



# Occupational and Work-Related Dermatitis: Definition and Classification

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## Definition

There are various definitions for occupational dermatosis. A concise and clear one was stated by a co-operative approach to industrial dermatologic problems undertaken in 1936 between the American Medical Association and the American Dermatological Association: an occupational dermatosis is “a pathological condition of the skin for which occupational exposure can be shown to be a major causal or contributory factor” [1]. Other definitions include the possible alterations in mucosa and annexes [2] or the need that the occupational disease affects certain groups of individuals to a significantly greater extent (at least with a twofold relative risk) than the general population [3].

But not always an occupational skin disease from a medical point of view is considered as such from a juristic perspective, with consequences in social and economic compensation for the patient. The legislation about what is an occupational dermatosis varies greatly from one country to another, and of course it also varies the compensation. The legal requisites to consider a skin disease as occupational depend finally on

political decisions. Thus, we can conclude that, from a legal point of view, an occupational skin disease is that one recognized in the legislation of the country where the patient is working.

The distinction between work-related diseases and occupational diseases is not always clear and stands a matter of discussion. Occupational diseases are considered as having a specific or a strong relation to occupation, generally with only one causal agent, and recognized as such. Nevertheless, work-related diseases have a complex etiology, with multiple causal agents, where factors in the work environment may play a role in the development of such diseases, together with other risk factors [4].

Work-related dermatoses are then defined as those diseases that have multiple causes, including factors of the work environment [5]. This concept entails a theoretical and legislative discussion that is not always clear and varies from one country to another. In this chapter we will refer to all these conditions as occupational dermatoses, but conceptually some of the entities discussed could be considered as work-related dermatoses.

The recognition of a disease as occupational is of such importance for the worker and other sectors of society (company, health insurance, and compensation systems) that it can be helpful following some criteria to define the disease in the most objective way possible. In view that contact dermatitis accounts for more than 90% of all

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worker's compensation claims for occupational skin diseases, Mathias established seven criteria to assess the probability of a causal relationship with employment of a contact dermatitis. He concluded that if the answer to at least four criteria should be "yes," the contact dermatitis is probably caused by a workplace exposure [6]:

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 non-occupational exposures excluded as probable causes?
6. Does dermatitis improve away from work exposure to the suspected irritant or allergen?
7. Do patch or provocation tests implicate a specific workplace exposure?

These criteria should be used with caution, as a guide, knowing that sometimes they could give false positives and negatives results. The core message is that the definition of an occupational skin disease resides on a detailed history, a clear temporal relationship of the onset of the skin disease and the job, relevant positive patch tests in the case of allergic contact dermatitis, and usually confirmation of the resolution when the causative agent is avoided.

Besides the causes of occupational dermatoses, we must be aware of some predisposing factors. Age is one of them: occupational dermatosis is more frequent in young individuals, due to work inexperience, poor protective measures, and more contact with irritants. Other factors are pre-existing dermatosis (such as atopic dermatitis and psoriasis), cold and hot temperature, low and high humidity, and difficulty accessing to hygiene and medical services.

## Classification

There does not exist a universal accepted classification for occupational dermatosis. The most common classification is based on the external factors that cause these diseases (mechanical, physical, biologic, and chemical) [7–9]. However, this classification is sometimes confusing, because separating chemical from physical or mechanical causes is not always obvious.

A better classification of occupational skin diseases would be by professions (dermatosis in construction workers, metalworkers, hairdressers, etc.). But the great variety of professions is such that it would not be a good schematic organization. Furthermore, it would not be entirely correct, since within the different professions there are various jobs or workplaces, with their specific risks. In fact, in some professions each workplace has its own special problems.

Although the true frequencies of occupational dermatosis are unknown, the great majority of them correspond to contact dermatitis to different chemicals that are present at workplace (70–90% of all occupational dermatosis, depending on different studies [10–12]). The resting 10–30% of occupational dermatosis are a heterogeneous group of different entities (acne, urticaria, infections, etc.). Besides being contact dermatitis so frequent in occupational dermatology, allergic contact dermatitis is the main cause of work inability, with the very important personal, social, and economic consequences that it produces.

