25

Skin Disorders

Loren C. Tapp and Boris D. Lushniak

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 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. *Biological causes*: Plants, bacteria, rickettsiae, 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 healthcare 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 emphasizes 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 are not 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 4,350 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 acute 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.[4,5] 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 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 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 dermatitis from allergic contact dermatitis, as both can have a similar appearance and both can be clinically

Figure 25-1. Acute contact dermatitis from exposure to ethylene oxide, a strong irritant.

Figure 25-2. Subacute dermatitis from the rubber accelerator mercaptobenzothiazole, which is found in the rubber in a work boot.

Figure 25-3. Chronic dermatitis from exposure to kerosene, a solvent that was used for cleaning the skin.

evident as an acute, subacute, or chronic condition (Figures 25-1, 25-2, and 25-3).

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 2012 skin rash was the principal reason for 12.5 million patient visits—1.3% 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 2010, the National Health Interview Survey (NHIS) included an Occupational Health Supplement, which included questions on dermatitis. The survey consisted of personal interviews of people in randomly selected households. For 17,524 workers participating in the NHIS, the overall prevalence was 9.8% for all dermatitis; 5.6% of these cases were attributed to work by health professionals. Projecting these results to the U.S. working population resulted in an estimate of 15.2 million workers with dermatitis and 850,000 workers with work-related dermatitis.[8]

Specific national occupational disease and illness data are available from the U.S. Bureau of Labor Statistics (BLS), which conducts annual surveys of approximately 200,000 employers selected to represent all private industries in the United States.[9] All occupational skin diseases or disorders, including contact dermatitis, are tabulated in this survey. Bureau of Labor Statistics 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 15 to 18% of all occupational illness.[9] A decline in this proportion may be partially related to an increase seen in disorders associated with repeated trauma.

Table 25-1. Reported Incidence of Occupational Skin Diseases, United States, 2002–2014

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
2008	35.8	38
2009	25.9	29
2010	24.9	29
2011*	–	–
2012*	–	–
2013	26.0	28
2014	21.8	23

* Bureau of Labor Statistics data from 2011 and 2012 contain incorrect national-level estimates and are not shown. http://www.bls.gov/bls/errata/iif_errata_1014.htm

Bureau of Labor Statistics data for occupational skin diseases for 2002 to 2014 are shown in Table 25-1. In 2014, the BLS estimated 21,800 cases of occupational skin diseases or disorders in the U.S. workforce.[9] 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.[10] This increase would potentially raise the number of occupational skin disease cases to between 218,000 and 1.1 million per year. In 2014, BLS data showed an annual incidence rate of 23 cases per 100,000 workers.[9]

In 2010, the Occupational Health Supplement of the NHIS indicated that the period prevalence for occupational contact dermatitis occurring in the preceding year was 0.55%. Projecting these results to the U.S. working population resulted in an estimate of almost 850,000 people with occupational contact dermatitis and a 1-year period prevalence of 550 per 100,000 workers for the year.[8] 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 lost productivity. 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" from 1993 through 1997. During these 5 years, close to 5,000 claims were accepted for work-related skin disorders and 42,471 lost workdays (days away from work) 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.[11]

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, illnesses 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.[12]

A review of 2007 BLS data showed that, of the 35,300 reported cases of occupational skin diseases, 5,640 (16%) resulted in days away from work.[9] The median time away from work was 4 days, but 22% of lost-workday cases had 11 or more days away from work. Of those with days away from work, 64% had a diagnosis of dermatitis. In 2014, of the 21,800 skin disease cases, 3,210 (15%) resulted in days away from work. Of these cases, 2,320 (72%) had a diagnosis of acute dermatitis, with a median of 2 days lost, and the remaining 890 cases were reported as skin and subcutaneous tissue disorders.[9] An additional 30 cases had a diagnosis of chronic dermatitis.[9]

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.[13] A questionnaire survey of 540 patients 1 year after initial diagnosis of occupational hand dermatitis found 41% were improved and 25% had 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.[14] A study that followed patients with occupational allergic contact dermatitis 2 years after diagnosis found that 89% had persistent dermatitis and that workers who had changed jobs improved significantly more frequently than those who did not.[15]

Persistent postoccupational dermatitis (PPOD) can occur following allergic or 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 in 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 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 2014 are shown in Table 25-2.[9] The greatest number of cases of occupational skin diseases is seen in education and health services, but the highest incidence rate is seen in the category natural resources and mining.

