CHAPTER 28

# Skin Disorders

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The skin plays an important role in providing a protective, living barrier between the external environment of the world around us and the internal environment of the human body. As a first-line protective barrier, the cutaneous surface is subjected to the hostile forces of the external environment and, as such, can be directly injured or damaged by these environmental forces.

In general, the causes of environmental skin disorders can be grouped into the following categories:

1. *Physical insults*: friction, pressure, trauma, vibration, heat, cold, variations in humidity, ultraviolet/visible/infrared radiation, ionizing radiation, and electric current.
2. *Biologic causes*: plants, bacteria, rickettsia, viruses, fungi, protozoa, parasites, and arthropods.
3. *Chemical insults*: water, inorganic acids, alkalis, salts of heavy metals, aliphatic acids, aldehydes, alcohols, esters, hydrocarbons, solvents, metallo-organic compounds, lipids, aromatic and polycyclic compounds, resin monomers, and proteins.

These insults are present everywhere in the environment, and the settings where they may threaten the skin include the home setting, during outdoor leisure activities, while involved in hobbies, and the work environment, which is likely to be the most important setting where physical, biological, and chemical insults can affect the skin.

*Occupational dermatology* is the facet of dermatology that deals with skin diseases whose etiology or aggravation is related to some exposure in the workplace. By its nature, occupational dermatology is also related to occupational and preventive medicine. The ideal role of a medical practitioner involved in occupational dermatology is not only to diagnose and treat patients but also to determine the etiology of the occupational skin disease and to make recommendations for its prevention. Making the diagnosis and offering treatment, determining etiology, and recommending preventive measures can all be difficult undertakings.

Environmental and occupational skin diseases can manifest themselves in a variety of ways. This chapter will emphasize skin conditions caused by environmental agents that have a direct effect on the skin. These include irritant contact dermatitis, allergic contact dermatitis, contact urticaria, skin infections, skin cancers, and a large group of miscellaneous skin diseases. Certain common skin diseases, such as atopic dermatitis and psoriasis, are exacerbated by environmental factors, but their etiology remains unclear and they will not be covered here.

## CONTACT DERMATITIS

Contact dermatitis is the most common occupational and environmental skin disease. Epidemiologic data show that contact dermatitis comprises 90 to 95 percent of all occupational skin diseases.[1] *Contact dermatitis*—both irritant and allergic—is an inflammatory skin condition caused by skin

*This chapter has been updated from a published U.S. government work.*

***FIGURE 28-1*** • Acute contact dermatitis from exposure to the strong irritant ethylene oxide.

contact with an exogenous agent or agents, with or without a concurrent exposure to a contributory physical agent, such as ultraviolet light. Contact dermatitis can result from a nonimmunologic reaction to chemical irritants (irritant contact dermatitis) or from an immunologic reaction to allergens (allergic contact dermatitis). *Irritant contact dermatitis* is a cutaneous inflammation resulting from a direct cytotoxic effect of a chemical or physical agent, whereas *allergic contact dermatitis* is a type IV, delayed or cell-mediated, immune reaction. There are more than 57,000 chemicals reported to cause skin irritation, but only 3,000 chemicals are potential human allergens. These allergens are mostly confined to large-molecular-weight proteins and to small-molecular-weight chemicals that act as haptens, and usually only a small proportion of people are susceptible to them.

In contact dermatitis, the skin initially turns red and can develop small, oozing vesicles and papules. After several days, crusts and scales form. Stinging, burning, and itching may accompany the skin lesions. With no further contact with the etiologic agent, the dermatitis usually disappears in 1 to 3 weeks. With chronic exposure, deep fissures, scaling, and hyperpigmentation can occur. Exposed areas of the skin, such as hands and forearms, which have the greatest contact with irritants or allergens, are most commonly affected. If the agent gets on clothing, it can induce dermatitis at areas of greatest contact, such as thighs, upper back, armpits, and feet. Dusts can produce dermatitis at areas where the dust accumulates and is held in contact with the skin, such as under the collar and belt line, at the tops of socks or shoes, and in flexural areas, such as the antecubital and popliteal fossae. Mists can produce a dermatitis on the face and anterior neck. Irritants and allergens can be transferred to other areas of the body, such as the trunk or genitalia, by unwashed hands or from areas of accumulation, such as under rings or interdigital areas. It is often impossible to clinically distinguish irritant contact from allergic contact dermatitis, as both can have a similar appearance and both can be clinically evident as an acute, subacute, or chronic condition (Figs. 28-1 through 28-3).

## Public Health Importance

Measures of the public health importance of a disease include the absolute number of cases, the incidence rate, the prevalence (rate), the economic impact of the disease, and the prognosis and preventability of the disease.[2]

Specific national data sources on contact dermatitis are limited. In the United States, data from the National Ambulatory Medical Care Survey, a national probability sample survey of nonfederal office-based physicians, showed that in 2002 skin rash was the principal reason for 11.8 million patient visits—1.3 percent of all visits for that year.[3] Based on previous surveys, it is estimated that approximately one-half of these visits would have had a diagnosis of contact dermatitis or other eczemas.