Taking these arguments together we have preferred a division of occupational dermatosis in two groups: contact dermatitis and non-contact dermatitis. Table 1.1 shows this classification, although not exhaustive, but containing the main entities in occupational dermatology.

**Table 1.1** Classification of occupational dermatosis

1. Contact dermatitis	
Allergic contact dermatitis	Multiple allergens. Clinical patterns: localized eczema, airborne, lichenoid, lymphomatoid, photocontact allergic dermatitis...
Irritant contact dermatitis	Acute; acute delayed; cumulative; traumatic; pustular; phototoxicity...
2. Non-contact dermatitis	
Urticaria	Immunological contact urticaria; no immunological contact urticaria; protein contact dermatitis.
Acne	Oil acne; coal tar acne; acne mechanica; chloracne; cosmetic acne; other forms of acne.
Pressure- and friction-induced disorders	Callus; blisters; fissures; hemorrhages; abrasions; nodules; Koebner isomorphic phenomenon (psoriasis, lichen planus, vitiligo, eczema...).
Vibration-associated disorders	Raynaud's phenomenon (traumatic vasospastic disease or hand-arm vibration syndrome).
Foreign body granulomas	Cobalt; beryllium; aluminum; zirconium; hair; others.
Pigmentary disorders	Post-inflammatory hyper- and hypopigmentation; hyperpigmentation from tattoos and chemicals; chemical leukoderma.
Low humidity-induced disorders	Pruritus; urticaria; eczema; semicircular lipoatrophy.
Heat-associated disorders	Miliaria; intertrigo; hyperhidrosis; erythema ab igne; thermal burns.
Cold-associated disorders	Frostbites; perniosis; acrocyanosis; cold panniculitis; Raynaud's phenomenon.
Skin cancer	Arsenic; polycyclic aromatic hydrocarbons (coal tar); ultraviolet light; ionizing radiation.
Infections and infestations	Virus (warts, herpes simplex, orf, milker's nodules...); bacteria (staphylococci, streptococci, anthrax, brucellosis, erysipeloid, tularemia, leptospirosis, mycobacteria, tick-borne diseases...); fungi (tinea pedis, candida infections, sporotrichosis...); scabies, etc.
Others	Radiodermatitis (acute and chronic); scleroderma-like disease (vinyl chloride, organic solvents); nail disorders (paronychia, dyschromia, onycholysis...); self-induced dermatosis (dermatitis artefacta); others.

## Occupational Contact Dermatitis

Contact dermatitis is the main problem in occupational dermatology. Although it will be treated in extension in other chapters, its key aspects will be highlighted below.

Contact dermatitis is a skin reaction (normally eczematous) caused by a chemical insult to the skin. There are numerous substances capable of producing a contact dermatitis in different jobs. It is subdivided into irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). In ACD there exists a delayed hypersensitivity reaction (type IV hypersensitivity), which is not present in ICD.

The incidence of occupational contact dermatitis is determined by the degree of socioeconomic

and industrial development in an area, resulting in a lot of geographical variation [11–15]. In general, ICD is responsible for at least of 80% of occupational contact dermatitis, while the remainder are ACD. This ratio varies among occupations, but ICD is always much more frequent than the allergic one. Anyway, ICD is not always declared, because normally it tends to present as mild dermatitis and resolves without specific treatment.

Most of the occupational contact dermatitis occur on the hands, due to the manipulation of many substances (Fig. 1.1). Other localizations may exist, depending on the contact with the offending substance. For example, when an airborne mechanism exists, dermatitis tends to affect the face, neck, and other areas exposed to the air (Fig. 1.2).



**Fig. 1.1** Allergic contact dermatitis to epoxy resin in a floor layer worker



**Fig. 1.2** Airborne allergic contact dermatitis to an intermediate compound in the synthesis of omeprazole in a pharmaceutical industry worker

It is very frequent to encounter an overlap between irritant and sensitizing reactions. For example, cement can cause an ICD due to the corrosive action of cement and/or an ACD from chromates. Soaps cause ICD due to surfactants and its capacity to modify the pH of the skin, but can also generate ACD from preservatives and fragrances.