In 2010, the NHIS found that the occupational groups with the highest prevalence of dermatitis included healthcare, personal care, and service workers; workers in life, physical, and social sciences; and workers in the arts, entertainment, and recreation industries.[8] 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 specialty, administrative support, executive, administrative, and sales employees.[12] Self-employed individuals, such as hairdressers and cosmetologists, are not represented in these claims.

Table 25-2. Incidence of Occupational Skin Diseases, by Industry Sector, 2014

Industry	Number	Rate (per 100,000)
Natural resources and mining	1,000	55
Education and health services	6,100	40
Manufacturing	4,400	36
Leisure and hospitality	3,000	32
Construction	1,100	20
Professional and business services	2,100	15
Trade/transport/utilities	3,100	14
Other services	300	11
Financial activities	400	6
Information	100	5
Total	**21,600**	**23**

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, cardboard, and other exposures.[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 skin patch test allergens found to be most relevant in North American dermatologic patients along with potential sources of exposure are shown in Table 25-3.[27] Recent studies of North American food service workers who were dermatologic patients found 55% with allergic contact dermatitis; most frequent allergens included thiuram mix and carba mix found in gloves.[28] Among North American hairdressers and cosmetologists who were dermatologic patients, 73% were diagnosed with allergic contact dermatitis; relevant occupational allergens included glyceryl thioglycolate, *p*-phenyldiamine, nickel sulfate, and 2-hydroxyethyl methacrylate found in hair and nail products.[29] Among North American mechanics and repairers who were dermatologic patients, rubber accelerators (from gloves and auto parts) and methylchloroisothiazolinone/methylisothiazolinone (a preservative in oils, lubricants, solvents, and fuels) were the most comment occupationally related allergens.[30] A study of workers' compensation records in Portland, Oregon, from 2005 to 2014 found that the most relevant skin allergens in

Table 25-3. North American Contact Dermatitis Group Patch-Test Results, Ranked by Relevance, 2013–2014[27]

Test Substance	Common Sources	Rank
Methylisothiazolinone 0.2% aq	Biocides, cosmetics, toiletries	1
Nickel sulfate 2.5% pet	Metals, jewelry	2
Fragrance mix I 8% pet	Toiletries, scented products	3
Methylchlorisothiazolinone/ methylisothiazolinone 0.01% aq	Biocides, cosmetics, toiletries	4
Myroxylon pereirae (Balsam of Peru) 25% pet	Skin and haircare products	5
p-Phenylenediamine 1% pet	Hair dyes, leather	6
Fragrance mix II 14% pet	Toiletries, scented products	7
Formaldehyde 2% aq	Textiles, skincare products	8
Lanolin alcohol 50% pet	Skincare products	9
Iodopropynyl butylcarbamate 0.5% pet	Paints, skincare and haircare products	10
Formaldehyde 1% aq	Textiles, skincare products	11
Bacitracin 20% pet.	Topical medicaments	12
Quaternium-15 2% pet	Cosmetics, sunscreens	13
Cinnamic aldehyde 1% pet	Scented toiletries	14
Carba mix 3% pet	Rubber, pesticides	15
Propylene glycol 100% aq	Cosmetics, topical medications	16
Methyldibromoglutaronitrile/ phenoxyethanol 2.0% pet	Biocides, skincare products	17
Cobalt chloride 1% pet	Metals, jewelry	18
Neomycin 20% pet.	Topical medicaments	19
OPDMA 0.1% aq		20

Note. Relevance was based on the Significance-Prevalence Index Number (SPIN), a weighted calculation depending on degree of certainty ascribed to relevance.
SPIN calculation = (proportion of population allergic) × $(1 \times R_{definitive} + 0.66 \times R_{probable} + 0.33 \times R_{possible}) \times 100$.
aq = in aqueous solution; pet = in petrolatum.

patients with occupational hand dermatitis who had been skin-patch tested were thiuram mix, carba mix, potassium dichromate, epoxy resin, chloroxylenol PCMX, formaldehyde, and Quaternium-15.[31] In healthcare workers with occupational contact dermatitis who were skin-patch tested, thiuram mix was the most common relevant allergen.[31]

Diagnosis

The environmental cause or work-relatedness of contact dermatitis may be difficult to determine. 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.[2,32] 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.[33] 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.[34] Skin patch tests should only be conducted by healthcare professionals trained in conducting and interpreting the tests. Skin patch tests should never be conducted with unknown substances.

