Periodic synopsis

This report reflects the best data available at the time the report was prepared, but caution should be exercised in interpreting the data; the results of future studies may require alteration of the conclusions or recommendations set forth in this report.

Occupational dermatoses

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This synopsis presents a broad overview of the subject of occupational dermatoses and is drawn principally from the perspectives of clinical diagnosis and primary care prevention. No attempt has been made to provide in-depth coverage of pathogenesis or treatment of these disorders, which are multiple and varied. Only those disorders most likely to be seen by clinical dermatologists have been covered. Readers who desire more in-depth coverage should consult one of the many available textbooks on this subject.

I. General textbook references
   A. Prosser-White R. The dermatergoses or occupational affections of the skin. London: HK Lewis, 1934.
      Contains excellent clinical descriptions of occupational skin diseases, many of which are still relevant today.
   B. Schwartz L, Tulipan L, Birmingham DJ. Occupational diseases of the skin. 3rd ed. Philadelphia: Lea & Febiger, 1957.
      Authored by US Public Health Service dermatologists, this text remains the most comprehensive treatise on this subject. Despite a lapse of 30 years since its last edition, it is surprising how little has changed.
   C. Adams RM. Occupational dermatology. New York: Grune & Stratton, 1983.
      Provides illustrated and succinct coverage of occupational skin diseases and should be required reading for dermatologists who are seriously interested in the subject.
   D. Maibach HI, ed. Occupational and industrial dermatology. 2nd ed. Chicago: Year Book, 1987.
      A collection of reviews that provides in-depth coverage of many current topics in occupational dermatology.
   E. Cronin E. Contact dermatitis. London: Churchill Livingston, 1980.
      A well-written and well-organized text that provided detailed information on contact dermatitis and allergy, much of which is applicable to occupational dermatology.
   F. Fisher AA. Contact dermatitis. 3rd ed. Philadelphia: Lea & Febiger, 1986.
      The most up-to-date comprehensive text on contact dermatitis and a valuable reference for evaluation of work-related contact dermatitis.

II. Background
   A. Health statistics
      1. Occupational skin diseases account for approximately 40% of all occupational illnesses.
      2. The incidence rate for occupational skin diseases was 6.3/10,000 full-time workers in the private sector of the United States in 1984.
      3. About 25% of all patients lose time from work, averaging 10 to 12 lost workdays each.
      4. Between 90% to 95% of all occupational skin diseases are diagnosed as contact dermatitis.

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B. Hazardous occupations and exposures
   1. On the basis of high incidence rates the most hazardous industries for occupational skin diseases are those involving the following goods or services: landscaping and horticulture; forestry; poultry dressing and processing; fresh and frozen packaged fish; beet sugar; surface active agents and penetrants (e.g., emulsifiers, wetting agents, finishing oils); miscellaneous agricultural chemicals; adhesives and sealants; leather tanning and finishing; abrasive products; plating and polishing operations; storage batteries; boat building and repairing; ophthalmic goods; miscellaneous sporting and athletic goods.
   2. The most frequent causal agents include poison oak and ivy; detergents and cleaning agents; solvents; fiberglass and particulate dusts; food products; plastics and resins; petroleum oil, greases, and lubricants; inedible plants or animals; agricultural chemicals; infectious agents; metals and metallic salts; machining oils and coolants; environmental conditions (e.g., heat, humidity); textiles, fabrics, and related materials; rubbish, dirt, and sewage.

C. References
   1. Wang CL. Occupational skin disease continues to plague industry. Monthly Labor Review 1979;121:17-22.
   2. California Department of Industrial Relations, Division of Labor Statistics and Research. Occupational skin disease in California (with special reference to 1977). San Francisco: California Department of Industrial Relations, 1982.
   3. Centers for Disease Control. Leading work-related diseases and injuries—United States. Dermatologic conditions. MMWR 1986;335:561-3.