A careful history and exploration are needed to achieve a correct diagnosis of contact dermatitis. Some symptoms and signs point toward one or another. For example, erythema, burning, and stinging immediately after contact with a substance suggest an irritant reaction whereas intense pruritus may support ACD [16].

In the study of these patients, we must know the irritant and allergic potential of the products managed at work, as well as the way they handle them. It is crucial to investigate the temporal relationship of the dermatitis with work periods, and the hobbies or other products that are in contact outside job. In general, occupational ACD tends to worsen more rapidly than ICD on return to work. The recovery period in ACD is normally longer (2–4 weeks) than in ICD (1–3 days in most of cases).

ACD generally presents as eczema, although rare variants exist (lichenoid, lymphomatoid, etc.). In contrast, ICD is more polymorphous. Some variants of ICD are acute ICD, acute delayed ICD, cumulative ICD, traumatic ICD, or pustular/acneiform ICD. As an example of this variability, fiberglass dermatitis is a special form of ICD that normally presents with a papular eruption that is very pruritic (Fig. 1.3). This pruritus associated with fiberglass exposure is typically sudden and intense [7]. Fiberglass is used in many industrial applications and workers often complain of pruritus on the neck, antecubital and popliteal fossae, forearms, wrists, and frictional sites, such as the belt and sock lines.

Patch tests are needed to diagnose contact dermatitis. They are positive in ACD and negative in ICD. It is essential to correctly interpret these patch test reactions, to differentiate between irritant and allergic reactions, and to search the relevance of each positive response.

Photocontact dermatitis is a variant of contact dermatitis where a substance needs ultraviolet



**Fig. 1.3** Fiberglass irritant contact dermatitis in a worker who manufactured insulating panels without adequate protection

(UV) light to produce a contact dermatitis. This can be an irritant reaction (phototoxicity, for example in the association of furocoumarins and UV light) or it can be an allergic reaction (photocontact allergic reaction, as can occur in the association of topical non-steroidal anti-inflammatory drugs and UV light).

Phototoxic dermatitis typically appears as a first- or second-degree sunburn, rather than as eczema. Hyperpigmentation frequently develops. In contrast, photoallergic dermatitis has eczematous morphologic features [17]. The diagnosis of allergic photocontact reaction can be made with proper photopatch tests.

Other entities can be present with or simulate contact dermatitis. Hence the importance of knowing endogenous eczematous diseases (atopic dermatitis, dyshidrotic eczema, seborrheic dermatitis...), as well as psoriasis, lichen planus, mycosis fungoides and in general the spectrum of skin diseases. A diagnostic of occupational contact dermatitis can open a litigating period with even judicial implications; so all efforts must be done in making a correct diagnosis.

### Occupational Non-contact Dermatitis

A heterogenous group of entities are responsible for occupational non-contact dermatitis (Table 1.1). These problems are found less frequently than contact dermatitis in the occupational setting. We present below the main pictures.

### Urticaria

Occupational contact urticaria can be immunological (IgE-mediated) or non-immunological. The clinical picture is that of wheals/redness/pruritus that appears suddenly, usually within 30 min after contact with the causative agent. It clears completely within hours and can be caused by a multitude of substances, from low molecular weight chemicals to proteins [18, 19].

Occupations at risk of developing contact urticaria are specially those who manage protein sources, as fruits, vegetables, spices, plants, animal tissues, grains, and enzymes [18–20]. Natural rubber latex emerged in the 1980s as a major cause of occupational contact urticaria, but fortunately the incidence has declined with different efforts, mainly the use of latex-free gloves and low-protein rubber gloves [21, 22].

Protein contact dermatitis is a special type of initially contact urticaria involved in the contact urticaria syndrome that appears on previously damaged skin with eczema. On this dermatitis areas, wheals or even a vesicular exacerbation can be noted a few minutes after contact with the causal protein [18, 23].

An atopic predisposition is the most important risk factor for contact or systemic urticaria caused by immediate hypersensitivity. The main complementary tool for the diagnosis is the prick test, although other diagnostic tests can be useful (measurement of specific IgE, basophil activation test, rubbing test, etc.).