Answers to the following questions can help determine 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?[32]

Prevention

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
- Providing educational programs to increase awareness in the workplace[35]

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).[36] 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 its selection.[37] Similarly, the excessive pursuit of personal hygiene in the workplace may actually lead to misuse of soaps and detergents, resulting in irritant contact dermatitis. Proper handwashing 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.[38-40]

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, 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 nonimmunologic, immunologic, or not known.[41] *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.[41-45] 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 2014, the BLS estimated 21,800 cases of occupational skin diseases or disorders in the U.S. workforce.[9] Further information is available on the 2,320 cases that involved days away from work. Of this subgroup, 60 (2.6%) had urticaria/hives. Their median time away from work was 1 day. Among 19 patients with contact urticaria to occupational natural rubber latex, all had persistent skin symptoms 2 years after diagnosis and only 42% reported improvement; these patients had a poorer prognosis than occupational allergic contact dermatitis patients.[15]

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; healthcare workers, dental professionals, and pharmaceutical industry workers; animal handlers, such as laboratory workers and veterinarians; gardeners, florists, woodworkers, and agricultural workers; and hairdressers.

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.[41,42,44,46-49] 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.[41,42,44,47]

In healthcare, 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, cephalosporins, chloramine, chloramphenicol, chlorhexidine, chlorocresol, ethylene oxide, gentamicin, neomycin, nitrogen mustard, penicillin, pentamidine isethionate, phenothiazines, piperacillin, rifamycin, and streptomycin.[41,42,48,49] Recent studies have found that

the increased use of nonpowdered latex gloves and nonlatex gloves in healthcare settings have resulted in fewer cases of natural rubber latex allergy, at one time an important cause of contact urticaria in healthcare professionals.[50–52]

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.[41,42,53] Slaughterhouse workers, laboratory workers, veterinarians and related workers, 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 elm, larch, mahogany, mulberry, obeche (African maple), and teak woods; and plants, such as algae, cacti, *Cannabis sativa*, chrysanthemum, *Ficus benjamina* (weeping fig), lilies, *Limonium tataricum, Phoenix canariensis* (canary palm), 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.[41,42] Hair-bleaching products, such as ammonia persulfate, are common nonprotein substances that can cause contact urticaria in hairdressers.[41,54]

A variety of industrial chemicals can cause contact urticaria, including 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 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 pyrrolidone, xylene, and other solvents.[41,42,48,49] Contact urticaria can occur with exposure to a variety of metal salts, including chromium, cobalt, iridium, nickel, platinum, and rhodium.[42]

Diagnosis

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 and body location of exposure
5. Resolution of the urticaria away from the exposure
6. Exclusion of nonenvironmental or nonoccupational 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 immunoglobulin E to suspect occupational antigens, such as by radioallergosorbent (RAST) assays. Evaluating with both prick and patch testing has been recommended.[41,47,55]

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 with chemicals that are nonallergenic
- Implementing 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.

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 are not discussed here.) Many environmental and occupational dermatologic infectious diseases cause not only cutaneous signs and symptoms but also systemic effects. Exposure can occur through direct skin contact (epicutaneous), by 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 2014, there were an estimated 21,800 cases of occupational skin diseases or disorders, or 2.3 per 10,000 workers.[9] Infections of the skin and subcutaneous tissue accounted for 3.4%, or 740 cases (0.1 per 10,000 workers). Most of these cases were listed as cellulitis or abscess (480).[9] Median time away from work was 7 days. In 2014, under a separate category of infectious and parasitic diseases, the BLS recorded 1,020 cases that resulted in at least 1 day away from work.[9] Few diagnoses were listed in this category, but diagnoses with potential skin manifestations included scabies/chiggers/mites (470) and viral diseases accompanied by exanthem (120), which included chickenpox (90) and herpes zoster (20).

