Guideline contact dermatitis

S1-Guidelines of the German Contact Allergy Group (DKG) of the German Dermatology Society (DDG), the Information Network of Dermatological Clinics (IVDK), the German Society for Allergology and Clinical Immunology (DGAKI), the Working Group for Occupational and Environmental Dermatology (ABD) of the DDG, the Medical Association of German Allergologists (AeDA), the Professional Association of German Dermatologists (BVDD) and the DDG

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Preamble

Purpose of the guidelines

The present guidelines on contact dermatitis aim to provide orientation to physicians of all disciplines tasked with treating contact dermatitis patients. They are intended to describe recognized diagnostic, therapeutic, and interventional approaches on the basis of current understanding of contact dermatitis.

Separate guidelines have been developed for hand dermatitis [1].

Development and consensus-building procedures

The guidelines were developed in formal consensus of a group of experts (see authors) put together by the medical specialty societies active in the field of contact dermatitis in Germany. These include the German Contact Allergy Group (DKG) of the Ger-

man Dermatology Society (DDG; Aberer, Kränke, Becker, Bircher, Brasch), the Information Network of Dermatological Clinics (IVDK; Schnuch, Aberer, Brasch), the German Society for Allergology and Clinical Immunology (DGAKI; Przybilla, Biedermann, Werfel), the Working Group for Occupational and Environmental Dermatology (ABD) of the DDG (John, Elsner, Diepgen), the Medical Association of German Allergologists (AeDA; Merk, Fuchs), the Professional Association of German Dermatologists (BVDD; Jung) and the DDG (*Trautmann*).

Taking the previous version of the guidelines [2] as a basis, the first author developed an initial draft updated in terms of formal structure and content. Additions and modifications were then made in the context of an email discussion involving all authors, until a general consensus among the authors was reached.

Taking into account German-language and PubMed medical specialist journals, the authors systematically evaluated the scientific literature on the topic of contact dermatitis. However, the authors' clinical experience was also taken into consideration. This decision seemed justified given that treatment approaches for contact dermatitis have been in use for decades and will continue to form the mainstay of clinical routine in spite of the fact that no efficacy studies according to currently valid criteria (double-blind, prospective, randomized) have been performed to date. Thus, disregarding empirical knowledge of this kind would have resulted in inadequate recommendations.

Limitations

These S1 guidelines were developed by the authors to the best of their knowledge and belief. However, the treating physician should review the adoption of these recommendations in each individual case, since deviations from recommended approaches may be necessary on the basis of individual circumstances. Studies in the future may also suggest alternative approaches.

Abbreviations

DD	Differential diagnosis
LST	Lymphocyte stimulation test
LTT	Lymphocyte transformation test
MELISA	Memory lymphocyte immunostimulation assay
PPD	Paraphenylenediamine
PUVA	Psoralen plus UV-A
ROAT	Repeated open application test
UV	Ultraviolet

Definition of contact dermatitis

An eczematous reaction is an inflammatory intolerance response characterized by successive and coexistent erythema, blisters, exudation, papules, and flaking. The term "dermatitis" is generally used as a synonym for "eczema". This response pattern is caused primarily by toxins that have an external, non-infectious, immunological, chemical, or physical effect. This is classically the case in contact dermatitis. However, eczematous skin reactions can also be triggered via endogenous pathways or by systemic allergen intake.

From an etiological perspective, a distinction is made between allergic – generally delayed type (type IV) and only rarely immediate type (type 1), as in protein contact dermatitis – and irritant (non-allergic) forms of contact dermatitis. Allergic forms presuppose sensitization to the offending allergen or a cross-reactive allergen. Irrespective of the varying etiology (type IV or type I allergy or skin irritation), a form of dermatitis develops. The irritant forms are also classified as toxic, degenerative, subtoxic, or cumulatively toxic. Many patients exhibit a combination of irritant and allergic mechanisms with an often synergistic effect [3].

Clinical symptoms alone often do not permit classification of the dermatitis as allergic or irritant contact dermatitis.

