

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 also subject 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 and occupational skin disorders can be grouped into the following categories:

1. *Physical insults:* Friction, pressure, trauma, vibration, heat, cold, variations in humidity, radiation (ultraviolet, visible, infrared, and ionizing), 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 in every aspect of our environment and can affect the skin in the home setting, during outdoor and leisure

activities, while involved in hobbies, and in the work environment. Occupational dermatology is the facet of dermatology that deals with skin diseases whose etiology or aggravation is related to some exposure in the workplace. The role of a health care practitioner involved in occupational dermatology is not only to diagnose and treat patients but also to determine the etiology of occupational skin diseases and make recommendations for their prevention. Making the diagnosis and offering treatment, determining etiology, and recommending preventive measures all can 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% of all occupational skin diseases.^{1,2} Contact dermatitis—both irritant and allergic—is an inflammatory skin condition caused by skin contact with an exogenous agent or agents, with or without a concurrent exposure to a contributory physical agent, such as ultraviolet light. It 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, while allergic contact dermatitis is a type IV, delayed or cell-mediated, immune reaction. There are over 57,000 chemicals reported to cause skin irritation, but only 3,700 chemicals are known skin allergens.³ These are mostly confined to small-molecular-weight chemicals that act as haptens, and usually only a small percentage 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. Over 80% of occupational contact dermatitis involves the hands.³⁻⁵ 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 in 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 remote 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. 22-1, 22-2 and 22-3).

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.⁶

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 2006 skin rash was the principal reason for 10.1 million patient visits—1.1% of all visits for that year.⁷ Based upon 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. Although dated, the Occupational Health Supplement data are the most complete available. The Supplement was scheduled to be repeated in 2010.⁸ The survey consisted of personal interviews of people in randomly selected households. For 30,074 people participating in the NHIS, the period prevalence was 11.2% for all dermatitis and 2.8% for contact dermatitis. 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.⁹



Figure 22-1. Acute contact dermatitis from exposure to ethylene oxide, a strong irritant.



Figure 22-2. Subacute dermatitis from the rubber accelerator mercaptobenzothiazole, which is found in the rubber in a work boot.



Figure 22-3. Chronic dermatitis from exposure to kerosene, a solvent that was used for cleaning the skin.

Table 22-1. Number and Rate of Occupational Skin Diseases per Year, United States, 2002–2007

Year	Number (in thousands)	Rate (per 100,000)
2002	44.9	51
2003	43.4	49
2004	38.9	44
2005	40.1	44
2006	41.4	45
2007	35.3	37

Source: Bureau of Labor Statistics Annual Survey, 1973–2007.

BLS estimated 35,300 cases of occupational skin diseases or disorders in the U.S. workforce.¹⁰ However, because of BLS survey limitations, it has been estimated that the number of actual occupational skin diseases may be 10 to 50 times higher than that reported by the BLS.¹¹ This increase would potentially raise the number of occupational skin disease cases to between 350,000 and 1.8 million per year. In 2007, BLS data showed an annual incidence rate of 37 cases per 100,000 workers.¹⁰

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%. Projecting these results to the U.S. working population resulted in an estimate of almost 1.9 million people with occupational contact dermatitis and a 1-year period prevalence of 1,700 per 100,000 workers for the year.⁹ 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 work days and loss of productivity. In 1984, the estimated annual direct and indirect costs of occupational skin diseases exceeded \$22 million.¹¹ 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 about \$220 million to \$1 billion.¹¹ (These estimates do not include costs of occupational retraining.)

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 176,000 employers selected to represent all private industries in the United States.¹⁰ All occupational skin diseases or disorders, including contact dermatitis, are tabulated in this survey. BLS data show that occupational skin diseases consistently accounted for 30% to 45% of all cases of occupational illnesses from the 1970s through the mid-1980s, and in recent years accounted for nearly 18% of all occupational illness.¹⁰ 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 2002 to 2007 are shown in Table 22-1. In 2007,

The Safety and Health Assessment and Research for Prevention (SHARP) program analyzed data from Washington State workers' compensation dermatologic claims and work-related skin diseases reported through SHARP's "sentinel provider network" in the 1993-1997 period. During these 5 years, close to 5,000 claims were accepted for work-related skin disorders and 42,471 lost workdays were reported, costing more than \$1.6 million in time loss payments and \$1.5 million in medical bills. Comparison with provider network data estimated that compensation data underrepresents the number of work-related skin disorders by more than four-fold.¹²