SPECIFIC OCCUPATIONAL DERMATOSES

Irritant contact dermatitis

I. Clinical features

A. The morphology is mostly nonspecific but typically has eczematous characteristics. There are no reliable features that allow the clinicians to distinguish reliably between irritant and allergic contact dermatitis on the basis of morphology alone. Irritant contact dermatitis of the hands may be indistinguishable in appearance from endogenous dyshidrotic or nummular eczema.

B. Irritation develops predominantly or exclusively on skin surfaces with obvious frequent or repetitive contact with the causal agent. This in turn depends on the physical form of the causal agent and the manner by which exposure occurs.
   1. Solids: irritation involves and remains confined to the skin in direct contact with the solid surface.
   2. Liquids: irritation occurs on skin with direct, obvious contact and rarely involves skin in which exposure is subtle or not obvious.
   3. Airborne particles and mists: irritation may develop on both covered (e.g., particles passing beneath rather than through protective clothing) and uncovered areas of skin and is often accentuated where skin folds or clothing may trap and concentrate the irritant (e.g., the neck, forearms, waist).
   4. Fumes and vapors: irritation usually affects the face, particularly the eyelids; covered skin generally is not affected. Symptoms of ocular or upper airway irritation also are usually present.

II. Common irritants

The most common industrial irritants include detergents, solvents, petroleum oil and grease, machining (cutting) fluids and lubricants, food substances, plants, and fiberglass or other particulate dusts.

III. Predisposing risk factors

The most important risk factors are atopy and preexisting skin disease. Although female sex and fair skin type are suspected, no epidemiologic data exist to support this.

IV. Confirmatory tests

There are none that are reliable. Diagnosis is primarily based on a history of exposure to a known potential irritant that is consistent with the observed clinical appearance and anatomic distribution. Patch testing may be required to exclude a diagnosis of contact allergy.

V. References

A. Dooms-Goosens AE, Debusschere KM, Gevers DM, et al. Contact dermatitis caused by airborne agents. J Am Acad Dermatol 1986;15:1-13.
B. Lachapelle JM. Industrial airborne irritant or allergic contact dermatitis. Contact Dermatitis 1986;14:137-45.
C. Shmunes E. The role of atopy in occupational

skin disease. Occup Med: State of the Art Rev 1986;1:219-28.

Allergic contact dermatitis

I. Clinical features
   A. Morphology is typically eczematous. Although allergic contact dermatitis is not reliably distinguishable from irritant contact dermatitis on the basis of morphologic findings alone, vesicles on skin surfaces other than the palms or fingers, or any unusual patterning (e.g., linear streaking of rhus dermatitis), is suggestive of contact allergy.
   B. The skin surfaces affected by contact allergy depend on the physical form of the allergen and the manner in which exposure occurs (see "Irritant Contact Dermatitis"). Additional involvement of skin surfaces without obvious contact is much more likely to occur with contact allergy than irritation.

II. Common allergens

The most common industrial allergens include *Rhus* and *Compositae* plant species; metallic salts and compounds of nickel, chromate, cobalt, and gold; various accelerators and antioxidants used in rubber; uncured epoxy, acrylic, and phenolformaldehyde resins (including hardeners and curing agents); organic dyes (paraphenylenediamine, photographic color developers, and countless others); biocides and germicides

III. Predisposing risk factors

None are known. Preceding irritant contact dermatitis is a suspected risk factor.

IV. Confirmatory tests

A positive patch test is the gold standard from which this clinical diagnosis is made. The presence of the allergen that produces a positive patch test reaction should be documented in the work environment.

V. Reference

Fisher AA. Contact dermatitis. Philadelphia: Lea & Febiger, 1986.