Acute, subacute, and chronic presentations can be distinguished according to morphology, development over time, and time of exposure to the toxin. This classification is also important for the choice of therapy.

Epidemiology

Allergen-specific (contact) sensitization is an essential precondition of allergic contact dermatitis. The prevalence of sensitization to individual contact allergens varies widely in Germany, Austria, and German-speaking areas of Switzerland according to patient populations, is partially occupation-related, and subject to special analyses and surveillance [4, 5, 6]. However, it is not possible to draw conclusions about the frequency of contact dermatitis directly from the prevalence of sensitization to contact allergens. The relevance of irritants as the potential causal agents of irritant contact dermatitis has been extensively investigated, particularly in certain occupational groups [7]. The likelihood of developing irritant contact dermatitis rises with the intensity and duration of exposure to the irritant. Depending on the occupational field investigated, irritant or allergic contact dermatitis represent the forms most commonly seen in terms of occupation-

al dermatitis [8]. Irritant contact dermatitis is often the precursor of further contact sensitization [9].

Allergic and irritant contact dermatitis are common diseases seen in many countries where they are by no means only work-related and where they generate considerable public-health and socio-economic costs [10, 11, 12].

Prevalence of contact dermatitis

The proportion of the German population estimated to be affected by some form of contact dermatitis is estimated at 15 %–20 % [12]. It is not unusual for children to be affected and some studies show that the incidence is rising among the pediatric population [13, 14, 15]. Contact eczema is also frequently seen in older adults as a result of age-related differences in exposure, changes in epidermal barrier function, and alterations in immune reactivity [16, 17]. According to a German health survey carried out in 2000 (Gesundheitssurvey 2000 [18]), the lifetime prevalence of allergic contact dermatitis is around 15 % and the annual prevalence approximately 7 %.

Danish studies in the 1990s reported a lifetime prevalence of hand dermatitis of 17 %, and already in adolescents aged between 12 and 16 years the prevalence was 7 % [19]. In Gothenburg, Sweden, the point prevalence of hand dermatitis among individuals aged between 20 and 65 years was 5.4 % and the 1-year prevalence 10.6 % [20]. On the basis of a review of original articles from the last 30 years, a point prevalence of hand dermatitis of 4 % was determined [21], whilst a 1-year prevalence of 2 % was given for acute contact allergic dermatitis of the hand. According to this review, the 1-year prevalence of hand dermatitis in general was approximately 10 % [21]. In a patient collective of the Information Network of Dermatological Clinics (IVDK), hand dermatitis accounted on average for approximately a third of all forms of contact dermatitis; assuming an equivalent proportion in Sweden would yield a prevalence of allergic contact dermatitis of 6 % [19]. Thus, the 1-year prevalence of 7 % determined by the German health survey appears to demonstrate relatively stable development over the decades [18]. Allergic contact dermatitis is undoubtedly a widespread disease with an incidence similar to that of diabetes.

Incidence of contact dermatitis

In the Netherlands, an incidence of 7.9 per 1000 person-years was observed for non-etiologically defined contact dermatitis [22]. Incidences determined for selected occupations are significantly