### Acne

Occupational acne can be seen in different workers, but especially those who are in contact with greasy and oily products. It may affect any follicle of the body, and not just the face or the typical localizations of acne vulgaris. People with predisposition to acne are at risk of developing occupational acne, but it also affects those without this predisposition. It has been described different types of occupational acne. The most important forms are oil acne, coal tar acne, acne mechanica, and chloracne.

Oil acne is the most common form of occupational acne, and it is caused mainly by greases and insoluble oils, that are comedogenic. Its incidence has declined in the past decades, due to the

decreased use of pure cutting fluids and the improved hygienic conditions. Machine tool operators, mechanics, roofers, petroleum refiners, rubber workers, textile mill workers, and road pavers are workers at risk of developing oil acne [24].

Oil acne is more common in areas where contact with oil takes place, such as dorsa of the hands and fingers, arms, forearms, and abdomen, presenting as follicular papules, pustules, and sometimes nodules (Fig. 1.4). Other localizations are possible, according to the contact with the oil and the occlusion. For example, acne on buttocks when the worker is seated on a contaminated seat or on thighs due to hand transportation. Avoiding oil acne includes good hygiene measures, isolation of the machinery, and frequent work clothes changes.

Certain coal tars are also responsible for occupational acne, due to obstruction of the sebaceous glands by the mixture, forming black plugs. Coal tar is used in many industries: in aluminum pro-

duction, steel and iron foundries, tar refineries, road paving, roof insulation, pavement sealcoat, and wood surfaces painting. These workers are exposed mainly through inhalation and dermal contact. Lesions in coal tar acne tend to affect the face, suggesting an airborne distribution [25].

Acne mechanica refers to a form of acne vulgaris aggravated by different traumas to the skin. This is sometimes the case of an occupational factor. The stressors provoking acne mechanica are diverse, as protracted pressure from tight clothing, helmets, packs, seats, resting the head in the hands in a particular way, friction, tension, or rubbing [26].

A type of acne mechanica is the one caused by the prolonged use of face masks, a current topic during the COVID-19 pandemic (“maskne”). This acneiform eruption is associated with prolonged wear and occlusion of masks, especially in healthcare workers. It must be differentiated from irritant contact dermatitis, which is another common issue due to masks, affecting the cheeks and nasal bridge due to pressure and friction, especially in association with personal history of atopic dermatitis. Masks are also responsible for allergic contact dermatitis to the elastic straps, glue, nickel [27], and formaldehyde released from the mask [28]. In summary, occupational acne due to masks must be differentiated from contact dermatitis and worsening of other processes, such as seborrheic dermatitis and rosacea [29].

Chloracne is a distinctive and often severe form of occupational acne caused by exposure to various chlorinated compounds, which may also be toxic to other organs, such as the liver and the neurologic system. Nowadays is rare, but still possible to encounter in some workers during chemical manufacturing. In the past chloracne was sometimes related to accidents, as the explosion of a reactor involved in trichlorophenol synthesis in Seveso (Italy) in 1969 [24, 30].

Chloracne may be caused both by contact of the skin with the implicated substance and by ingestion and/or inhalation of fumes. Malar region and behind the ears are the most common sites of chloracne. Lesions tend to be monomorphic, with multiple closed comedones and micro-



**Fig. 1.4** Oil acne in a metalworker of a small workshop. An outbreak was observed in several workers due to poor hygiene and the lack of preventing measures against cutting fluids

cysts. Inflammatory lesions can occur but are less frequent. Associated with acne lesions, xerosis, hypertrichosis, melanosis, and frequent itching can be seen in chloracne [24, 30]. A special attention should be paid to possible involvement of internal organs if chloracne is suspected.

Other forms of occupational acne may be seen. Cosmetic acne presents in actors, actresses, and cosmetologists who use oily cosmetics for prolonged periods of time, causing mainly comedonal lesions on the face. Occupational acne may also appear in pharmaceutical synthesis of steroids and other medications. Sometimes acne also affects kitchen workers exposed to grease [24]. Finally, Favre-Racouchot disease presents with comedones and epidermal cysts mainly in periorbital regions. It is due to UV-light damage and can be considered also a form of occupational acne in sun-exposed workers.