Photodermatitis

I. Clinical features
   A. Phototoxic dermatitis typically appears as a first- or second-degree sunburn rather than as eczema. Burning and stinging usually are the predominant sensations in its early stages. It involves sun-exposed skin but typically spares shaded areas such as the upper eyelids and submental area of the chin. Hyperpigmentation frequently develops, particularly when the phototoxic reaction has been caused by psoralens.
   B. Photoallergic dermatitis has eczematous morphologic features, and itching is the predominant symptom. It involves the same sun-exposed areas as phototoxic dermatitis, but in severe cases even the shaded areas of the face or skin covered by clothing may be involved.

II. Common phototoxins and photoallergens
   A. Common occupational phototoxins include crude coal tar and its derivatives (e.g., creosote and pitch); psoralens in celery infected by "pink rot" fungus or uninfected grocery store celery.
   B. There are no "common" photoallergens that cause photoallergic reactions more frequently in occupational settings than in the general environment.

II. Predisposing risk factors

Tanning salon use has been implicated in phytophotodermatitis among grocery store workers.

IV. Confirmatory test

A positive photopatch test is required for the diagnosis of photoallergy.

V. References

A. Lim HW, Baer RL, Gange RW. Periodic synopsis. Photodermatoses. J Am Acad Dermatol 1987;17:293-9.
B. Dooms-Goosens AE, Debusschere KM, Gevers DM, et al. Contact dermatitis caused by airborne agents. J Am Acad Dermatol 1986; 15:1-13.
C. Centers for Disease Control. Phototoxic dermatitis in grocery workers. MMWR 1984;34: 11-3.
D. Berkley SF, Hightower AW, Beier RC, et al. Dermatitis in grocery workers associated with high natural concentrations of furanocoumarins in celery. Ann Intern Med 1986;105:351-5.

Toxic vitiligo (chemical leukoderma)

I. Clinical features
   A. The clinical appearance may be indistinguishable from idiopathic vitiligo.
   B. The anatomic distribution frequently resembles that of idiopathic vitiligo. Areas of involvement depend on the route of exposure. Lesions frequently are widespread, involving areas of direct skin contact, and accidental transfer from hands to other body parts. Systemic absorption by accidental ingestion or percutaneous absorption has been postulated to explain

some cases in which lesions occur on skin that has not been obviously exposed.

II. Common causes

These include *para*-substituted phenols and catechols: *p-tert*-butylphenol; *p-tert*-amylphenol; *p*-phenyphenol; *p*-octylphenol; *p*-nonylphenol; *p*-cresol; *p*-methylcatechol; *p*-isopropylcatechol; hydroquinone; and the monomethyl, monoethyl, and monobenzyl ethers of hydroquinone. Other miscellaneous chemical depigmenting agents include triethylenethiophosphoramide (thio-TEPA); mercaptoamines such as β-mercaptoethylamine and N-(2-mercaptoethyl)-dimethylamine; diisopropyl fluorophosphate, and physostigmine.

III. Predisposing risk factors

Persons with idiopathic vitiligo may be more susceptible to chemical depigmentation.*

IV. Confirmatory tests

There are none that are reliable. Lesions are histopathologically indistinguishable from idiopathic vitiligo. Persons with concomitant allergic contact dermatitis may show depigmentation at the patch test site to the causal agents. Epidemiologic investigation of multiple cases may be necessary to establish a probable cause-and-effect relationship. Definitive conclusions may be impossible if only a single case has occurred.

V. References

A. Fisher AA. Contact dermatitis. Philadelphia: Lea & Febiger, 1986:675-85.

B. Gellin GA. Pigment responses: Occupational disorders of pigmentation. In: Maibach HI, ed. Occupational and industrial medicine. 2nd ed. Chicago: Year Book, 1987:134-41.

Oil acne and chloracne

I. Clinical features

A. Oil acne is characterized by multiple open comedones and sterile, inflammatory papules and pustules that occur on skin surfaces exposed to the causal agent. Lesions tend to be more severe on covered areas where clothing has been saturated with oil. Open comedones (blackheads) are the predominant (and occasionally the only) lesions when the disease is caused by heavy lubricating petroleum oils.