Table 1

"Classic" clinical forms of dermatitis

Form of dermatitis	Description
Irritant contact dermatitis	– Lesions restricted to the site of toxin exposure – Clearly demarcated in the acute stage – Broad spectrum of erythema thru to necrosis – Presentation strongly dependent on acuteness and toxin – No spreading
Allergic contact dermatitis	– Specific immunological sensitization to contact allergens – Area and configuration of the generally unclearly demarcated dermatitis are suggestive of the triggering agent – Spreading reactions, moving outwards from the primary site of exposure, are typical
Airborne allergic contact dermatitis	– Dermatitis on exposed areas of the body due to airborne allergens (in wall paint, plants, etc.)
Photo-contact dermatitis	– Occurs primarily in areas exposed to light – Substances that have a toxic effect when exposed to light (e.g., furocoumarin) trigger irritant dermatitis in the absence of sensitization – Photoallergies require prior sensitization
Asteatotic dermatitis	– Dry, cracked skin with red fissures, particularly in aging or damaged skin (incorrect care, excessive washing)
"Dry" chronic contact dermatitis	– On fingers and hands due to occupational dermatosis in dentists and gardeners
Dyshidrotic dermatitis or pompholyx	– Special clinical form of contact dermatitis (DD, special form of atopic dermatitis)
Hematogenous contact dermatitis	
Transfer contact dermatitis	– The allergen is transferred to other areas of skin without primary allergen contact, e. g., to the eyelids
Connubial contact dermatitis	For example, facial contact dermatitis following sensitization to PPD due to partner's dyed hair

DD, differential diagnosis; PPD, para-phenylenediamine

higher [23, 24]. An incidence of allergic contact dermatitis of 28 per 1000 per year was calculated in a collective made up of students at a university dermatological out-patient department in the US [19]. Using urban sample populations, British colleagues calculated an incidence of 0.6 per 1000 per year. If this rate were to be corrected by a factor that takes the consultation rate into consideration, the incidence would stand at 1.6 per 1000 per year.

Using a calculation model for a "moderate" scenario (assumptions lying somewhere between the two possible extremes), the incidence of allergic contact dermatitis was estimated at 3 per 1000 per year [25].

Maxim: As a public health problem, allergic contact dermatitis affects all age groups with a high prevalence and incidence.

Clinical picture of contact dermatitis

Clinical presentations of contact dermatitis

Tab. 1 shows the "classic" clinical forms of contact dermatitis.

Clinical symptoms depend primarily on whether the dermatitis is acute or chronic, as well as on the toxin involved, type of contact, pathomechanism, and localization, among other factors (**Tab. 2**) [26]. Although all types of dermatitis generally share common features, the classic eczematous stages in contact dermatitis (allergic and irritant) are most readily identifiable. Therefore, this particular variant of dermatitis is considered the classic example [26, 27].

Acute-stage eczematous reaction

Acute contact dermatitis is characterized by a largely uniform metachronous sequence of pathological symptoms over the entire lesion.

- Mild form: erythema at the site of exposure to the toxin, contact traces, and itching are possible.
- Severe form: ranging from vesicular papules (histologically: spongiotic blisters) to blisters, usually causing strong itching. A feeling of tightness of the skin and even pain may occur. Blister rupture is followed by weeping, scab formation, and later by scaliness, generally culminating in restitutio ad integrum. Spreading reactions are possible in the case of an allergic trigger.

Acute irritant contact dermatitis is characterized by: rapid onset (within hours) following generally easy-to-identify exposure, rapid clinical course, and usually also rapid resolution; its monomorphic and often highly intensive clinical symptoms (including possible skin necrosis); subjective symptoms perceived more as burning pain than itching; and clearly demarcated borders around the area of contact and the absence of spreading.

Table 2

Exogenous and endogenous factors affecting the inflammatory reaction and thus the clinical characteristics of dermatitis (adapted from [26])

Exogenous factors	Type of toxin (allergen, irritant, chemical structure, pH)
	Quantity of the penetrating substance (solubility, vehicle, concentration, type and duration of application)
	Body area
	Body temperature
	Mechanical factors (pressure, friction, abrasion)
	Chemical and physical factors (water, solvent, cold, UV radiation, etc.)
	Climatic conditions (temperature, humidity, wind)
	Partner contact
Endogenous factors	Individual sensitivity to the irritant
	Specific immunological sensitization
	Primary hyperirritable (sensitive) skin
	Predisposition to atopic dermatitis
	Incapacity to "harden"
	Secondary hyperirritability (status eczematicus)
	Ethnic factors
	Age
	Sensitivity to UV radiation
	Genetic disposition
	Polysensitization
	Pre-existing dermatoses (e.g., lower leg dermatitis)

Chronic dermatitis

Chronicity occurs when the skin continues to be exposed to the toxin, thereby preventing spontaneous healing of the dermatitis, or when the dermatitis persists even in the absence of the toxin.