In 1988, the National Health Interview Survey (NHIS) included an Occupational Health Supplement, which included questions on dermatitis. The

***FIGURE 28-2*** • Subacute dermatitis from the rubber accelerator, mercaptobenzothiazole, from the rubber in a work boot.

survey consisted of personal interviews of people in randomly selected households. For 30,074 people participating in the NHIS, the period prevalence for all dermatitis was 11.2 percent and for contact dermatitis was 2.8 percent. Projecting these results to the U.S. working population resulted in an estimate of 13.7 million people with dermatitis and 3.1 million people with contact dermatitis.[4]

More information is available on the public health impact of occupational contact dermatitis. Specific national occupational disease and illness data are available from the U.S. Bureau of Labor Statistics (BLS), which conducts annual surveys of approximately 160,000 employers selected to represent all private industries in the United States.[5] All occupational skin diseases or disorders, including contact dermatitis, are tabulated in this survey. BLS data show that occupational skin diseases accounted for a consistent 30 to 45 percent of all cases of occupational illnesses from the 1970s through the mid-1980s and in recent years accounted for 12 percent of all occupational illness.[5] A decline in this proportion may be partially related to an increase seen in disorders associated with repeated trauma.

BLS data for occupational skin diseases for 1973 to 2001 are shown in Fig. 28-4. In 2001, BLS estimated 38,900 cases of occupational skin diseases or disorders in the U.S. workforce.[5] However, because of BLS survey limitations, it has been estimated that the number of actual occupational

***FIGURE 28-3*** • Chronic dermatitis from exposure to kerosene, a solvent that was used for cleaning the skin.

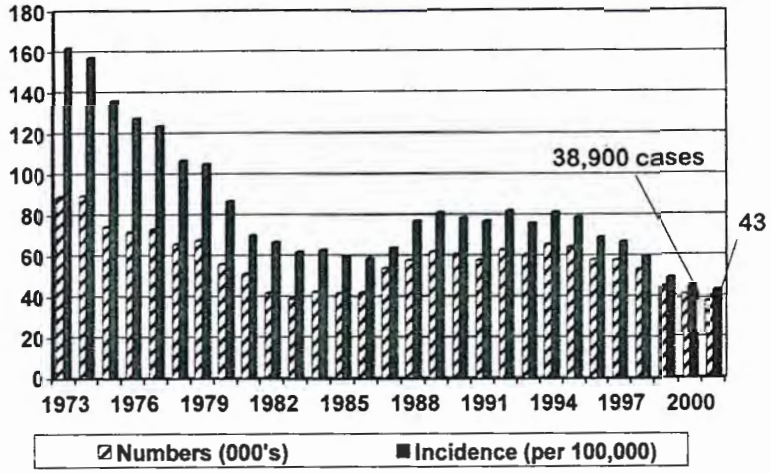


***FIGURE 28-4*** • Annual incidence (number and rate) of occupational skin diseases per year, United States, 1973–2001. (Source: U.S. Bureau of Labor Statistics Annual Survey, 1973–2001.)

skin diseases may be of the order 10 to 50 times higher than that reported by the BLS[6]. This increase would potentially raise the number of occupational skin disease cases to between 400,000 and 2 million per year. In 2001, BLS data showed an annual incidence rate of 43 cases per 100,000 workers.[5]

In 1988, the Occupational Health Supplement of the NHIS indicated that the period prevalence for occupational contact dermatitis occurring in the preceding year was 1.7 percent. Projecting these results to the U.S. working population resulted in an estimate of 1.87 million people with occupational contact dermatitis and a 1-year period prevalence of 1,700 per 100,000 workers for the year.[4] The numbers and rates in the BLS and NHIS surveys are not directly comparable because they rely on different information sources with different ascertainment methods and different case definitions.

The economic impact of a disease can be measured by the direct costs of medical care and workers' compensation or disability payments and the indirect costs associated with lost workdays and loss of productivity. In 1984, the estimated annual direct and indirect costs of occupational skin diseases exceeded $22 million.[6] However, considering that the actual annual incidence may be 10 to 50 times greater than reported in the BLS data, the total annual cost of occupational skin diseases in 1984 may have ranged from $222 million to $1 billion.[6] (These estimates do not include costs of occupational retraining.)