### Pressure- and Friction-Induced Disorders

Many jobs lead to localized pressure and friction, with the subsequent formation of callus, blisters, fissures, or hemorrhages. Normally this is solved with an appropriate protection and reducing repetitive tasks.

Athletes have dermatoses due to trauma, mainly on feet. Repeated trauma can cause erythema, edema, and separation of toenail (“jogger’s toe”), hyperkeratotic patches on heel from running, splinter hemorrhages of toenails (“tennis toe”), punctate hemorrhages on heels (“black heel” in sports as basketball), blisters from friction, shin abrasions and erosions (“skier’s shins”), or subcutaneous masses (“surfer’s nodules”) [7].

During the COVID-19 pandemic it has been observed an increased number of cases of erythema and desquamation in areas of more contact with masks and goggles, due to maintained pressure and friction of these protective equipment [29, 31].

The Koebner (isomorphic) phenomenon from occupational trauma in workers with psoriasis is very common. In fact, psoriasis on sites of trauma or friction can be the first appearance of psoriasis in a previously unaffected individual. Work-related psoriasis lesions appear very frequently



**Fig. 1.5** Koebner phenomenon in an informatic with mild psoriasis elsewhere. Psoriasis was intense in areas of friction with the computer mouse

on skin of palmar surfaces where the worker applies a repetitive pressure (Fig. 1.5). Many dermatologists may overlook the cause of such lesions, and treatments tend to fail if a Koebner phenomenon is not suspected. Preventive measures for such cases include use of padded gloves and alternative methods to realize the repetitive task that is responsible for the isomorphic phenomenon. Other dermatologic diseases, such as lichen planus, vitiligo, or even eczema (post-traumatic eczema [32]), can manifest as isomorphic reactions from work-related traumas, although less frequently than psoriasis.

### Vibration-Associated Disorders

A picture similar to Raynaud’s phenomenon can occur in workers who manage vibrating tools, like chain saws, pneumatic hammers, and chipping tools. This entity is also known as traumatic vasospastic disease, vibration white finger, or the hand-arm vibration syndrome [17, 33]. It is characterized by paroxysmal vasospasm of the hands, with blanching accompanied by numbness and reduced sensitivity, particularly in winter. Attacks may last 1–60 min and generally resolve rewarming the hands, accompanied by hyperemia and pain. Continued vibration exposure, the attacks become more frequent, also more prolonged and in all seasons. Ultimately, ulcers can occur. Besides this vasospastic symptoms, neurological symptoms can exist (sensory and motor changes).

There is a controversy about possible carpal tunnel syndrome, bone and joint pathology associated with this syndrome [33].

The hand-arm vibration syndrome may be unilateral but is otherwise clinically indistinguishable from Raynaud's phenomenon. It is not associated with an underlying systemic connective tissue disorder [17]. There exists a latent period between the onset of exposure to vibration and the beginning of symptoms. This period is variable and is dependent upon factors, including tool vibration frequencies, total vibration exposure, hand grip strength, climatic conditions, individual susceptibility, and smoking habit [33]. Although this syndrome appears more frequently in workers managing vibrating tools in cold environments, it also exists in temperate climates [34].

### Foreign Body Granulomas

The introduction through the skin of substances or products used at work can generate nodules with occasional inflammatory reaction and fistulation. Various metals can produce granulomas, such as cobalt, beryllium, aluminum, or zirconium. Barbers and hairdressers suffer sometimes this granulomatous reaction due to a cut hair, normally present in an interdigital space (Fig. 1.6) [35].

### Pigmentary Disorders

Post-inflammatory hyperpigmentation due to work-related traumas, burns, or inflammatory diseases is quite often. Hyperpigmentation can result also from tattoos following injuries with coal dust, dyes, and various metals. Post-inflammatory hypopigmentation is less frequent, but it can also follow various traumas and skin diseases from work.