B. Chloracne is predominantly characterized by closed comedones and small cysts; inflammatory lesions occur but are far fewer in number. The malar crescent of the cheeks and the retroauricular folds are the most frequently involved sites, followed by the cheeks, forehead, and neck, but the nose usually is spared. The genitalia are also sensitive areas. Lesions occur on the back, shoulders, chest, abdomen, and buttocks as toxicity increases. The hands, feet, forearms, and thighs are usually involved only in severe cases.

II. Common causes

A. Oil acne is caused most frequently by heavy lubricating petroleum oils and greases (e.g., those used in automotive and heavy equipment repair); cutting fluid emulsions (machinists); miscellaneous oils (e.g., animal or vegetable oils).

B. Because of tight control of industrial processes, true chloracne is a relatively uncommon occurrence today except for industrial accidents. Chemical causes include polyhalogenated biphenyls (e.g., polychlorinated biphenyl [PCB]); polyhalogenated dibenzofurans; polyhalogenated naphthalenes; trichlorophenol and pentachlorophenol that are contaminated with dioxin; dichloroaniline and related derivatives that are contaminated with tetrachloroazoxybenzene or tetrachloroazobenzene.

III. Predisposing risk factors

Poor personal and environmental hygiene are the most important factors. It has not been established whether a history of teenage acne predisposes adults to chloracne.

IV. Confirmatory tests

There are none that are reliable. The value of a skin biopsy is disputed. Blood levels of chloracnegens do not correlate with clinical chloracne.

V. References

A. Taylor JS. Environmental chloracne. Update and overview. Ann NY Acad Sci 1979; 320:295-307.

B. Crow KD. Chloracne. A critical review including a comparison of two series of cases of acne from chloronaphthalene and pitch fumes. Trans St John's Hosp Dermatol Soc 1970;56:79-99.

C. Suskind RR, Hartzberg VS. Human health effects of 2,4,5-T and its toxic contaminants. JAMA 1984;251:2372-80.

D. Tindall JP. Chloracne and chloracnegens. J Am Acad Dermatol 1985;13:539-58.

E. Crow KD, Puhvel M. Chloracne (halogen acne). In: Marzulli FN, Maibach HI. Derma-

*Nordlund, James, M.D., Cincinnati, OH. Personal communication, 1986.

totoxicology. 3rd ed. Washington, DC: Hemisphere: 1987;515-34.

Systemic urticaria, contact urticaria, and flushing

I. Clinical features
   A. Occupationally induced systemic urticaria is characterized by wheals that are usually accompanied by other symptoms of inhalant allergy (conjunctivitis, rhinitis, asthma), because the principal route of exposure usually is airborne.
   B. Contact urticaria is characterized by wheals that occur at sites of primary skin contact. Because of percutaneous absorption, lesions also may occur at distant sites if the mechanism involves immediate hypersensitivity.
   C. Flushing reactions are characterized by transient erythema that usually involves the face and neck, and occasionally the chest; wheals do not occur.

II. Common causes
   A. Occupational systemic urticaria is most often caused by "house dusts" (e.g., in janitors and warehouse workers) or particulates of molds, grasses, and pollens (e.g., in outdoor workers); castor bean pomace; coffee bean dust; platinum salts. Reports linking systemic urticaria to chemical fumes and vapors (e.g., formaldehyde, ammonia, sulfur dioxide) are mostly anecdotal.
   B. Contact urticaria is most frequently caused by foods containing allergens and vasoactive substances, including dairy products, fish, citrus fruits, nuts, spices, grains, meats, and vegetables (e.g., in food handlers); animal hair and tissues (e.g., in veterinarians and slaughter house workers); plants and grasses (e.g., gardeners); medicinal rubefacients and allergenic antibiotics (e.g., in physicians, nurses, veterinarians).
   C. Flushing reactions usually are attributable to alcohol ingestion and concomitant occupational exposure to disulfiram (e.g. in rubber workers); trichloroethylene (e.g., in degreasers); dimethylformamide and butyraldoxime. Flushing reactions attributed to visual display terminal work remain a subject of controversy.