A review of 1993 BLS data showed that of 60,200 cases of occupational skin diseases, 12,613 (21 percent) resulted in days away from work.[7] The mean time away from work was 3 days, but 17 percent of lost workday cases had more than 11 days away from work. Of those with days away from work, 70 percent had a diagnosis of dermatitis. In 2001, of the 38,900 skin disease cases, 6,051 (16 percent) resulted in days away from work, with a median of 3 days lost.[5] Of these, 78 percent had dermatitis. A study of 235 Canadian workers with occupational skin diseases showed that 35 percent had been away from work for greater than 1 month, 14 percent between 1 week and 1 month, 17 percent less than 1 week, and 33 percent did not lose workdays because of the skin condition.[8]

Studies on the prognosis of occupational contact dermatitis point out that primary prevention is very important. For example, of 555 patients completing a follow-up questionnaire 2 to 3 years after diagnosis, only 26 percent of the women with contact dermatitis had complete healing (22 percent had continual symptoms and 52 percent had recurring symptoms), and only 31 percent of the men had complete healing (29 percent had continual symptoms, 40 percent had recurring symptoms).[9] A telephone survey of 235 occupational skin disease patients, conducted a mean of 4 years after

diagnosis, showed that 40 percent had continuing dermatitis, although of this group, 76 percent reported an improvement in their skin condition.[8] Outcomes may or may not be influenced by leaving the dermatitis-provoking job. In addition, many skin disorders, including contact dermatitis, have been shown to have a significant impact on quality of life.[10]

Over the years, there have been changes in the epidemiology of occupational skin diseases. A decrease in the absolute number of cases and the incidence rate in the BLS survey from the 1970s to the early 21st century may be attributable to several factors, including changes in industry and industrial practices, increased awareness and preventive measures, and possible underreporting, underrecognition, and misclassification. Still, occupational contact dermatitis remains a relatively common disease with a noteworthy public health impact. These factors, along with the potential chronicity of the disorder, its effect on an individual's vocational and avocational activities, and its preventability, make occupational contact dermatitis a disease of public health importance.

## Population at Risk and Etiologic Agents

There is a myriad of occupations that have unique exposures resulting in occupational contact dermatitis. Total numbers and incidence rates of occupational dermatologic conditions, by major industry division, based on the BLS survey for 2001 are shown in Table 28-1.[5] The greatest number of cases of occupational skin diseases is seen in manufacturing, but the highest incidence rate is seen in agriculture/forestry/fishing.

In the NHIS, the occupational groups with the highest prevalence of self-reported occupational contact dermatitis included physicians, dentists, nurses, pharmacists, and dieticians (5.6 percent); public transport attendants, cosmetologists, and other personal service occupations (4.9 percent); health care therapists, technologists, technicians, and assistants (3.5 percent); and mechanics and repairers of vehicles, engines, heavy equipment, and machinery (3.5 percent).[4] Of all accepted workers' compensation claims for occupational contact dermatitis in Oregon, the most common occupations were laborers (14.2 percent), food service workers (13.8 percent), machine operators

**TABLE 28-1**

**Number and Incidence Rate of Occupational Skin Diseases, by Industry Sector, 2001**

| | Number | Incidence Rate (per 100,000) |
|---|---|---|
| Agriculture/forestery/ fisheries | 2,600 | 175 |
| Manufacturing | 16,100 | 93 |
| Services | 13,400 | 48 |
| Transport/utilities | 1,600 | 24 |
| Construction | 1,400 | 23 |
| Wholesale and retail trade | 3,200 | 14 |
| Finance/insurance/real estate | 600 | 9 |
| Mining | <50 | 2 |
| Total/Overall | 38,900 | 43 |

(13.1 percent), agricultural workers (9.0 percent), health professionals (8.2 percent), and janitors/maids (6.4 percent), followed by production crafts workers, mechanics, construction workers, and hairdressers/cosmetologists.[11]

The most frequent causes of irritant contact dermatitis include soaps and detergents, fibrous glass and other particulate dusts, food products, cleaning agents, solvents, plastics and resins, petroleum products and lubricants, metals, and machine oils and coolants.[1] Causes of allergic contact dermatitis include plants (poison ivy, poison oak, and poison sumac), metallic salts, organic dyes, plastic resins, rubber additives, and germicides.[12] The most common skin patch test allergens found to be positive in patients along with potential sources of exposure are shown in Table 28-2.[13] In patients with occupational contact dermatitis who were skin patch tested, the common allergens included carba mix, thiuram mix, formaldehyde, epoxy resin, and nickel.[14]

## Diagnosis

The environmental cause or work-relatedness of contact dermatitis may be difficult to prove. The accuracy of the diagnosis is related to the skill level, experience, and knowledge of the medical professional who makes the diagnosis and confirms

