dye.³

Diagnosis. A tentative diagnosis may be made on the basis of the history and clinical findings. For example, allergic contact dermatitis caused by exposure to a member of the Rhus genus may be suspected in a patient who presents with vesicles or bullae arrayed in a linear fashion on the forearm two to three days after performing outdoor tasks in an area that contains poison oak or poison ivy. Direct patch skin testing is recommended for more definitive diagnosis and identification of the sensitizing agent. Skin testing can also be used to screen for an allergic cause of a dermatitis of long duration or unknown etiology, or to identify a suspected allergic component to irritant contact dermatitis. Photopatch testing with ultraviolet light should be used to diagnose photoallergic dermatitis.¹⁰

The radioallergosorbent test (RAST) is a blood-testing technique rather than a direct skin test. RAST measures specific immunoglobulin antibodies to sensitizing substances (e.g., latex IgE for latex allergy). Controversy exists regarding the sensitivity and specificity of RAST compared with direct patch or scratch skin testing. The "modified" RAST is more sensitive but less specific than the standard RAST.¹¹

Treatment and Prevention. Allergic contact dermatitis improves with removal of the sen-

Causal agents	Workers at risk	Clinical features and diagnosis	Prevention
Severe irritants (immediate reaction): strong acids and alkalis, heavy metals	Factory and semiconductor workers	 Symptoms: immediate pain and burning Appearance (easy to recognize): red blisters, ulcers, erosion, necrosis Location: exposed or contact areas, more often in thin skin (e.g., dorsum of hand) than thick skin (e.g., palm); spread is rare unless worker has autosensitivity or allergic sensitivity; may occur in groin and areas not cleaned well (e.g., under watch or ring, between fingers) Diagnosis: clinical improvement on removal of causal agent; patch skin testing not recommended 	Workplace protection: reengineering and shielding; change to less irritating substance; washing facilities (convenient location, nonirritating soaps for worker use) Personal protection: barrier creams, special clothing (e.g., masks, gloves, aprons), good hygiene
Weak irritants (cumulative reaction over time): soaps, detergents, solvents, synthetic oils, temperature (thermal burns), sunlight (sunburn)	Nurses, dental hygienists, dentists, waiters, butchers, bartenders, dishwashers, food handlers	Symptoms: delayed pain, some pruritus, cumulative Appearance: vesicles, chronic lichenified scaling with fissures, eczema, and cracking; less red and harder to recognize than dermatitis from severe irritants Location and diagnosis: same as for strong irritants	Same as for strong irritants

TABLE 3 Irritant Contact Dermatitis*

*—Responsible for 80% to 90% of contact dermatitis cases in the workplace.²

Information from references 2 and 3.