An analysis of Oregon workers' compensation claims data for 1990 through 1997 estimated the average claim rate of occupational dermatitis to be 5.7 per 100,000 workers. In this 8-year period, 727 workers' compensation claims were filed for occupational dermatitis, of which 611 were determined to be compensable. The total cost of all dermatitis claims was \$2.2 million, averaging about \$270,000 annually. Oregon claim rates are lower than other states since reporting is not mandatory unless the incident requires 3 or more days of disability leave, and injuries from self-employed workers, such as hairdressers, are not reported. The average cost per claim was \$3,552, and the average disability time was 24 days.¹³

A review of 2001 BLS data showed that, of the 38,900 reported cases of occupational skin diseases, 6,051 (16%) resulted in days away from work.¹⁰ The mean time away from work was 3 days, but 19% of lost workday cases had 11 or more days away from work. Of those with days away from work, 78% had a diagnosis of dermatitis. In 2007, of the 35,300 skin disease cases, 5,640 (16%) resulted in days away from work, with a median of 4 days lost.¹⁰ Of these, 64% had dermatitis.

Studies on the prognosis of occupational contact dermatitis stress the importance of primary prevention. A questionnaire survey of 124 patients 5 years after they were initially diagnosed with irritant hand dermatitis found 18% with low, 50% with medium, and 32% with severe hand dermatitis. Severity was measured by self-reported frequency of relapses, frequency of dermatologist visits, and use of topical corticosteroids.¹⁴ A questionnaire survey of 540 patients 1 year after

initial diagnosis of occupational hand dermatitis found 41% improved and 25% with persistently severe or aggravated symptoms. Poor prognosis was associated with the presence of atopic dermatitis and being 25 years of age or older. Prognosis was not affected by whether the dermatitis was irritant or allergic. Those with severe occupational hand dermatitis at baseline had a higher risk of taking sick leave and job loss in the following year than those with mild cases. The study found no significant improvement in the disease after the change of job. Severe impairment of quality of life at baseline was a strong predictor of prolonged sick leave, but the presence of depression did not affect prolonged sick leave.¹⁵

Persistent postoccupational dermatitis (PPOD) can occur following allergic and irritant contact dermatitis. Persistent postoccupational dermatitis begins as a clear-cut occupational contact dermatitis, initially gets better when removed from exposure, but with time the capacity for resolution is lost and persistent dermatitis develops. Predictive factors for PPOD include duration of disease, inability to avoid causative agents, and age.¹⁶ Widespread hand dermatitis on initial examination was found to be the greatest factor for a poor long-term prognosis; other important factors identified include young age at onset of hand dermatitis, history of childhood eczema, and contact allergy.¹⁷ 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.¹⁸⁻²¹

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 twenty-first 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 are many 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 2007 are shown in Table 22-2.¹⁰ The greatest number of cases of occupational skin diseases is seen in manufacturing, but the highest incidence rate is seen in the category agriculture, forestry, fishing, and hunting.

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%); public transport attendants, cosmetologists, and other personal service occupations (4.9%); health care therapists, technologists, technicians, and assistants (3.5%); and mechanics and repairers of vehicles, engines, heavy equipment, and machinery (3.5%).⁹ Of all accepted workers' compensation claims for occupational contact dermatitis in Oregon, the occupations with highest claim rates were farming, fishing, and forestry workers (18.2%); machine operators and assemblers (16.5%); service-related workers (15.3%); laborers (13.7%); precision production crafts workers (8.0%); and protective services workers (5.7%), followed by technicians and related support workers, transportation and material movers, and professional

Table 22-2. Number and Incidence of Occupational Skin Diseases, by Industry Sector, 2007

Industry	Number	Incidence per 100,000
Agriculture/forestry/fishing/hunting	1,200	123
Manufacturing	8,800	62
Education and health services	7,700	57
Leisure and hospitality	4,100	46
Construction	2,600	36
Professional and business services	3,600	26
Other services	800	26
Trade/transport/utilities	5,500	24
Information	400	17
Mining	<100	13
Financial activities	600	8
Total	35,300	37

Source: Bureau of Labor Statistics Survey

specialty, administrative support, executive, administrative, and sales employees.¹³ Self-employed individuals, such as hairdressers and cosmetologists, are not represented in these claims.