III. Predisposing risk factors

An atopic predisposition (i.e., a tendency to develop multiple IgE mediated allergies) is the most important risk factor for contact or systemic urticaria caused by immediate hypersensitivity.

IV. Confirmatory tests

These diagnoses require confirmation by open or closed patch tests with immediate readings; prick tests; radioallergosorbent test for urticaria because of immediate hypersensitivity reactions; provocation tests for systemic nonimmunologic urticaria and flushing reactions when necessary.

V. References
   A. Key MM. Some unusual allergic reactions in industry. Arch Dermatol 1961;83:3-6.
   B. Burdick AE, Mathias CGT. The contact urticaria syndrome. Dermatol Clin 1985;3: 71-84.
   C. Mooney E. The flushing patient. Int J Dermatol 1985;24:549-54.
   D. Berg M, Liden S. Skin problems in video display terminal users. J AM ACAD DERMATOL 1987;17:682-4.

Neoplasms

Some cases of squamous cell carcinoma, basal cell carcinoma, and malignant melanoma may have an occupational cause.

I. Clinical features

Occurrence of squamous or basal cell carcinoma on skin that is not chronically exposed to sunlight should raise suspicion of work-related chemical carcinogen exposure.

II. Common occupational causes
   A. Squamous cell carcinoma may be caused by occupational exposure to ultraviolet radiation (e.g., in outdoor work), ionizing radiation (e.g., in medicine and dentistry), crude coal tar, pitch, and unrefined derivatives (e.g., in roofing, road construction, and petroleum mining and refining), arsenic (e.g., in smelting and manufacture or application of arsenic-containing wood preservatives and pesticides).
   B. Basal cell carcinoma may be caused by occupational exposure to ultraviolet radiation and arsenic.
   C. Malignant melanoma may be caused by occupational exposure to ultraviolet radiation. Chemical carcinogens have been implicated by some epidemiologic studies but are not firmly established as causes.

III. Predisposing risk factors

The most important include fair skin and increased susceptibility to sunburn; and Irish-Celtic-Scottish ancestry (ultraviolet radiation–induced skin cancers).

IV. Confirmatory tests

No clinical or histopathologic features indisputably establish an occupational cause. Epidemiologic investigation of multiple cases may be required. Definite conclusions may be impossible if only a single case has occurred.

V. References

A. Vickers DHF. Industrial carcinogenesis. Br J Dermatol 1981;105 (Suppl 21):57-61.

B. Vitaliano PP, Urbach F. The relative importance of risk factors in nonmelanoma carcinoma. Arch Dermatol 1980;116:454-6.

C. Lee JAH. Melanoma and exposure to sunlight. Epidemiol Rev 1982;4:110-36.

Infections and infestations

I. Clinical features

The cutaneous manifestations depend on nature of the infectious agent.

II. Common causes

These include anthrax (in wool handlers, dock and freight workers handling imports); erysipeloid (in fishermen, butchers, meat handlers); *Mycobacterium marinum* infections (in fishermen, aquarium or swimming pool workers); herpes simplex (nurses, physicians, dentists); or (in farmers and ranchers); milker's nodule (in dairy farmers); sporotrichosis (in gardeners, nursery workers); paronychia (in bartenders, beauticians, janitors, other "wet" work); poultry mite (in poultry farmers and processors) and grain mite (in grain farmers) infestations.

III. Predisposing risk factors

Cutaneous trauma or irritation may predispose persons to skin infection. Atopic persons are more prone to bacterial skin infections.

IV. Confirmatory tests

The causal organism should be appropriately demonstrated (culture or smear) in both patient and source.