**TABLE 28-2**

**North American Contact Dermatitis Group Patch-Test Results,[a] 1998 to 2000**

| Test Substance | Common Sources | Percent Positive |
|---|---|---|
| Nickel sulfate 2.5% | Metals, jewelry | 16.2 |
| Balsam of Peru 25% | Perfumes, creams | 12.3 |
| Neomycin | Creams, lotions | 11.5 |
| Fragrance mix 8% | Toiletries, scented products | 10.9 |
| Thimerosal 0.1% | Cosmetics, cleansers | 10.8 |
| Sodium gold thiosulfate 0.5% | Jewelry, dental products | 10.5 |
| Formaldehyde 1% aqueous | Fabrics, skincare products | 9.2 |
| Quaternium-15 2% | Cosmetics, sunscreens | 9.2 |
| Bacitracin 20% | Ointments, creams | 9.2 |
| Cobalt chloride 1% | Metals, jewelry | 7.6 |
| Methyldibromo glutaronitrile phenoxyethanol 2.5% | Biocides, skincare products | 6.0 |
| Potassium dichromate 0.25% | Cement, leather | 5.8 |
| Ethyleneurea melamine formaldehyde resin 5% | Textiles | 5.0 |
| *p*-Phenylenediamine 1% | Hair dyes, leather | 4.9 |
| Carba mix 30% | Rubber, pesticides | 4.8 |
| Thiuram 1% | Rubber, pesticides | 4.7 |
| Propylene glycol 30% aqueous | Cosmetics, topical meds | 3.7 |
| Cinnamic aldehyde 1% | Fragrances, flavorings | 3.6 |
| Methyldibromo glutaronitrile phenoxyethanol 0.4% | Biocides, skincare products | 3.5 |
| Amidoamine 0.1% aqueous | Shampoos, liquid soap | 3.4 |

[a] Prevalence of 20 most common positive reactions (n varies from 5,770 to 5,835).

From Marks JG, Belsito DV, DeLeo VA, et al. North American Contact Dermatitis Group patch-test results, 1998 to 2000. Am J Contact Dermatitis 2003;14:59–62.

the relationship with environmental or workplace exposures. Guidelines are available for assessing the work-relatedness of dermatitis, but even with guidelines the diagnosis may be difficult.[15] The diagnosis is based on the medical and occupational histories and physical findings. The importance of the patient's history of exposures and disease onset is clear. Standardized questionnaires for surveying work-related skin diseases are available and can be helpful in the workplace.[16] In irritant contact dermatitis, there are no additional confirmatory tests. Patch tests or provocation tests are discouraged because of a high false-positive rate. In many instances, allergic contact dermatitis can be confirmed by skin patch tests using specific standardized allergens or, in some circumstances, by provocation tests with nonirritating dilutions of industrial contactants. Skin patch tests should only be conducted by health care professionals trained in conducting and interpreting the tests. Skin patch tests should never be conducted with unknown substances.

The following questions can be used as criteria for determining work-relatedness:

1. Is the clinical 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 cutaneous exposure in relation to the job task?
4. Is the temporal relationship between exposure and onset consistent with contact dermatitis?
5. Are nonoccupational exposures excluded as probable causes?

6. Does dermatitis improve away from the exposure to the suspected irritant or allergen?
7. Do patch tests or provocation tests identify a probable causal agent?[15]

### Treatment and Prevention

Avoiding etiologic irritants and allergens is the first step in any treatment regimen. Dermatitis is treated according to its clinical stage. Acute dermatitis treatment options can include a short course of systemic steroids, topical steroids, and soothing compresses or baths. Antihistamine therapy or use of sedatives may be helpful to decrease pruritus. If secondary infection is present, topical or systemic antibiotics are indicated. Subacute dermatitis and chronic dermatitis are usually treated with topical steroid therapy and lubrication of the skin. Potential dangers of long-term use of topical steroids, especially high-potency steroids, include systemic effects and skin atrophy. In addition, contact dermatitis can be caused by ingredients found in topical agents, including antibiotics, fragrances, vehicles, or steroids.

Strategies for the prevention of occupational contact dermatitis include:

- identifying irritants and allergens;
- substituting chemicals that are less irritating or allergenic;
- establishing engineering controls to reduce exposure;
- using personal protective equipment (PPE), such as gloves and special clothing;
- emphasizing personal and occupational hygiene; and,
- establishing educational programs to increase awareness in the workplace.[17]

Chemical changes in industrial materials have proved to be beneficial. For example, the addition of ferrous sulfate to cement to reduce the hexavalent chromium content was effective in reducing occupational allergic contact dermatitis in Europe. The use of PPE must be considered carefully, as it may actually create problems by occluding irritants or allergens or by directly irritating the skin. Similarly, the excessive pursuit of personal hygiene in the workplace may actually lead to misuse of soaps and detergents and resulting irritant contact dermatitis. The effectiveness of gloves depends on the specific exposures and the types of gloves used. The effectiveness of barrier creams is controversial, as there are limited data on the protective nature of these topical products during actual working conditions involving high-risk exposures. Other interventions, including providing advice on PPE and educating the workforce about skin care and exposures, are beneficial.[18]

## CONTACT URTICARIA

*Urticaria* is defined as the transient appearance of elevated, erythematous pruritic wheals or serpiginous exanthem, usually surrounded by an area of erythema. In addition, areas of macular erythema or erythematous papules may also be present. These skin lesions appear and peak in minutes to hours after the etiologic exposure, and individual lesions usually disappear within 24 hours. Urticarial lesions usually involve the trunk and extremities, although they can involve any epidermal or mucosal surface. Large wheal formation, where the edema extends from the dermis into the subcutaneous tissue, is referred to as *angioedema*. This condition is more commonly seen in the more distensible tissues, such as the eyelids, lips, ear lobes, external genitalia, and mucous membranes.