From a morphological perspective, there are eczematous plaques with focal emphasis in more exudative or scalier areas. The initially relatively sharp demarcation becomes increasingly indistinct. The skin has a thickened appearance due to the infiltration of inflammatory cells and skin folds become accentuated (lichenification). The clinical picture is increasingly dominated by hyperkeratoses, rhagades, and lichenification.

The onset of chronic degenerative contact dermatitis is first seen after exposure lasting in some cases for up to years. The initial symptom is generally uncomfortable dryness of the skin, followed by erythema and flaking. Thereafter, it is characterized by a dry, hyperkeratotic-scaly, fissured/rhagade-like lesion of a less exudative nature. It follows a slow clinical course and heals only in a delayed manner, is largely – but not exclusively – restricted to the area of contact, and does not show a tendency to spread.

Table 3

The most important non-eczematous symptoms of contact allergic reactions

Erythema multiform-like reactions, e.g., following contact with topical medications (antiphlogistic agents, antibiotics) or plant allergens
Pigmented purpura or pigmented contact dermatitis, e.g., due to colorants and latex allergens
Lichen planus-like or lichenoid contact reactions in mucosa to dental allergens (e.g., in chronic metal contact)
Bullous, papular-nodular and pustular reactions, particularly to metal
Lymphomatoid or primarily dermally localized variants, e. g., to metal or hydroquinone
Primarily edematous reactions, e. g., due to PPD or azodyes
Granulomatous reactions to metal salts, e.g., in tattoos (DD sarcoidosis: further diagnostic steps may be required!)
Scleroderma-like lesions (due to organic solvents)

DD, differential diagnosis; PPD, para-phenylenediamine

Table 4

Important differential diagnoses in contact dermatitis

Atopic dermatitis
Seborrheic dermatitis
Stasis dermatitis
Nummular dermatitis
Mycosis
Cutaneous T-cell lymphoma (notably parapsoriasis en plaques)
Pityriasis rosea
Plaque psoriasis and pustular palmoplantar psoriasis
Lichen planus
Lupus erythematosus
Dermatomyositis

Variables

The development of dermatitis varies not only according to its course over time, but also according to body region and is co-determined by the type and aggressivity of the triggering agent and other parameters (**Tab. 2**). Relevant genetic factors are currently the subject of numerous studies [28, 29, 30, 31, 32, 33, 34, 35].

The varying localizations of dermatitis are not only suggestive of possible triggers, but sometimes also of the pathogenetic mechanisms involved. Typical sites of predilection for the initial symptoms of allergic contact dermatitis include the back of the hand and the lateral sides of the finger. Dermatitis triggered by ultraviolet (UV) light is, at least initially, restricted to areas exposed to light and spares facial areas shaded by the chin, ears, etc., as well as areas of the body covered by hair or clothing. Dermatitis on exposed areas of the body can also be triggered by airborne allergens, such as plant allergens or volatile substances in the workplace (e.g., epoxide resins) [36] (airborne contact dermatitis).

The various localizations of dermatitis can produce specific morphology. For example, angioedema-like swelling of the eyelids is often the only manifestation seen in the facial area, whereas dermatitis on the lower legs can appear "striped" following contact with plants, and textile dermatitis is typically worse in areas coming into intense contact with fabric (often intertriginous areas, e.g., armpits, groin). Due to the thick stratum corneum on the palms of the hands and soles of the feet, microscopic blisters can develop into large eruptions by means of confluence (Cheiropodopompholyx). Occasionally, once healed, post-inflammatory hypo- or hyperpigmentation may be seen. Scarring or granulomas develop only in very rare cases.

In contrast to irritant contact dermatitis, allergic contact reactions may exhibit spreading phenomena. Although the precise pathomechanism of these spreading reactions is unknown, intensive allergen contact and hematogenous distribution of the allergen or generalized activation of immunological effector cells are suspected pathways [37], among others.