There are numerous chemicals that affect pigment cells, resulting in hyper- or hypopigmentation. A special concern is chemical leukoderma because it mimics idiopathic vitiligo. Most chemicals that produce this leukoderma are aromatic or aliphatic derivatives of phenols and catechols, but also mercury, arsenics, cinnamic aldehyde, p-phenylenediamine, corticosteroids, and other substances. Occupations at risk are workers in contact with glues, paints, oils, dyes,



**Fig. 1.6** Diverse fistulas due to hairs in the interdigital space of a dog groomer worker

and antioxidants, among others. The differentiation of chemical leukoderma from vitiligo is very important, because avoidance of the offending chemical shows a better outcome than vitiligo [36, 37].

### Low Humidity-Induced Disorders

Low humidity occupational dermatosis occurs in environments of low relative humidity (35% approximately), particularly if the temperature is high. Symptoms include pruritus, urticaria, and mild eczema in different locations. The problem is solved when the average relative humidity is raised to approximately 50% [38].

Semicircular lipoatrophy could be considered a special low humidity disorder, at least indirectly. This entity presents as a subcutaneous atrophy mainly on the anterior thighs of women who work in offices and other enclosed build-



ings. Several hypotheses have been suggested, but the increase of electrostatic energy could be an important factor. Enclosed buildings with cooling systems generate low levels of relative humidity, and this favors the presence of an increase in electrostatic energy. Women wearing synthetic clothes are at risk, because clothing discharge is produced through the area where the pressure is highest, which usually coincides with the anterior region of the thighs. Removing electrostatic charge generation in the workplace leads to resolution of lesions [39].

### Heat-Associated Disorders

Workers exposed to heat may develop different skin lesions. Miliaria can appear, especially with hot and humid environments, such as tropical climates. Intertrigo and hyperhidrosis can occur from work practice that requires safety shoes and a great deal of foot activity. Occluded feet in hot and wet environment can cause whitening and wrinkling of the sole with pain or altered sensation, fissure-like grooves, severe maceration between and under the toes, and erosions. Military combat personnel operating in tropical areas are at special risk, but these changes can appear in other workers with continuous water contact [7].

Erythema ab igne is caused by infrared radiation emitted by a heat source. It was more frequent in the past in bakers, fire fighters, and other workers with a history of chronic heat exposure. The condition is found predominantly on the inner and outer aspects of the shins [7]. Different localizations may exist if the source of heat affects other body areas. For example, working with laptops in contact with the body is a current cause of erythema ab igne on thighs, abdomen, or breasts, depending on where workers place them extensively [40]. Erythema ab igne has a potential to develop malignant lesions after many years, so prudent avoidance of triggers and follow-up in developed cases must be done.

Burns can result from different insults to the skin, like heat (thermal burns), chemicals, electricity, or radiation. Thermal burns account for almost 30% of all work-related burns. Hands, arms, and face are the most frequently affected

areas. These burns are normally due to accidents in heating, cleaning machinery like grills and ovens, and hanging power tools, like torches [41]. They also can be seen on the legs from hot asphalt used by road workers [7]. Prognosis of thermal burns depends on different factors, such as the depth of the burn (first/second/third degree), the localization, the total body surface area affected, the age, or the associated inhalational injury [42].

### Cold-Associated Disorders

Workers who spend long periods of time outdoors in cold climates or those who work in refrigerated rooms are prone to cold injuries, such as frostbites, pernio, acrocyanosis, cold panniculitis, or Raynaud's phenomenon [7]. Some of these entities have a genetic background and collagen-associated diseases that must be evaluated.

### Skin Cancer

Skin cancer from occupational origin may develop due to some chemicals, ultraviolet (UV) light, and ionizing radiation. It is widely suspected that the true incidence of occupational skin cancer is underreported [43]. Its etiologic factors need a chronic exposure and have a long latent period until the malignancy develops, hindering the diagnostic of occupational origin and emerging doubts on recreative activities and other non-occupational exposures.

Workers exposed to arsenic may develop skin cancer. Arsenic is used in the production of glass, the making of semiconductors, in the manufacture of insecticides and herbicides, in smelting and mining [44]. These workers may develop arsenical keratoses that are well-circumscribed keratotic papules on palms and soles. These arsenical keratoses may progress to squamous cell carcinoma. Induration, inflammation, and ulceration occur when the lesion becomes malignant [7]. Intra-epidermal carcinoma or multiple basal cell carcinomas may also be associated with arsenic exposure [44].