Urticarial lesions can be classified in one or more of the following categories based on characteristic features:

1. Duration or chronicity: acute or chronic.
2. Clinical distribution of the lesions or the extradermal manifestation: localized, generalized, or systemic associated with rhinitis, conjunctivitis, asthma, or anaphylaxis.
3. Etiology: idiopathic or cause-specific.
4. Routes of exposure: direct contact, inhalation, or ingestion.
5. Mechanisms: nonimmunologic, immunologic, or idiopathic.

Acute urticaria ranges from a single episode to recurrences over a period of less than 6 weeks. Common causes of acute urticaria include insect bites or stings and food or drug allergies. Chronic urticaria occurs daily, or almost daily, over a period longer than 6 weeks. Food, drugs, and infections can also be causes of chronic urticaria. However, in the chronic form, the exact causative agents may never be identified. In most cases of urticaria, the cause is unknown.

Occupational urticaria is presumed or proved to be caused by exposure to one or more substances or physical agents in the workplace. Occupational

urticaria may be acute or chronic, localized or generalized, or associated with systemic manifestations, such as asthma. In occupational settings, direct contact with substances, and possibly inhalation, may be the most common routes of exposure inducing urticaria. The pathologic mechanisms may be nonimmunologic, immunologic, or not known. Contact urticaria is defined as urticaria that occurs after direct skin contact with a substance. Urticarias that result from nonchemical exposures are commonly classified as physical urticarias. These include mechanical urticarias, caused by trauma, pressure, friction, and vibration; and urticaria resulting from local exposure to physical agents, such as cold, heat, solar radiation, and water.

## Public Health Importance

Data specific for environmental and occupational urticaria are limited. In 2001, BLS estimated 38,900 cases of occupational skin diseases or disorders in the U.S. workforce.[5] Further information is available on the 6,051 cases that involved days away from work. Of this subgroup, 336 (5.5 percent) had urticaria/hives; their median time away from work was 3 days.

## Population at Risk and Etiologic Agents

In general, risk factors for contact urticaria include a history of atopy; a compromise to the barrier function of intact skin, due to conditions such as eczema, abrasions, ulcers; and, in some cases, occupation. Based on reviews of epidemiologic studies, exposures, and patterns seen in case reports, several occupations may be at higher risk for the development of contact urticaria. These include food handlers, cooks, caterers, and bakers; general health care workers, dental professionals, and pharmaceutical industry workers; animal handlers, such as laboratory workers and veterinarians; and gardeners, florists, woodworkers, and agricultural workers.

For food handlers, cooks, caterers, and bakers, the following foods have been reported to induce contact urticaria: apples, beans, beer, caraway seeds, carrots, eggs, endives, fish, garlic, kiwi fruit, lettuce, meat (beef, chicken, lamb, liver, pork, and turkey), milk, peaches, potatoes, rice, shellfish, spices, and strawberries.[19,20] Bakers can develop contact urticaria and other systemic symptoms after exposure to cereal flours, buckwheat flour, and additive flour enzymes such as alpha-amylase.

In health-care, dental, and pharmaceutical environments, exposure to a variety of medications or chemical disinfectants can put workers at risk. Exposures that can cause contact urticaria include aminothiazole, bacitracin, benzocaine gel, cephalosporins, chloramine, chloramphenicol, chlorhexidine, chlorocresol, ethylene oxide, gentamicin, neomycin, nitrogen mustard, penicillin, pentamidine isethionate, phenothiazines, rifamycin, and streptomycin.[19,20] Furthermore, natural rubber latex has been found to be an important cause of contact urticaria in health care professionals.[21] Natural rubber latex gloves were the most common source of exposure.

Contact urticaria has been found to be caused by animal hair, insects, dander, animal placenta, saliva, seminal fluid, and serum. Slaughterhouse workers can develop contact urticaria upon exposure to animal blood. Contact urticaria can be seen in veterinarians after exposure to cow's hairs and placenta, horse dander, and pig's bristles.

Certain woods and plants can cause contact urticaria. These include the larch, limba, obeche (African maple), and teak woods and plants, such as chrysanthemum, Ficus benjamina (weeping fig), lilies, *Limonium tataricum*, *Phoenix canariensis* (canary palm), *Spathiphyllum walisii* (spathe flower), tulips, and fungi (shiitake mushroooms). High-risk occupations include agricultural workers, carpenters, florists, gardeners, and woodworkers. Caterpillar hair, insect stings, and moths can also cause contact urticaria in outdoor workers. Agricultural workers may also be exposed to fertilizers and pesticides, some of which can cause contact urticaria.