Individual predisposing factors, particularly in chronic-degenerative dermatitis, are of considerable relevance, such as an underlying predisposition to atopic dermatitis or – usually age- or care-related – exsiccation of the skin. This frequently results in a combined pathogenesis of dermatitis. "Grafted allergies", i.e., the development of a contact allergy in the setting of an existing chronic-degenerative dermatitis, are not uncommon. Mixed clinical presentations comprising allergic and chronic-degenerative dermatitis are often challenging to classify from a clinical and differential diagnostic point of view. Secondary bacterial colonization (frequently with staphylococci, particularly in weeping dermatitis), dermatophytes (notably on hands and feet), or candida infections (body folds, particularly in infants and diabetics), and less commonly viruses make diagnosis difficult and complicate therapy. Pustules in the setting of dermatitis can lead not only to secondary infections, but also to weeping dermatitis in cases where inadequate occlusive ointment treatment is administered.

In brief: Dermatitis seen as the "final common pathway" of widely varying entities is affected by multiple variables. Diagnosis requires a differentiated approach, usually involving patch testing.

Non-eczematous clinical presentations of contact allergic reactions

These may be triggered by epicutaneous, cutaneous, and systemic allergen exposure [27, 37, 38, 39, , 40, 41]. Unusual contact allergens may be relevant [42]. The most important non-eczematous reaction patterns are shown in **Tab. 3**.

Note: Skin reactions to external contact agents can sometimes produce a clinical picture that is not suggestive of dermatitis at first glance.

Diagnosis

Patient history and the clinical picture are crucial to the diagnostic process. The most important differential diagnoses are summarized in **Tab. 4**.

Histological analysis of a skin biopsy is indicated in all cases showing atypical symptoms or clinical course.

Patient history includes questions relating to the development of the dermatitis and allergen exposure, as well as an assessment of causality. Once patch test results are available, questions relating to allergen exposure often need to be repeated in a second patient history. Due to the complexity of possible types of exposure, supplementary questionnaires to aid patient history taking have been developed for a number of occupations [43, 44, 45, 46, 47, 48, 49, 50].

The suspicion that dermatitis has been caused by exposure to an exogenous trigger is formed on the basis of allergen and/or toxin exposure and the clinical picture. In irritant contact dermatitis, the trigger is usually exposure of the skin to an irritant, such as frequent or prolonged contact with water, solvents and cleaning agents, dust, etc., that predominantly cause irritant reactions. The diagnosis of allergic contact dermatitis is made by detecting contact sensitization to causative allergens by means of patch testing. A detailed description of how to perform patch tests and evaluate their relevance is given in the relevant DDG guidelines [51]. It is essential to: use approved test substances (e. g., as in the series of tests recommended by the DKG), apply the patches in a methodically correct manner, and take a reading of reactions by the third day at the latest.

If no plausible result is achieved using a conventional patch test despite suspected contact allergy, modified patch testing methods are considered [52, 53, 54, 55, 56, 57, 58] (**Tab. 5**).

The methods described in **Tab. 5** require particular experience and should therefore only be carried out by specialists.

Table 5

Modifications and additions to patch testing

For the "strip" patch test, the horny layer is reduced prior to allergen application
For the repeated open application test (ROAT), a suspected allergen is repeatedly applied openly over several days
Although the atopy patch test enables atopic individuals to be investigated for airborne and food allergens following late phase reactions [52], the test has not yet been sufficiently validated [3][59]. For certain substances (e.g., drug preparations approved for intravenous use) intracutaneous testing with delayed readings can be helpful; however, cross-center validation is still lacking for this method
Additional scratch testing can be helpful if adequate transepidermal administration of the test substance is not possible with patch testing. Delayed readings over several days are necessary
Prick testing (or intracutaneous testing) can also be helpful in the case of suspected protein contact allergy; again, delayed readings are required

In brief: The diagnosis of contact dermatitis is based on patient history, clinical examination, and skin testing. Additional investigations may be necessary.