Population at Risk and Etiologic Agents

Environmental and occupational dermatologic infectious diseases can be grouped by etiologic agent into categories: bacterial, rickettsial, viral, superficial fungal, subcutaneous fungal, systemic fungal, and parasitic.[56] 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 coworkers, 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 25-4. In addition, laboratory personnel working directly with pathogens are at risk of infection. There has also been concern over possible work-duty exposures for first responders and healthcare professionals during a bioterrorist attack.

Diagnosis

In many cases, it is often difficult to prove definitively 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?

Diagnosis is disease-specific and therefore beyond the scope of this chapter.

Prevention

Clinicians should view each case of a potential environmental or occupational dermatologic

Table 25-4. Exposures Associated with Dermatologic Infectious Diseases

People, Patients, and Children
Tuberculosis (cutaneous)
Methicillin-resistant *Staphylococcus aureus*
Herpetic whitlow
Warts
Measles
Rubella
Chickenpox
Herpes zoster (shingles)
Hand-foot-mouth disease
Erythema infectiosum (fifth disease)
Dermatophytes (anthropophilic)
Scabies

Ticks and Insects
Lyme disease
Tularemia
Rocky Mountain spotted fever
Typhus
Ehrlichiosis
Leishmaniases

Animal and Animal Products
Anthrax
Brucellosis
Cat scratch disease
Erysipeloid
Mycobacterium bovis
Tularemia
Methicillin-resistant *Staphylococcus aureus*
Orf
Milker's nodules
Monkeypox
Warts
Dermatophytes (zoophilic)
Mites

Water, Marine, Fish, and Shellfish Exposures
Erysipeloid
Mycobacterium marinum granuloma
Tularemia
Vibrio vulnificus infection
Aeromonas hydrophila infection
Photobacterium (Vibrio) damsela infection
Vibrio parahaemolyticus infection
Pseudomonas aeriginosa infection
Warts
Cercarial dermatitides

Soil and Plants
Anthrax
Dermatophytes (geophilic)
Chromomycosis
Mycetoma
Sporotrichosis
Blastomycosis
Paracoccidioidomycosis
Cutaneous larva migrans

Wet Work and Hot and Moist Environments
Candidiasis
Dermatophytoses
Tinea versicolor

Source: Wawrose, DJ, Lushniak, BD. Occupational infectious diseases with dermatologic features. In Wright WE (ed.). Occupational and environmental infectious diseases (2nd ed.). Beverly Farms, MA: OEM Press, 2009, pp. 404–421.

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 CANCER

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, and skin cancer.[57] 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 concluded 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 73% of skin cancer deaths in the United States. The American Cancer Society estimated that, in 2016, over 76,000 people

in the United States would be diagnosed with melanoma and over 10,000 would die of this disease.[58] 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 5 million new cases and about 2,000 deaths each year.[58]

Population at Risk and Etiologic Agents

Implicated etiologies for skin cancers include non-ionizing 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,[59,60] and rates for some skin cancers among outdoor workers have been associated with cumulative UVR exposure.[61] Studies have found an increased risk of skin cancer among agricultural workers, welders, watermen, police officers, physical education teachers, pilots, and cabin attendants.[62] According to the BLS, in 2015, over 3% of the workforce (more than 4 million workers) were included in occupations frequently associated with outdoor work: construction, farm, and forestry workers; fishing workers; gardeners; groundskeepers; mail carriers; amusement/recreation attendants; and surveying and mapping workers.[9] 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 of skin cancer. 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, having many moles or nevi, family history of skin cancer, sunburn in childhood or adolescence, a weakened immune system, and older age.[58,63]

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.[64] 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.

The *2014 Surgeon General's Call to Action to Prevent Skin Cancer* emphasized the importance of workplace exposures and advocated for increasing the availability of sun protection for outdoor workers, integrating sun safety into workplace health education and promotion programs, and incorporating sun safety into workplace policies and safety training.[65]

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 25-5).

Table 25-5. 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 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

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, rosacea exacerbated by heat, 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 and occupational history and a review of exposures. The National Institute for Occupational Safety and Health is revamping its skin notations for use in distinguishing between systemic, localized, and sensitizing health effects of dermal chemical exposures.[66] 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.

AUTHORS' NOTE

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or the University of Maryland.