The etiology of irritant contact dermatitis is often multifactorial, but the most common skin irritant is wet work, defined as exposure of skin to liquid for more than 2 hours per day, use of occlusive gloves for more than 2 hours per day, or frequent hand cleaning.^{22,23} Other common causes of irritant contact dermatitis include soaps and detergents, solvents, food products, cleaning agents, plastics and resins, petroleum products and lubricants, metals, and machine oils and coolants.^{22,23} Frictional irritant contact dermatitis can be caused from low humidity, heat, paper, tools, metals, fabrics, plastics, fibrous glass and other particulate dusts, and cardboard, among other causes.^{24,25} Causes of allergic contact dermatitis include plants (poison ivy, poison oak, and poison sumac), metallic salts, germicides, plastic resins, rubber additives, and fragrances.²⁶ The most common skin patch test allergens found to be positive in North American dermatologic patients along with potential sources of exposure are shown in Table 22-3.²⁷ In health care workers with occupational contact dermatitis who were skin-patch tested, the most common relevant allergens included thiuram mix, carba mix, and glutaraldehyde.²⁸ The most relevant skin allergens in patients with occupational hand dermatitis who were skin-patch tested included thiuram mix, carba mix, bacitracin, methyl dibromoglutaronitrile/phenoxyethanol, formaldehyde, nickel, and cobalt.²⁹

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 health professional who makes the diagnosis and confirms 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.^{2,30} The diagnosis is based on the medical history, including the occupational and environmental history, and physical findings.

Table 22-3. North American Contact Dermatitis Group Patch-Test Results, 2003–2004²⁷

Test Substance	Common Sources	Percent Positive
Nickel sulfate 2.5% pet	Metals, jewelry	18.7
Neomycin sulfate 20% pet	Creams, lotions	10.6
<i>Myroxylon pereirae</i> 25% pet		10.6
Fragrance mix 8% pet	Toiletries, scented products	9.1
Quaternium-15 2% pet	Cosmetics, sunscreens	8.9
Sodium gold thiosulfate 0.5% pet	Jewelry, dental products	8.7
Formaldehyde 1% aq	Fabrics, skincare products	8.7
Cobalt chloride 1% pet	Metals, jewelry	8.4
Bacitracin 20% pet	Ointments, creams	7.9
Methyl dibromoglutaronitrile/phenoxyethanol 2.5% pet	Biocides, skincare products	6.1
p-Phenylenediamine 1% pet	Hair dyes, leather	4.7
Thiuram mix 1% pet	Rubber, pesticides	4.6
Potassium dichromate 0.25% pet	Cement, leather	4.3
Carba mix 3% pet	Rubber, pesticides	4.0
Diazolidinylurea 1% pet		3.5
Propylene glycol 30% aq	Cosmetics, topical medications	3.3
Imidazolidinylurea 2% pet		2.9
Colophony 20% pet		2.8
Triamcortol-21-pivalate 1% pet		2.7
Diazolidinylurea 1% aq		2.5
Ethylenediamine dihydrochloride 1% pet		2.4

Note: Prevalence of 20 most common positive reactions (n varies from 5,106 to 5,145).
aq, in aqueous solution; pet, in petrolatum.

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.³¹ In irritant contact dermatitis, there are no additional confirmatory tests. Patch tests may be used to distinguish allergic contact dermatitis from irritant contact dermatitis.²³ 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. Irritant contact dermatitis may be overestimated (and allergic contact dermatitis underestimated) due to time, expense, and availability of skin patch testing; physician experience; and the limited availability of allergens in the United States.³² 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 when the person is away from the exposure to the suspected irritant or allergen?
7. Do patch tests or provocation tests identify a probable causal agent?³⁰

Treatment and Prevention

Avoiding etiologic irritants and allergens, in addition to wet work, 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. Liberal use of skin moisturizers helps to prevent contact dermatitis by maintaining a healthy skin barrier, and it also helps to repair this barrier if it has been compromised.²³ 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 in the prevention of occupational contact dermatitis include the following:

- Identifying irritants and allergens
- Substituting chemicals that are less irritating or allergenic
- Establishing engineering controls to reduce exposure
- Utilizing personal protective equipment (PPE), such as gloves and special clothing
- Emphasizing personal and occupational hygiene
- Establishing educational programs to increase awareness in the workplace³³