V. Reference

Adams RM. Occupational dermatology. New York: Grune & Stratton, 1983:42-54.

Connective tissue disorders

I. Clinical features

A. Traumatic vasospastic disease ("vibration white finger") is characterized by paroxysmal vasospasm of the hands. It may be unilateral but is otherwise clinically indistinguishable from Raynaud's phenomenon. It is not associated with an underlying systemic connective tissue disorder. In mild cases symptoms are usually provoked only by work with vibratory tools, but cold may precipitate attacks in more severe cases.

B. Acroosteolysis is characterized by lytic lesions of the distal and middle phalanges and is associated with Raynaud's phenomenon, sclerodactyly, papular cutaneous sclerosis, and fibrosis of the lungs, liver, and spleen. Angiosarcoma of the liver has been reported.

C. Occupational scleroderma is clinically indistinguishable from idiopathic scleroderma.

II. Common causes

A. Traumatic vasospastic disease may be caused by work that involves the use of vibratory tools such as chain saws, pneumatic hammers, and chipping tools.

B. Acroosteolysis has been caused only by occupational exposure to vinyl chloride monomer (manufacture of polyvinylchloride plastics)

C. Scleroderma may be caused by occupational exposure to silica (mining operations); epoxy hardener (bis-4-amino-3-cyclohexyl-methane); chlorinated organic solvents as trichloroethylene and perchloroethylene (clinical evidence is mostly circumstantial, but case reports are increasing).

III. Predisposing risk factors

There are no known predisposing risk factors for any of these disorders, but smoking and cold exposure are suspected to contribute to the degree of injury in traumatic vasospastic disease.

IV. Confirmatory tests

Ice water immersion may provide observable vasospasm in some cases of traumatic vasospastic disease, but there are no reliable objective tests for mild disease. X-ray films of the hands that show lytic lesions of the distal or middle phalanges are highly suggestive of acroosteolysis. No tests indisputably establish an occupational cause for scleroderma. Epidemiogic investigation of multiple cases may be necessary to establish an occupational cause. Definite conclusions may be impossible when only one case has occurred.

V. References

A. Taylor W, Brammer AJ. Vibration effects on the hand and arm in industry. An introduction and review. In: Brammer AJ, Taylor W. Vibration effects on the hand and arm in industry. New York: John Wiley, 1982.

B. Markowitz SS, McDonald CJ, Fethure W, et al. Occupational acroosteolysis. Arch Dermatol 1972;106:219-33.

C. Haustein UF, Ziegler V. Environmentally induced systemic sclerosis-like disorders. Int J Dermatol 1985;24:147-51.

Climatic disorders

I. Clinical features

A. Miliaria rubra (prickly heat) is characterized by inflammatory papules on covered parts of the body (usually the trunk), which are provoked by work in hot, humid environments. An admixture of burning, stinging, or itching sensations accompany the lesions.

B. Asteatotic eczema (winter eczema) may be provoked by a combination of low humidity and low temperature, leading initially to severe dryness and chapping of the skin, followed later by dermatitis. Similar climatic conditions may be mimicked by refrigerated air conditioning systems in the summertime.

C. Low humidity dermatosis is provoked by a combination of water temperature and relatively low humidity. Cutaneous changes occur most frequently on the face or neck, but they are nonspecific; they include slight dryness of the skin, intense pruritus, and, sometimes, mild dermatitis.

II. Common causes

Climatic conditions that may lead to the development of these disorders include temperatures exceeding 80 to 85° F and relative humidity more than 80% (miliaria); cold temperatures below 32° F (winter eczema); indoor relative humidity below 35% to 45% (low humidity dermatoses).

III. Predisposing risk factors

No predisposing factors for these disorders have been identified.

IV. Confirmatory tests

Temperature and humidity measurements are required.

VI. Reference

Rycroft RJG. Low humidity dermatoses. Dermatol Clin 1984;2:553-9.