REFERENCES

1. Lushniak BD. Occupational contact dermatitis. Dermatologic Therapy 2004; 17: 272–277.

2. Ingber A, Merims S. The validity of the Mathias criteria for establishing occupational causation and aggravation of contact dermatitis. Contact Dermatitis 2004; 51: 9–12.
3. De Groot AC. Patch testing (3rd ed.). Wapserveen, The Netherlands: Acdegroot Publishing, 2008.
4. Flyvholm MA, Bach B, Rose M, Jepsen KF. Self-reported hand eczema in a hospital population. Contact Dermatitis 2007; 57: 110–115.
5. Warshaw E, Lee G, Storrs FJ. Hand dermatitis: A review of clinical features, therapeutic options, and long-term outcomes. American Journal of Contact Dermatitis 2003; 14: 119–137.
6. Lushniak BD. The importance of occupational skin diseases in the United States. International Archives of Occupational and Environmental Health 2000; 76: 325–330.
7. Centers for Disease Control and Prevention, National Center for Health Statistics. National Ambulatory Medical Care Survey: 2012 State and National Summary Tables. Available at: http://www.cdc.gov/nchs/data/ahcd/namcs_summary/2012_namcs_web_tables.pdf. Accessed November 21, 2016.
8. Luckhaupt SE, Dahlhamer JM, Ward BW, et al. Prevalence of dermatitis in the working population, United States, 2010 National Health Interview Survey. American Journal of Industrial Medicine 2013; 56: 625–634.
9. Bureau of Labor Statistics. Occupational injuries and illnesses in the United States. U.S. Department of Labor, Bureau of Labor Statistics. Available at: http://www.bls.gov/iif/oshsum.htm. Accessed October 18, 2016.
10. Mathias CGT. The cost of occupational skin disease. Archives of Dermatology 1985; 121: 332–334.
11. Sama SR, Bushley A, Cohen M, et al. Work-related skin disorders in Washington State, 1993–1997. Safety and Health Assessment and Research for Prevention, Washington State Department of Labor and Industries, Report 36-4-1998, 1998.
12. McCall BP, Horwitz IB, Feldman SR, Balkrishnan R. Incidence rates, costs, severity, and work-related factors of occupational dermatitis: A workers' compensation analysis of Oregon, 1990–1997. Archives of Dermatology 2005; 141: 713–718.
13. Jungbauer FH, van der Harst JJ, Groothoff JW, Coenraads PJ. Skin protection in nursing work: Promoting the use of gloves and hand alcohol. Contact Dermatitis 2004; 51: 135–140.
14. Cvetkovski RS, Zachariae R, Jensen H, et al. Prognosis of occupational hand eczema: A follow-up study. Archives of Dermatology 2006; 142: 305–311.
15. Clemmensen KKB, Caroe TK, Thomsen SF, et al. Two-year follow-up survey of patients with allergic contact dermatitis from an occupational cohort: Is the prognosis dependent on the omnipresence of the allergen? British Journal of Dermatology 2014; 170: 1100–1105.
16. Sajjachareonpong P, Cahill J, Keegel T, et al. Persistent post-occupational dermatitis. Contact Dermatitis 2004; 51: 278–283.
17. Meding B, Wrangsjo K, Jarvholm B. Fifteen-year follow-up of hand eczema: Predictive factors. Journal of Investigative Dermatology 2005; 124: 893–897.
18. Lan CC, Feng WW, Lu YW, et al. Hand eczema among University Hospital nursing staff: Identification of high-risk sector and impact on quality of life. Contact Dermatitis 2008; 59: 301–306.
19. Fowler JF, Ghosh A, Sung J, et al. Impact of chronic hand dermatitis on quality of life, work productivity, activity impairment, and medical costs. Journal of the American Academy of Dermatology 2006; 54: 448–457.
20. Cvetkovski RS, Rothman KJ, Olsen J, et al. Relation between diagnoses on severity, sick leave and loss of job among patients with occupational hand eczema. British Journal of Dermatology 2005; 152: 93–98.
21. Kadyk DL, McCarter K, Achen F, Belsito DV. Quality of life in patients with allergic contact dermatitis. Journal of the American Academy of Dermatology 2003; 49: 1037–1048.
22. Slodownik D, Lee A, Nixon R. Irritant contact dermatitis: A review. Australasian Journal of Dermatology 2008; 49: 1–9.
23. Chew AI, Maibach HI. Occupational issues of irritant contact dermatitis. International Archives of Occupational and Environmental Health 2003; 76: 339–346.
24. McMullen E, Gawkrodger DJ. Physical friction is under-recognized as an irritant that can cause or contribute to contact dermatitis. British Journal of Dermatology 2006; 154: 154–156.
25. Morris-Jones R, Robertson SJ, Ross JS, et al. Contact dermatitis and allergy: dermatitis caused by physical irritants. British Journal of Dermatology 2003; 147: 270–275.
26. Mathias CGT. Prevention of occupational contact dermatitis. Journal of the American Academy of Dermatology 1990; 23: 742–748.
27. DeKoven JG, Warshaw EM, Belsito DV, et al. North American Contact Dermatitis Group