Chemical changes in industrial materials have been beneficial. For example, the addition of ferrous sulfate to cement to reduce the hexavalent chromium content has been effective in reducing occupational allergic contact dermatitis in Europe. Protective gloves can reduce or eliminate skin exposure to hazardous substances if used correctly, but they may actually cause or worsen hand dermatitis (by permeation and penetration) if selected poorly and used improperly (by contamination).³⁴ The use of PPE may occlude irritants or allergens next to the skin, and PPE components may directly irritate the skin, so the correct use of PPE is at least as important as their selection.³⁵ Similarly, the excessive pursuit of personal hygiene in the workplace may actually lead to misuse of soaps and detergents and resulting irritant contact dermatitis. Proper hand-washing methods and adequate moisturizing is valuable in preventing contact dermatitis.⁵ The effectiveness of barrier creams is controversial since there are limited data on the protective nature of these topical

products during actual working conditions involving high-risk exposures. Educating the workforce about skin care, exposures, and PPE use is an especially important measure in the prevention of occupational contact dermatitis.³⁶⁻³⁸

CONTACT URTICARIA

Urticaria is defined as the transient appearance of elevated, erythematous pruritic wheals or ser-piginous 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, earlobes, external genitalia, and mucous membranes.

Urticarial lesions can be classified in one or more of the following categories based upon 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 proven 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 non-immunologic, immunologic, or not known.³⁹ Contact urticaria is defined as urticaria that occurs after direct skin contact with a substance. Another type of immediate skin reaction, "protein contact dermatitis," has clinical features of both immediate and delayed hypersensitivity and is associated with atopy. Pruritis, erythema, and urticarial or vesicular lesions occur within 30 minutes of contact with proteins (fruits, vegetables, spices, plants, grains, enzymes, or animal proteins) followed by eczematous dermatitis. Protein contact dermatitis typically affects the hands.³⁹⁻⁴² 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 water or to physical agents, such as cold, heat, and solar radiation.

Public Health Importance

Data specific for environmental and occupational urticaria are limited. In 2007, BLS estimated 35,300 cases of occupational skin diseases or disorders in the U.S. workforce.¹⁰ Further information is available on the 5,640 cases that involved days away from work. Of this subgroup, 80 (1.4%) had urticaria/hives. Their median time away from work was 4 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, and ulcers; and, in some cases, occupation. Based upon 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, bananas, beans, beer, caraway seeds, carrots, eggs, endives, fish, garlic, grains, kiwi fruit, lettuce, meat (beef, chicken, lamb, liver, pork, and turkey), milk, onions, olives, peaches, potatoes, rice, shellfish, spices, strawberries, and tomatoes.^{39,41,43-46} Bakers can develop contact urticaria and other systemic symptoms after exposure to rye, wheat, barley, oat, and buckwheat flours, cinnamon, vanillin, and additive flour enzymes, such as alpha-amylase.^{39,41,44}

In health care, dental, and pharmaceutical environments, dermal 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, cefotiam, cephalosporins, chloramine, chloramphenicol, chlorhexidine, chlorocresol, ethylene oxide, gentamicin, neomycin, nitrogen mustard, penicillin, pentamidine isethionate, phenothiazines, piperacillin rifamycin, and streptomycin.^{39,45,46} Recent studies have found that the increased use of non-powdered latex gloves and nonlatex gloves in health care settings have resulted in fewer cases of natural rubber latex allergy, at one time an important cause of contact urticaria in health care professionals.⁴⁷⁻⁴⁹

Contact urticaria has been found to be caused by animal hair, dander, placenta, saliva, seminal fluid, amniotic fluid, milk, blood, insects, and bacterial and fungal enzymes.^{39,50} Slaughterhouse workers, laboratory workers, veterinarians and their staff, and dairy farmers are at risk for developing contact urticaria when exposed to these allergens.