PREVENTION

Engineering

I. Process engineering

Skin exposure should be prevented by isolation, containment, or enclosure of the process from which exposure to the causal agent is occurring. Ventilation control usually is not helpful except when exposure occurs primarily from fumes, vapors, or fine, airborne dusts or mists.

II. Chemical substitution

Replacement or elimination of allergens or other noxious substances may sometimes be a feasible alternative to process engineering.

III. Reference

Adams RM. Allergen replacement in industry. Cutis 1977;20:511-6.

Personal protection

I. Protective clothing

Glove protection usually is effective for irritants, but gloves must have appropriate chemical resistance, physical resistance, and flexibility for the job task. Allergens may penetrate through various glove materials to provoke dermatitis. Disposable gloves that can be changed frequently may be the only feasible option for allergic persons. Dermatitis may be caused or aggravated by protective clothing as a result of:

A. Nonspecific irritation from sweat entrapment and friction of the clothing against the skin

B. Accidental entrapment and occlusion of chemical substances against the skin

C. Development of contact allergy to protective clothing (e.g., accelerators or antioxidants in rubber gloves)

II. Barrier creams

With the exception of sunscreens to prevent exposure to ultraviolet radiation, the clinical effectiveness of such preparations is controversial and unsupported by controlled clinical trials. Barrier creams may facilitate personal hygiene efforts by making it easier to wash oils and greases off the skin.

III. References

A. Moursiden HT, Faber O. Penetration of protective gloves by allergens and irritants. Trans St John's Hosp Dermatol Soc 1973;59:230-4.

B. Orchard S. Barrier creams. Dermatol Clin 1984;2:619-29.

Hygiene

I. Environmental

The worker's environment should be clean so that contamination of work surfaces is not an important route of exposure.

II. Personal

Irritants, allergens, or other noxious agents should be removed from the skin as soon as possible after contact occurs. Overuse or improper use of skin cleaning agents may cause or aggravate work-related dermatitis.

A. Abrasive cleaners work by "stripping" off

superficial layers of stratum corneum. Use should be restricted to thick palmar skin.

B. Waterless cleaners contain solvents that dissolve oily substances which soil the skin. Use should be followed by gentle washing and rinsing with mild hand soap to remove any residual film of waterless cleaner.

C. The use of a skin moisturizer may help to retard any irritating effects of harsh workplace cleaners or frequent handwashing.

III. Reference

Mathias CGT. Contact dermatitis from use or misuse of soaps, detergents, and cleaners in the workplace. Occup Med: State of Art Rev 1986;1:205-18.

Preemployment screening

I. Atopy

Although the presence of atopic traits increases risk, the actual likelihood of the development of occupational contact dermatitis in a person with atopic traits, but without active atopic dermatitis, is unknown.

II. Active skin disease

Persons with active skin disease should be identified at the time of hire and placed in job assignments without risk of secondary aggravation.

III. Reference

Adams RM. High risk dermatoses. J Occup Med 1981;23:829-34.

WORKERS' COMPENSATION, IMPAIRMENT, AND DISABILITY

The clinical dermatologist's responsibility

The fundamental role of the clinical dermatologist is to evaluate the medical impairment of the injured worker with regard to:

I. Was skin disease caused by an occupational exposure?

II. To what extent does the injured worker require continued medical treatment as a result of skin disease?

III. To what extent is the injured worker precluded on the basis of skin disease from returning to a usual and customary job or to any other job?

The workers' compensation bureau's responsibility

The fundamental role of a workers' compensation bureau is to evaluate the overall disability of the injured worker with regard to:

I. Medical impairment as determined by the dermatologist's evaluation

II. Ability to be retrained or gainfully employed in other jobs on the basis of socioeconomic considerations (e.g., age, education, skills)

Reference

American Medical Association. Guides to the evaluation of permanent impairment. 2nd ed. Chicago: American Medical Association, 1984.