patch-test results, 2013–2014. Dermatitis 2016; 28: 33–46.
28. Warshaw EM, Kwon GP, Mathias CGT, et al. Occupationally related contact dermatitis in North American food service workers referred for patch testing, 1994–2010. Dermatitis 2013; 24: 22–26.
29. Warshaw EM, Wang MZ, Mathias CGT, et al. Occupational contact dermatitis in hairdressers/cosmetologists: Retrospective analysis of North American Contact Dermatitis Group data, 1994–2010. Dermatitis 2012; 23: 258–268.
30. Warshaw EM, Hagen SL, Sasseville D, et al. Occupational contact dermatitis in North American mechanics and repairers referred for patch testing: Retrospective analysis of cross-sectional data from the North American Contact Dermatitis Group 1998 to 2014. Dermatitis 2016; 28: 47–57.
31. Coman G, Zinsmeister C, Norris P. Occupational contact dermatitis: workers' compensation patch test results of Portland, Oregon, 2005–2014. Dermatitis 2015; 26: 276–283.
32. Mathias CGT. Contact dermatitis and workers' compensation—Criteria for establishing occupational causation and aggravation. Journal of the American Academy of Dermatology 1989; 20: 842–848.
33. Susitaival P, Flyvholm MA, Meding B, et al. Nordic occupational skin questionnaire (NOSQ-2002): A new tool for surveying occupational skin diseases and exposure. Contact Dermatitis 2003; 49: 70–76.
34. Kucenic MJ, Belsito DV. Occupational allergic contact dermatitis is more prevalent than irritant contact dermatitis: A 5-year study. Journal of the American Academy of Dermatology 2002; 46: 695–699.
35. National Institute of Occupational Safety and Health. Proposed national strategy for the prevention of leading work-related diseases and injuries—dermatological conditions. DHHS (NIOSH) Publication 89-136. Cincinnati, OH: Author, 1988.
36. Foo CC, Goon AT, Leow YH, Goh CL. Adverse skin reactions to personal protective equipment against severe acute respiratory syndrome: A descriptive study in Singapore. Contact Dermatitis 2006; 55: 291–294.
37. Kwon S, Campbell LS, Zirwas MJ. Role of protective gloves in the causation and treatment of occupational irritant contact dermatitis. Journal of the American Academy of Dermatology 2006; 55: 891–896.
38. Weisshaar E, Radulescu M, Bock M, et al. Educational and dermatological aspects of secondary individual prevention in healthcare workers. Contact Dermatitis 2006; 54: 254–260.
39. Loffler H, Bruckner T, Diepgen T, Effendy I. Primary prevention in health care employees: A prospective intervention study with a 3-year training period. Contact Dermatitis 2006; 54: 202–209.
40. Schwanitz HJ, Riehl U, Schlesinger T, et al. Skin care management: Educational aspects. International Archives of Occupational and Environmental Health 2003; 76: 374–381.
41. Holness DL, Arrandale VH, Mathias CGT. Occupational urticarial and allergic contact dermatitis. In: Malo JL, Chan-Yeung M, Bernstein DI (eds.). Asthma in the workplace (4th ed.). Boca Raton, FL: CRC Press, Taylor & Francis, 2013, pp. 418–433.
42. Gimenez-Arnau A, Maurer M, De La Cuadra J, Maibach H. Immediate contact skin reactions, an update of contact urticaria, contact urticarial syndrome and protein contact dermatitis—"A Never Ending Story." European Journal of Dermatology 2010; 20: 552–562.
43. Killig C, Werfel T. Contact reactions to food. Current Allergy and Asthma Reports 2008; 8: 209–214.
44. Amaro C, Goossens A. Immunological occupational contact urticaria and contact dermatitis from proteins: A review. Contact Dermatitis 2008; 58: 67–75.
45. Doutre MS. Occupational contact urticaria and protein contact dermatitis. European Journal of Dermatology 2005; 15: 419–424.
46. Williams JDL, Lee AYL, Matheson MC, et al. Occupational contact urticaria: Australian data. British Journal of Dermatology 2008, 159: 125–131.
47. Usmani N, Wilkinson SM. Allergic skin disease: Investigation of both immediate- and delayed-type hypersensitivity is essential. Clinical and Experimental Allergy 2007; 37: 1541–1546.
48. Reitschel RL, Fowler JF. Contact urticaria. In: Reitschel RL, Fowler JF (eds.). Fisher's contact dermatitis (6th ed.). Hamilton, Ontario: BC Decker, 2008, pp. 615–633.
49. Taylor JS, Leow YH, Fisher AA. Contact urticaria. In Adams RM (ed.). Occupational skin disease (3rd ed.). Philadelphia: W.B. Saunders, 1999, pp. 111–134.
50. Suneja T, Belsito DV. Occupational dermatoses in health care workers evaluated for suspected allergic contact dermatitis. Contact Dermatitis 2008; 58: 285–290.
51. Filon FL, Radman G. Latex allergy: A follow up study of 1040 healthcare workers. Occupational and Environmental Medicine 2006; 63: 121–125.