A variety of industrial chemicals can cause contact urticaria, including acrylic monomers (plastics), aliphatic polyamines (epoxy resins), alkyl-phenol novolac resin, ammonia, castor bean (fertilizers), diethyltoluamide (DEET), formaldehyde (used in clothing, leather, fumigation, and resins), lindane (a parasiticide), paraphenylenediamine, phenylmercuric priopionate (an antibacterial fabric softener), plastic additives (such as butylhydroxytoluene and oleylamide), reactive dyes, sodium sulfide (used in photographs, dyes, and tanning), sulfur dioxide, vinyl pyrilidine, and xylene and other solvents.[19,20] Contact urticaria can occur

with exposure to a variety of metal salts, including iridium, nickel, platinum, and rhodium.

### Diagnosis and Treatment

The diagnosis of environmental or occupational urticaria is based on the medical and exposure history, physical findings, and *in vitro* or *in vivo* testing. Proving etiology or work-relatedness may be difficult. Suggested criteria include[22]:

1. Documentation of urticaria by physical examination;
2. Exposure to an agent known or presumed to cause urticaria;
3. A temporally consistent relationship between exposure and onset of urticaria (usually 30 to 60 minutes);
4. Associated medical symptoms and localization of urticaria consistent with the route of exposure;
5. Resolution of the urticaria away from the exposure;
6. Exclusion of nonenvironmental or nonoccupational causes; and
7. Medical testing results indicating allergy to a substance in the environment or workplace. Useful medical tests include the open or closed patch test, prick or scratch test, and tests demonstrating specific IgE to suspect occupational antigens, such as by radioallergosorbent (RAST) assays.

In cases of environmental or occupational urticaria where a specific causal agent can be identified, the initial treatment is avoidance of the offending agent. First-generation antihistamines, such as diphenhydramine or hydroxyzine, which block H1 receptors, can be employed initially, but they can cause sedation; this may present a safety issue for certain occupations, such as heavy-equipment operators. When sedation occurs or presents a safety concern, nonsedating, second-generation antihistamines may be employed. When H1 histamine blockers alone are not sufficient, they may be combined with H2 blockers or doxepin, a tricyclic antidepressant with potent H1 and H2 blocking activity. Doxepin is extremely sedating and should be used cautiously, if at all, when safety concerns arise on the job. Oral corticosteroid therapy may be employed for severe cases of chronic urticaria, especially those associated with angioedema.

### Prevention

Strategies in the prevention of environmental and occupational urticaria overlap with those strategies used in the prevention of contact dermatitis and include:

- identifying allergens;
- substituting chemicals that are nonallergenic;
- establishing engineering controls to reduce exposure;
- using PPE, such as gloves and special clothing;
- emphasizing personal and occupational hygiene; and
- establishing educational programs to increase awareness in the workplace.

Recommendations for preventing allergic reactions to natural rubber latex in the workplace have been published by NIOSH.[23]

## DERMATOLOGIC INFECTIOUS DISEASES

Environmental or occupational dermatologic infectious diseases are diseases that have a major manifestation on the skin surface and that result from exposure to an infectious agent found in the environment or workplace. (The very common secondarily-infected wounds will not be discussed here.) Many of the environmental and occupational dermatologic infectious diseases result not only in cutaneous signs and symptoms but also in systemic effects as well. The exposure can occur through direct skin contact (epicutaneous), inoculation (percutaneous), or through the respiratory system (inhalational).

### Public Health Importance

Epidemiologic data specifically related to environmental or occupational dermatologic infectious diseases are very limited. Other than limited descriptions in case presentations, case studies, and epidemic investigation reports, little is known about the epidemiology of most of these diseases in the United States. For the infectious diseases that are nationally reportable, it is impossible to determine what proportion are due to occupational exposures. In 2001, BLS estimated 38,900 cases of occupational skin diseases or disorders in the U.S. workforce.[5] Further information is available on the 6,051 cases that involved days away from work. Of this subgroup, 710 (11.7 percent) had infections of

the skin and subcutaneous tissue; their median time away from work was 5 days.

### Population at Risk and Etiologic Agents

Environmental and occupational dermatologic infectious diseases can be grouped by etiologic agent into the following disease categories: bacterial, rickettsial, viral, superficial fungal, subcutaneous fungal, systemic fungal, and parasitic.[24] In general, risk of infection can be associated with individual susceptibility, which includes factors such as immune status and trauma to the skin breaching its protective barrier; the distribution of the pathogen in the environment; and exposure to the pathogen, considering its reservoir, mode of transmission, and conditions in which the pathogen thrives. Reservoirs and fomites of the pathogens include people, such as co-workers, clients, patients, or children; animals and animal products; soil and plant materials; ticks and insects; and water and marine life. Conditions in which pathogens can thrive and increase susceptibility include wet conditions, such as wet work, and hot and humid environments. The environmental and occupational dermatologic infectious diseases associated with these sources and conditions are listed in Table 28-3. In addition, laboratory personnel working directly with pathogens are at risk of infection. Recently, there has been concern over possible work-duty exposures for first responders and health care professionals as part of bioterrorist events, such as deliberate releases of anthrax or smallpox.