The lymphocyte-transformation or -stimulation tests (LTT or LST) and their modifications (e. g., memory lymphocyte immunostimulation assay, MELISA) should be used for scientific or highly specialized clinical investigations. Performing these tests is technically challenging and the methods are poorly standardized; thus, LTTs should remain the reserve of specialist laboratories that have particular experience with these test methods and the interpretation of their results. In the absence of a critical evaluation of LTT results in comparison with patch test results, possibly also a repeated open application test (ROAT) or exposed control person, their relevance is questionable and should not form the basis for prophylactic or therapeutic measures [60, 61, 62]. In exceptional cases involving very strong patch test reactions to para-phenylenediamine (PPD), LTTs can be helpful in preventing reactions due to cross-sensitization in further testing [61, 63]. Other in vitro methods for the diagnosis of contact allergies are not validated.

The guidelines of the German Association of Scientific Medical Societies (AWMF) (register number: 061/017, Renz et al.) state in this regard: "In high concentrations, some contact allergens can also function as mitogens (i. e., obligatory stimuli), making individual titration necessary. Whether the of-

ten poor specificity of LST for the analysis of metal compounds can be attributed to non-optimized conditions is unclear. Especially good correspondence between LST and patch testing is achieved for nickel sulfate in particular. However, from a dermatological point of view, there is no clinical indication to favor the complex in vitro test that is not validated for most allergens over the patch test, thereby leaving the real value of the LST in relation to contact allergens squarely in the domain of scientific investigations (and further development of the test system). Indiscriminate use of LST (or modifications thereof, such as MELISA) in the diagnosis of mercury allergies should be rejected."

Note: LTT (LST) is indicated in scientific, however generally not in clinical investigations of contact allergies.

There is currently no useful diagnostic test for the direct identification of irritant contact dermatitis [64]. Alkaline resistance testing, the Nitrazine yellow swab test, or measuring transepidermal water loss do not represent reliable diagnostic aids. Thus, the diagnosis of irritant contact dermatitis is made on the basis of patient history and clinical picture – once possible causal contact sensitization has been excluded – and can be indirectly confirmed by subsequent resolution following cessation of toxin exposure.

Treatment

Patient information

The successful treatment of contact dermatitis requires patient cooperation. The information provided to the patient and their mastery of the treatment, as well as care and protection measures, can contribute significantly both in terms of treatment and prophylaxis, particularly where occupation-related dermatitis triggers are relevant [65, 66, 67].

Avoiding the noxa

Contact dermatitis is triggered by exogenous toxins in the vast majority of cases. The most important therapeutic approach, therefore, is to cease causal exposure – no form of symptomatic treatment can substitute for this approach. Attempts to induce tolerance to contact allergens by means of immunotherapy have been hitherto unsuccessful [7, 68].

Where it is not possible to fully eliminate or avoid a triggering contact substance (allergen or irritant) in the individual's immediate environment, protective measures to prevent renewed skin contact are indicated. These include: personal protective clothing (often primarily protective gloves in the case of hazardous activities), work-related precautionary measures (modifying work processes, avoiding wet/humid work conditions, using extraction systems), and consistent stage-related treatment [69, 70]. These measures need to be tailored to the individual situation (toxic substance, type of exposure). Prolonged use of gloves should be avoided due to their occlusive effect, although these effects are apparently milder than originally assumed [71]. Adjuvant use of suitable skin barrier creams can be helpful [72, 73]. The selection of gloves and barrier creams should be made on the basis of their efficacy against the relevant toxins [74].

Dietary measures can be helpful in cases where a systemic hematogenous triggering of contact dermatitis in the setting of high-grade sensitization to an orally-ingested contact allergen is diagnosed (as evidenced by patient history, patch testing, exclusion diet, and diagnostic provocation). Under this premise, a low-nickel diet may improve symptoms in individuals allergic to nickel [75, 76, 77, 78], whilst chelating agents have also been described as helpful [79, 80].