52. Taylor JS, Erkek E. Latex allergy: Diagnosis and management. Dermatologic Therapy 2004; 17: 289–301.
53. Foti C, Antelmi A, Mistrello G, et al. Occupational contact urticaria and rhinoconjunctivitis from dog's milk in a veterinarian. Contact Dermatitis 2007; 56: 169–171.
54. Bensefa-Colas L, Telle-Lamberton M, Faye S, et al. Occupational contact urticarial: Lessons from the French National Network for Occupational Disease Vigilance and Prevention (RNV3P). British Journal of Dermatology 2015; 173: 1453–1461.
55. Holness L, Mace SR. Results of evaluating health care workers with prick and patch testing. American Journal of Contact Dermatitis 2001; 12: 88–92.
56. Wawrose DJ, Lushniak, BD. Occupational infectious diseases with dermatologic features. In Wright WE (ed.). Occupational and environmental infectious diseases (2nd ed.). Beverly Farms, MA: OEM Press, 2009, pp. 404–421.
57. Gallagher RP, Lee TK. Adverse effects of ultraviolet radiation: A brief review. Progress in Biophysics and Molecular Biology 2006; 92: 119–131.
58. American Cancer Society website. Available at: http://www.cancer.org/cancer/index. Accessed November 21, 2016.
59. Holman CDJ, Gibson IM, Stephenson M, et al. Ultraviolet radiation of human body sites in relation to occupation and outdoor activity: field studies using personal UVR dosimeters. Clinical and Experimental Dermatology 1983; 8: 869–871.
60. Hammond V, Reeder AI, Gray A. Patterns of real-time occupational ultraviolet radiation exposure among a sample of outdoor workers in New Zealand. Public Health 2009; 123: 182–187.
61. Vitasa BC, Taylor HR, Strickland PT, et al. Association of nonmelanoma skin cancer and actinic keratosis with cumulative solar exposure in Maryland watermen. Cancer 1990; 65: 2811–2817.
62. Ramirez CC, Federman DG, Kirsner RS. Skin cancer as an occupational disease: The effect of ultraviolet and other forms of radiation. International Journal of Dermatology 2005; 44: 95–100.
63. Leiter U, Eigentler T, Garbe C. Epidemiology of skin cancer. Advances in Experimental Medicine and Biology 2014; 810: 120–140.
64. Glanz K, Buller DB, Saraiya M. Reducing ultraviolet radiation exposure among outdoor workers: State of the evidence and recommendations. Environmental Health 2007; 6: 22.
65. U.S. Department of Health and Human Services. The Surgeon General's Call to Action to Prevent Skin Cancer. Washington, DC: U.S. Department of Health and Human Services, Office of the Surgeon General, 2014.
66. National Institute for Occupational Safety and Health. Current intelligence bulletin 61: A strategy for assigning new NIOSH skin notations. DHHS (NIOSH) Publication No. 2009–147. Cincinnati, Ohio: NIOSH, 2009.