### Diagnosis and Treatment

Specifics on diagnosis and treatment are disease-specific and are beyond the scope of this chapter. In many cases, it is often difficult to definitively prove the environmental or occupational relatedness of the disease process. The questions to be answered by the clinician include the following:

1. Is the patient's condition a dermatologic infectious disease?
2. Is the organism found in the patient's environment?
3. Was there an opportunity for the person/worker to become infected in the environment/workplace?
4. What other exposures, such as recreational activities, must be considered?

### Prevention

The clinician should view each patient with a potential environmental or occupational dermatologic infectious disease from a broader public health perspective. A case of a dermatologic infectious disease should be viewed as a potential sentinel health event. This recognition and resultant action by the clinician, in appropriate consultation with public health authorities, could lead to potential disease prevention in other people. This can only occur with proper diagnosis, a high level of suspicion on the part of the clinician in suspecting environmental/workplace exposures, ultimate confirmation of the association to the exposures that caused the disease, and finally, steps taken to modify those exposures. If successful, this approach would lead to the prevention of relapses and of new cases of dermatologic infectious diseases.

## SKIN CANCERS

As early as 1894, Dr. P.G. Unna in Germany drew attention to the association between chronic sun exposure and skin cancers in outdoor workers, such as farmers and sailors. Skin cancers include melanoma, basal cell carcinoma, and squamous cell carcinoma. Studies have shown an association between excessive sun exposure and premature skin aging, pre-skin cancers (actinic keratoses), and skin cancer. Non-ionizing ultraviolet radiation (UVR) from the sun is the primary cause of skin cancer, in general, and is also the primary cause of occupational skin cancer. In addition, a variety of chemical exposures may play a role in the etiology of skin cancers.

### Public Health Importance

Melanoma is the least prevalent of the three skin cancers but has the greatest risk of fatality, accounting for 85 percent of skin cancer deaths. The American Cancer Society estimated that, in 2004, more than 55,000 Americans would be diagnosed with melanoma and 7,900 would die of this disease.[25] Melanoma is likely to be related to excessive sun exposure, although the relationship is complex; it seems to be associated with severe sunburns during childhood. Basal cell carcinoma and squamous cell carcinoma are more clearly related to sun exposure, probably as a result of cumulative, chronic exposure. Basal cell and squamous cell skin cancers are, by far, the most common cancers in the United

**TABLE 28-3**

**Exposures Associated with Dermatologic Infectious Diseases**

| Other People, Patients, and Children | Animal and Animal Products | Soil and Plants |
|---|---|---|
| Tuberculosis (cutaneous) | Anthrax | Anthrax |
| Herpetic whitlow | Brucellosis | Dermatophytes (geophilic) |
| Warts | Cat scratch disease | Chromomycosis |
| Measles | Erysipeloid | Mycetoma |
| Rubella | Tuberculosis (cutaneous) | Sporotrichosis |
| Chickenpox | Tularemia | Blastomycosis |
| Herpes zoster (shingles) | Orf | Paracoccidioidomycosis |
| Hand-foot-mouth disease | Milker's nodules | Cutaneous larva migrans |
| Erythema infectiosum (fifth disease) | Cowpox | **Wet Work and Hot and Moist Environments** |
| Dermatophytes (anthropophilic) | Monkeypox | Candidiasis |
| Scabies | Warts | Dermatophytoses |
| **Ticks and Insects** | Dermatophytes (zoophilic) | *Tinea versicolor* |
| Lyme disease | **Water, Marine, Fish, and Shellfish Exposures** | |
| Tularemia | Erysipeloid | |
| Spotted fevers (Rocky Mountain spotted fever) | *Mycobacterium marinum* granuloma | |
| Typhus | Tularemia | |
| Ehrlichiosis | *Vibrio vulnificus* infection | |
| Leishmaniases | *Aeromonas hydrophila* infection | |
| | *Vibrio parahaemolyticus* infection | |
| | *Pseudomonas aeruginosa* infection | |
| | Warts | |
| | Cercarial dermatitis | |

States with up to 1 million Americans affected each year and more than 2,000 deaths. Accurate data on the general prevalence of skin cancers related to occupational exposures are not available.

## Population at Risk and Etiologic Agents

Implicated etiologies for skin cancers include nonionizing radiation from sunlight exposure and other sources of UVR, ionizing radiation, and thermal and chemical stimuli. Outdoor workers may receive up to six to eight times the dose of UVR compared to indoor workers,[26] and rates for some skin cancers among outdoor workers have been associated with cumulative UVR exposure.[27] According to the BLS, in 2003, more than 6 percent of the workforce (more than 8 million workers) were listed in the following potential outdoor occupations: construction, farm, and forestry workers; fishing workers; gardeners; groundskeepers; mail carriers; amusement/recreation attendants; and surveying and mapping workers. There are likely many more workers occupationally exposed to UVR from sunlight. In addition, workers exposed to chemical agents, such as polycyclic aromatic hydrocarbons, arsenic, alkylating agents, and nitrosamines, may be at increased risk. Arsenic intoxication, which can result from ingestion of contaminated well water, has resulted in hyperpigmentation, palmar and plantar arsenical keratoses, and superficial squamous cell and basal cell carcinomas. Other risk factors for skin cancers include Northern European or Celtic family origins and fair skin types.