Certain woods and plants can cause contact urticaria. These include the larch, limba, obeche (African maple), and teak woods and plants, such as algae, cacti, chrysanthemum, *Ficus benjamina* (weeping fig), lilies, *Limonium tataricum*, *Phoenix canariensis* (canary palm),

Spathiphyllum walisii (spathe flower), tobacco, tulips, and fungi (shiitake mushrooms). 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.³⁹

A variety of industrial chemicals can cause contact urticaria, including the following: acrylic monomers (plastics), polyfunctional aziridine hardener (aziridine reacted with a multifunctional acrylic), aliphatic polyamines (epoxy resins), alkyl-phenol novolac resin, ammonia, castor bean (fertilizers), diethyltoluamide (DEET), formaldehyde (used in clothing, leather, fumigation, and resins), isocyanates, lindane (a parasiticide), paraphenylenediamine, phenylmercuric propionate (an antibacterial fabric softener), plastic additives (such as butylhydroxytoluene and oleylamide), reactive dyes, sodium sulfide (used in photographs, dyes, and tanning), sulfur dioxide, vinyl pyrrolidone, xylene, and other solvents.^{39,45,46} 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 the following:

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 non-occupational causes

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. Evaluating with both prick and patch testing has been recommended to avoid missing a common accompanying allergic contact dermatitis.^{39,44,51}

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
- Utilizing PPE, such as gloves and special clothing
- Emphasizing personal and occupational hygiene
- 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 the National Institute for Occupational Safety and Health [NIOSH].⁵²

DERMATOLOGIC INFECTIOUS DISEASES

Environmental or occupational dermatologic infectious diseases are diseases that result from exposure to an infectious agent found in the environment or workplace and have a major manifestation on the skin surface. (Secondarily infected wounds will not be discussed here.) Many environmental and occupational dermatologic infectious diseases not only cause cutaneous signs and symptoms but also systemic effects. Exposure can occur through direct skin contact (epicutaneous), inoculation (percutaneous), or via 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. In many cases, it is difficult to definitively prove that the disease process is occupationally related. There is limited information on occupational dermatologic infectious diseases in the BLS data. In 2006, there were an estimated 41,400 cases of occupational skin diseases or disorders, or 4.5 per 10,000 workers.¹⁰ Of these, 5,720 resulted in one or more days away from work. Infections of the skin and subcutaneous tissue accounted for 27%, or 1,570 cases (0.2 per 10,000 workers). Most of these cases were listed as cellulitis and abscess (690); the others were included in unspecified diseases (630), not elsewhere classified (180), and pilonidal cyst (40).¹¹ Median time away from work was 10 days. In 2006, under a separate category of infectious and parasitic diseases, the BLS recorded 2,550 cases, which resulted in at least 1 day away from work.¹⁰ Few diagnoses were listed in this category, but diagnoses with potential skin manifestations included scabies/chiggers/mites (1,330), viral diseases accompanied by exanthem (380), chickenpox (280), herpes

zoster (40), dermatophytosis, including athlete's foot and tinea (40), and Lyme disease (20).

Population at Risk and Etiologic Agents

Environmental and occupational dermatologic infectious diseases can be grouped by etiologic agent into the following categories: bacterial, rickettsial, viral, superficial fungal, subcutaneous fungal, systemic fungal, and parasitic.⁵³ In general, risk of infection can be associated with individual susceptibility, including 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 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 22-4. 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 during a bioterrorist attack.

Diagnosis and Treatment

In many cases, it is often difficult to definitively prove the environmental or occupational relatedness of the disease process. 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 to become infected in the workplace or general environment?
4. What other exposures, such as recreational activities, must be considered?

Table 22-4. Exposures Associated with Dermatologic Infectious Diseases

People, Patients, and Children	Animals and Animal Products	Soil and Plants
Tuberculosis (cutaneous)	Anthrax	Anthrax
Methicillin-resistant <i>Staphylococcus aureus</i> infection	Brucellosis	Dermatophytes (geophilic)
Herpetic whitlow	Cat scratch disease	Chromomycosis
Warts	Erysipeloid	Mycetoma
Measles	<i>Mycobacterium bovis</i> infection	Sporotrichosis
Rubella	Tularemia	Blastomycosis
Chickenpox	Methicillin-resistant <i>Staphylococcus aureus</i> infection	Paracoccidioidomycosis
Herpes zoster (shingles)	Orf	Cutaneous larva migrans
Hand-foot-mouth disease	Milker's nodules	Wet Work and Hot and Moist Environments
Erythema infectiosum (fifth disease)	Monkeypox	Candidiasis
Dermatophytes (anthropophilic)	Warts	Dermatophytoses
Scabies	Dermatophytes (zoophilic)	Tinea versicolor
	Mites	
Ticks and Insects	Water, Marine, Fish, and Shellfish Exposures	
Lyme disease	Erysipeloid	
Tularemia	<i>Mycobacterium marinum</i> granuloma	
Rocky Mountain spotted fever	Tularemia	
Typhus	<i>Vibrio vulnificus</i> infection	
Ehrlichiosis	<i>Aeromonas hydrophila</i> infection	
Leishmaniasis	<i>Photobacterium (Vibrio) damsela</i> infection	
	<i>Vibrio parahaemolyticus</i> infection	
	<i>Pseudomonas aeruginosa</i> infection	
	Warts	
	Cercarial dermatitides	