FURTHER READING

National Institute for Occupational Safety and Health. Occupational dermatoses. Available at: http://www.cdc.gov/niosh/topics/skin/occderm-slides/ocderm.html.
NIOSH offers this occupational dermatoses photolibrary and program for physicians on its website.

Adams RM. Occupational skin disease (3rd ed.). Philadelphia: WB Saunders, 1999.

Honari G, Taylor SJ; Sood A. Occupational skin diseases due to irritants and allergens. In: Goldsmith LA, Katz SI, Gilchrest BA, et al. (eds.). Fitzpatrick's dermatology in general medicine (8th ed). New York: McGraw-Hill, 2012, Chapter 211.

Holness DL, Arrandale VH, Mathias CGT. Occupational urticarial and allergic contact dermatitis. In: Malo JL, Chan-Yeung M, Bernstein DI (eds.). Asthma in the workplace (4th ed.). Boca Raton, FL: CRC Press, Taylor & Francis. 2013, pp. 418–433.

Johansen JD, Frosch PJ, Lepoittevin JP (eds.). Contact dermatitis (5th ed.). Berlin: Springer-Verlag, 2011.

Marks JG, Elsner P, DeLeo VA. Contact and occupational dermatology. St. Louis MO: Mosby, 2002.

Reitchel RL, Fowler JF. Fisher's contact dermatitis (6th ed.). Hamilton, Ontario: BC Decker, 2008.

Rustemeyer T, Elsner P, John SM, Maibach HI (eds.). Kanerva's occupational dermatology. Berlin: Springer-Verlag, 2012.

Wawrose DJ, Lushniak, BD. Occupational infectious diseases with dermatologic features. In Wright WE (ed.). Occupational and environmental infectious diseases (2nd ed.). Beverly Farms, MA: OEM Press, 2009, pp. 404–421.
These eight books and book chapters are excellent references on occupational and environmental skin disorders

Occupational and Environmental Health

Recognizing and Preventing Disease and Injury

Seventh Edition

Edited by
Barry S. Levy
David H. Wegman
Sherry L. Baron
Rosemary K. Sokas

with the assistance of
Heather L. McStowe

OXFORD
UNIVERSITY PRESS

OXFORD
UNIVERSITY PRESS

Oxford University Press is a department of the University of Oxford. It furthers the University's objective of excellence in research, scholarship, and education by publishing worldwide. Oxford is a registered trade mark of Oxford University Press in the UK and certain other countries.

Published in the United States of America by Oxford University Press
198 Madison Avenue, New York, NY 10016, United States of America.

Fourth edition published by 2000
Fifth edition published by 2006
Sixth edition published by 2011
Seventh edition published by 2018

Library of Congress Cataloging-in-Publication Data
Names: Levy, Barry S., editor. | Wegman, David H., editor. | Baron, Sherry, L. editor. |
Sokas, Rosemary K., editor.
Title: Occupational and environmental health : recognizing and preventing disease and injury /
edited by Barry S. Levy, David H. Wegman, Sherry L. Baron, Rosemary K. Sokas, with the assistance of Heather L. McStowe.
Other titles: Occupational health (Levy) Description: Seventh edition. | Oxford ; New York : Oxford University Press, [2018] |
Includes bibliographical references and index.
Identifiers: LCCN 2017012926 (print) | LCCN 2017014701 (ebook) |
ISBN 9780190662684 (updf) | ISBN 9780190662691 (epub) | ISBN 9780190662677 (pbk. : alk. paper)
Subjects: | MESH: Occupational Diseases—prevention & control | Occupational Exposure—prevention & control |
Environmental Exposure—prevention & control | Occupational Health | Environmental Health
Classification: LCC RC963 (ebook) | LCC RC963 (print) | NLM WA 440 |
DDC 616.9/803—dc23
LC record available at https://lccn.loc.gov/2017012926

9 8 7 6 5 4 3 2 1

Printed by Sheridan Books, Inc., United States of America