### Diagnosis and Treatment

Diagnosis is based on history, physical findings, and pathology results. Treatment of specific skin cancers, which is beyond the scope of this chapter, depends on the specific type of skin cancer, size, depth, and location of the lesion, and evidence of metastases.

### Prevention

The strategies for prevention include preventing excessive UVR exposure by limiting exposure to sunlight, introducing changes in practices to limit sun exposure during peak UVR hours (10 a.m. to 4 p.m.), wearing UVR-protective clothing and wide-brimmed hats, using broad-spectrum sunscreens (blocking both UVA and UVB), and wearing UV-blocking sunglasses. Limiting skin exposure to chemicals known to play a role in skin cancers is also important.

In many areas, the National Weather Service, in cooperation with the Environmental Protection Agency, issues daily predictions for UVR exposure. The daily UV Index, reported on a scale from 0 up to 11+ (11+ being extreme), is part of selected local weather broadcasts and can be used to warn outdoor workers and others of potential high-exposure days, when prevention strategies should be emphasized.

## OTHER SKIN DISEASES

Many other skin diseases may be related to environmental and occupational exposures (Table 28-4). Other skin diseases may not be caused by occupational exposures but may be exacerbated by such exposures. Examples include lesions of psoriasis produced at sites of skin friction or injury, heat exacerbating rosacea, and wet work initiating dyshidrotic eczema.

## CONCLUSION

Environmental and occupational skin diseases include allergic contact dermatitis, irritant contact dermatitis, contact urticaria, a variety of infectious diseases, skin cancers, and other diseases.

**TABLE 28-4**

**Other Environmental and Occupational Skin Disorders and Examples of Associated Exposures**

| Disorder | Associated Exposures |
|---|---|
| Hyperkeratoses/calluses/fissuring/blistering | Mechanical trauma |
| Burns | Heat, electricity, radiation, acids, alkalis |
| Frostbite/immersion foot, chilblain | Cold, moist environments |
| Folliculitis/furuncles and acneform dermatoses | Oils, greases |
| Chloracne | Chlorinated hydrocarbons |
| Photodermatitis (phototoxic and photoallergic) | Plants, coal tar, creosote, fragrances |
| Depigmentation/leukoderma | Phenols, hydroquinones |
| Hyperpigmentation/occupational melanosis | Coal tar, pitch |
| Skin discolorations | Silver, gold |
| Occupational Raynaud disease/vibration white finger | Tools causing hand/arm vibration |
| Miliaria rubra/prickly heat | Hot, humid work environments |
| Asteatotic eczema/winter eczema | Cool, dry work environments |
| Granulomatous dermatoses | Beryllium, zirconium |
| Ulcerative lesions | Chromium, chemical burns |
| Connective tissue disorders such as scleroderma | Silica, vinyl chloride |
| Nail disorders | Mechanical trauma, contact dermatitis, infections |
| Alopecia | Chlorbutadine, dimethylamine |

Thorough investigations of workers with occupational skin diseases can be difficult. Workers should be encouraged to report all potential work-related skin problems to their employers and to their physicians. Because the work-relatedness of skin diseases may be difficult to prove, each person with possible work-related skin problems needs to be fully evaluated by a physician, preferably one familiar with occupational/dermatological conditions. A complete evaluation would include a full medical and occupational history and a review of exposures; a medical examination; diagnostic tests, such as skin patch tests to detect causes of allergic contact dermatitis; and complete follow-up to note the progress of the affected worker. Individuals with occupational skin diseases should be protected from exposures to presumed causes or exacerbators of the disease. In some cases of allergic contact dermatitis and contact urticaria, workers may have to be reassigned to areas where exposure is minimized or nonexistent.

Environmental and occupational skin diseases as diseases have a major public health impact. They are common, often have a poor prognosis, and result in a noteworthy economic impact for both affected individuals and society as a whole, as they effect vocational and avocational activities. They are also diseases amenable to public health interventions. The U.S. Public Health Service goal for 2010, as stated in its *Healthy People 2010: National Health Promotion and Disease Prevention Objectives*, is to reduce national occupational skin disorders or diseases to an incidence of no more than 46 per 100,000 full-time workers.[28] Both irritant and allergic contact dermatitis are considered priority research areas, as outlined in the National Occupational Research Agenda, introduced in 1996 by NIOSH.[29] Increased knowledge and awareness of environmental and occupational skin diseases by health care professionals will assist in achieving the national public health goals.

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