Polycyclic aromatic hydrocarbons are also associated with pre-malignant lesions, basal cell carcinomas, keratoacanthomas, and squamous

cell carcinomas. Industries in which these chemicals are produced include gas production from coal, coke plants, aluminum production, steel and iron foundries, and exposure to diesel engine exhaust fumes [44]. Cancer as an effect of coal tar on skin has been extensively investigated. Many studies support the tumorigenic potential of coal tar but indicate the necessity for chronic exposure. Now many countries have a strict regulation about its use. Nevertheless, significant sources of exposure still exist [25]. Occurrence of squamous cell carcinoma on skin that is not chronically exposed to sunlight should raise suspicion of work-related chemical carcinogen exposure [17].

UV-light exposure occurs mainly in outdoor workers, such as farmers, sailors, or construction workers. Besides the sun, other sources of UV-light exposure must be considered, as welding [45]. Legislation about contemplating a UV-light skin cancer as an occupational disease varies from one country to another. For example, in Germany, squamous cell carcinoma or multiple actinic keratoses have been recognized as an occupational disease since 2015, but they must meet certain requirements, such as the absence of other non-occupational risk factors or the additional UV exposure of at least 40% at work [3].

Ionizing radiation is well-recognized as having the potential to cause squamous cell carcinoma and pre-malignant changes from the experiences of the first scientists and physicians to use X-rays and other radiation sources [44]. Sometimes an occupational disease, and it is important to be aware of its cancerogenic potential [46].

Skin cancer can also arise from chronic scars due to occupational accidents. Its recognition is also important for the worker, in terms of possible compensations [47].

## Infections

The variety of infections that can be acquired at work is enormous, and in general parallel those acquired in the non-occupational setting. Nevertheless, some infections are characteristic of certain occupational activities and must be known by the physician.



**Fig. 1.7** Worker of a slaughterhouse affected with numerous viral warts during an outbreak in several workers manipulating lambs

Viral warts can be seen in meat and fish handlers (butcher's warts), sometimes with several workers affected in a workplace (Fig. 1.7). Viral warts may also appear in healthcare workers following laser surgery or cryotherapy, due to contamination [48]. Healthcare workers are also at risk of herpes simplex virus infections. Orf (ecthyma contagiosum) is caused by poxviruses that through the infection of goats and sheep can infect farmers, veterinarians, and shepherds. With clinical similarities to orf, milker's nodules are transmitted from cows.

Bacterial infections can be caused by staphylococci and streptococci acquired at workplace from diverse injuries. Anthrax, brucellosis, erysipeloid, tularemia, leptospirosis, and some mycobacteria infections can affect workers exposed to animals. Tick-borne diseases, such as Lyme disease and Rocky Mountain spotted fever, can affect outdoor and forestry workers.

Fungal infections may also have an occupational origin. Tinea pedis is more frequent with high humidity and occlusive footwear: miners, sailors, farmers, construction workers, and sportsmen are at increased risk. Candida colonization can contribute to chronic paronychia in workers exposed to water, cooking and irritants, such as dishwashing, bar work, laundering, food handling, and exposure to cutting oils [49]. Sporotrichosis occurs in miners, florists, and nurserymen. Coccidioidomycosis, mycetoma, and chromoblastomycosis may appear in agricultural and farm workers in some regions [7].

## Conclusion

The study of occupational dermatosis requires a broad knowledge of general dermatology, but particularly of contact dermatitis conditions and other eczematous entities with which its differential diagnosis is usually made. The existence of a temporal relationship between work and the appearance of an occupational dermatosis is key to establishing the diagnosis. Complementary tests (mainly patch- and prick-tests) must be carried out with the allergens to which the worker is exposed. It is essential the correct differentiation between irritant and allergic responses, as well as the establishment of the relevance of the sensitizations. It should not be forgotten that other entities, such as some forms of acne, psoriasis, or vitiligo, are many times caused or aggravated by work.

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