TABLE 4 Allergic Contact Dermatitis*

Causal agents	Groups at risk	Clinical features and diagnosis	Prevention
Rhus genus (e.g., poison oak, poison ivy)	Firefighters and outdoor workers, including surveyors, maintenance workers, utility workers, farm workers, and landscapers	With first exposure, reaction in 5 to 28 days; with reexposure, reaction in 6 to 72 hours Appearance: vesicles and bullae in unilateral linear pattern; in severe cases, oozing blisters and pronounced edema Location: contact area, with spread to genitalia, face, and trunk (spares mucous membranes) Diagnosis: clinical appearance; limited usefulness for patch skin testing; 70% of persons are sensitive.	Workplace protection: respirators Personal protection: protective gloves and clothing, removal of exposed clothing and shoes, showering, effective laundering, instruction in recognizing members of Rhus genus; desensitization (limited usefulness)
Epoxy resins	Workers in high-technology industries (e.g., computers, chemicals, electronics), wire and cable workers, floor layers, pipe workers	Appearance: vesicles, erythema, occasional bullae; unilateral, asymmetric Location: dorsum of hands, fingers, feet; lesions at distant sites (transfer by hands) Diagnosis: clinical improvement with removal of causal agent; patch skin testing	Workplace protection: isolation of epoxy mixing, use of less sensitizing resin, no smoking or eating in work area Personal protection: gloves and aprons during mixing, washing on contact, protection from sun (photosensitivity induced)
Nickel	Dental technicians, mechanics, cashiers, grinders, casters, jewelers, battery makers, electroplaters, miners, refiners, ceramic workers, computer workers, textile workers, electronic workers	Appearance: small eruption, often in shape of object contacted (e.g., button); spreads bilaterally and symmetrically Location: sun-exposed areas (phototoxic), frequently palmar; often widespread and chronic Diagnosis: clinical improvement with removal of causal agent; patch skin testing	Workplace protection: substitution of aluminum, covering tools with plastic Personal protection: gloves, measures that decrease sweating (to diminish solubilization)
Chromates	Arc welders, cutting oil users, painters, dyers, electroplaters, leather tanners, battery workers, cement workers, lithographers	Appearance: nummular eczema; often xerotic and lichenified Location: can become widespread and, over years, persistent Diagnosis: appearance; patch skin testing less helpful	Workplace protection: in electroplating, change hexavalent for less sensitizing trivalent chromium; use premixed cement; periodic examination of workers' skin and mucous membranes; respirators Personal protection: gloves, work shoes; wash on contact; protection from sun (photosensitivity induced)
Acrylics	Dentists, dental technicians (denture making), printers, orthopedic surgeons (methyl methacrylate)	Appearance: eczematous eruptions, peripheral neuropathy Location: exposed areas Diagnosis: patch skin testing using low dilution	Workplace protection: heat curing rather than cold curing (cold curing is more often associated with skin disorders) Personal protection: wash on contact
Topical medications: neomycin (e.g., Neosporin), benzocaine (e.g., Anbesol, Hurricaine, Orajel), ethylenediamine (stabilizer)	Medical personnel, pharmacists, all workers	Appearance: pruritic eruption of erythematous vesicles Location: area of application Diagnosis: patch skin testing	Personal protection: use nonsensitizing topical medications (e.g., 5 percent doxepin cream [Zonalon])

*—Responsible for 10 to 20 percent of contact dermatitis cases in the workplace.²

Information from references 2 and 3.

sitizing agent. The previously described general principles of treatment should be followed. Desensitization to agents such as Rhus antigens provides protection that is incomplete and lasts for no more than a few months; furthermore, desensitization must be repeated each year to maintain partial resistance.⁶

OIL ACNE AND FOLLICULITIS

Pathophysiology. Over one million workers in the United States regularly use solvents and lubricants (oils and greases) on the job. Exposure to these substances and resultant mechanical blockage of pilosebaceous units can lead to "oil acne."^{12,13} Irritants or sensitizing agents in oils and greases can also cause contact dermatitis.

Clinical Features. Comedones, pustules, and papules may be present. A key feature is the occurrence of these lesions in areas with exposure to oil-soaked clothing (e.g., hands, arms, thighs, groin). Occupational acne may also present as aggravation of existing acne, usually in the face or neck area. Secondary infection from bacterial folliculitis is common.

Treatment and Prevention. Patients with occupational acne should be advised to avoid contact with oils and greases. Frequent routine cleansing of the skin and daily washing of work clothes are necessary. If preventive measures are ineffective, routine acne therapy is indicated.

OCCUPATIONAL SKIN NEOPLASMS

Skin tumors can result from exposure to substances such as polycyclic hydrocarbons, inorganic metals, and arsenicals.^{3,14} These lesions can also develop because of trauma, burns, and exposure to ultraviolet light or ionizing radiation (particularly in developing countries).^{3,14} Cocarcinogenesis, such as the interaction of sunlight and tar, is often implicated. Frequently, the skin tumors do not appear until two or three decades after the exposure.¹⁵

Establishment of an occupational etiology

for skin tumors is complicated because of the mobile work force in the United States, numerous job changes by individual workers, and exposures that occur both at work and away from the job. All patients should be provided with information on how to prevent skin cancer.¹⁶

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