Diagnosis and treatment are disease-specific and thus beyond the scope of this chapter.

Prevention

Clinicians should view each case of a potential environmental or occupational dermatologic infectious disease from a broader public health perspective as a potential sentinel health event. This recognition and resultant action by clinicians, in appropriate consultation with public health officials, could lead to potential disease prevention in other people. This can only occur with proper diagnosis, a high level of suspicion by the clinician in suspecting environmental or workplace exposures, ultimate confirmation of the association to the exposures that caused the disease, and implementation of measures to reduce these exposures. If successful, this approach would lead to the prevention of

relapses and of new cases of dermatologic infectious diseases.

SKIN CANCERS

In 1775, Sir Percival Pott in England first made the link between occupational exposures (soot clinging to skin in chimney sweeps) and skin cancer (squamous cell carcinoma of the scrotum). In 1894, Dr. Paul 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. Excessive sun exposure is associated with premature skin aging, actinic keratosis (a type of premalignant skin change), and skin cancer.⁵⁴ Nonionizing 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. The International Agency for Research on Cancer has noted that there is sufficient evidence to establish UVR as a human carcinogen. 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 it carries the greatest risk of fatality, accounting for 85% of skin cancer deaths in the United States. The American Cancer Society estimated that, for 2008, there would be 62,000 new cases of melanoma and 8,400 deaths due to this disease.⁵⁵ 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 States, with over 1 million new cases and over 2,200 deaths each year.

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,⁵⁶⁻⁵⁷ and rates for some skin cancers among outdoor workers have been associated with cumulative UVR exposure.⁵⁸ Studies have found an increased risk of skin cancer among agricultural workers, welders, watermen, police officers, physical education teachers, pilots, and cabin attendants.⁵⁹ According to the BLS, in 2003, over 6% of the workforce (over 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 as well as artificial sources, such as welding arcs. In addition, workers exposed to ionizing radiation and 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 fair skin types, fair hair, blue eye color, and having many moles or nevi.

Diagnosis and Treatment

Diagnosis is based upon 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 of prevention are primarily based on preventing excessive UVR exposure.⁶⁰ This can be accomplished 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, generously applying broad-spectrum, water-resistant sunscreens that block both UVA and UVB, and wearing UV-blocking sunglasses. Limiting skin exposure to chemicals known to play a role in skin cancer 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 <2 (very low) to 11+ (very high), 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 ENVIRONMENTAL AND OCCUPATIONAL SKIN DISEASES

Many other skin diseases may be related to environmental and occupational exposures (Table 22-5).

Table 22-5. Other Environmental and Occupational Skin Diseases and Examples of Associated Exposures

Condition	Associated Exposures
Hyperkeratoses/calluses/fissuring/blistering	Mechanical trauma
Burns	Heat, electricity, radiation, acids, alkalis
Frostbite/immersion foot, chilblain	Cold, moist environments
Folliculitis/furuncles and acneiform 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

Other skin diseases may not be caused by occupational exposures, but they 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. 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 and dermatological conditions. A complete evaluation includes a full medical history, including an occupational and environmental history, and a review of exposures. NIOSH is revamping its skin notations for use in distinguishing between systemic, localized, and sensitizing health effects of dermal chemical exposures.⁶¹ A complete evaluation also includes a

physical examination; diagnostic tests, such as skin patch tests to detect causes of allergic contact dermatitis; and follow-up to assess the clinical course of the affected person. 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 minimal or nonexistent.

Environmental and occupational skin diseases have a major public health impact. They are common, often have a poor prognosis, and result in a substantial economic impact for both affected individuals and society as a whole. Importantly, these diseases are amenable to public health interventions.

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FURTHER READING

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