Maxim: Avoiding the diagnostically determined noxa(e) is crucial.

Symptomatic treatment of contact dermatitis

Topical treatment is generally sufficient. As with other inflammatory dermatoses, the base in which the active substance is applied must be tailored to the severity of the dermatitis. Acute dermatitis is generally moist and needs to be treated with a hydrophilic preparation (gel, lotion, cream), whereas chronic disease is more likely to require a water-in-oil-based preparation (ointment). Needless to say, the base should not contain any allergens that may be relevant to the patient.

Corticosteroids

The efficacy of topical treatment with class-II or -III corticosteroids in acute allergic contact dermatitis is undisputed [81]; stronger preparations are required only in exceptional cases. However, weaker preparations at least do not always produce any detectable effect in irritant contact dermatitis [53]. The selection of a suitable corticosteroid with the appropriate efficacy should be made on the basis of the localization of skin lesions, as well as the severity and acuteness of the dermatitis, whilst bearing the therapeutic index in mind [82, 83]. Where long-term therapy is indicated, preparations bearing low risk of atrophy (e. g. mometasone furoate, methylprednisolone aceponate, hydrocortisone butyrate) are preferred [84, 85].

The general principles governing the use of corticosteroids apply equally to their use in the treatment of contact dermatitis. The known side effects of topical treatment must be borne in mind when deciding upon the type and duration of treatment.

Hence, topical corticosteroids represent the medication of first choice for the symptomatic treatment of contact dermatitis.

Calcineurin antagonists

In Germany, Austria, and Switzerland, calcineurin antagonists are only approved for the treatment of atopical dermatitis. They are less effective than strong corticosteroids in manifest contact dermatitis [87, 88, 89, 90, 91, 92, 96]. However, if long-term use is indicated, topical calcineurin antagonists may be beneficial in contact dermatitis compared to corticosteroids, particularly in sensitive areas of the skin (e. g., face, intertriginous areas), since they carry no atrophy risk [93]. With regard to safety, the reader is referred to the AWMF guidelines of the DDG on topical calcineurin antagonists and neurodermatitis [85, 94].

Ultraviolet therapy

Short-wave ultraviolet light (UVB) and PUVA (psoralen plus UV-A) are effective in chronic dermatitis, most notably in hand dermatitis [70, 95, 96, 97, 98]. In some forms of hand dermatitis, topical application of psoralens is advisable in the context of PUVA therapy in order to intensify the therapeutic effect. It appears possible to achieve a certain degree of "protective hardening" using UVB [101]. Positive data are also available on the use of UVA1 and narrow-band UVB, particularly in hand dermatitis [102, 103, 104].

Other external agents

Due to its antiphlogistic and antiproliferative effects, the use of coal tar as a follow-up treatment is still reasonable today in cases where other external agents are ineffective or declined by the patient. There is no evidence to support the fear that local treatment with coal tar is carcinogenic [105, 106, 107, 108, 109]. However, the known side effects of coal tar treatment (skin irritation and discoloration, acnegenic effect, photosensitization) must be borne in mind. Antiseptic agents such as triclosan, polyhexanide, octenidine, etc., are helpful in the elimination of germs in pathogenic microbial colonization. Iontophoresis can be beneficial in dyshidrotic dermatitis [110]. Soft X-ray therapy and Grenz ray therapy have proven to be helpful in the treatment of dermatitis [111, 112, 113, 114]. However, due to the harmful cumulative effects of X-rays to the skin, these methods are in principal contraindicated today and only justified in exceptional cases. The efficacy of topical non-steroidal antiphlogistic agents in contact dermatitis has not been sufficiently proven; in addition, there is a relevant risk of contact sensitization to these substances when used topically in dermatitis [115, 116]. Although Bufexamac has had its approval withdrawn by the European Medicines Agency due to its sensitization potential, it is still available in Switzerland and outside Europe. Moreover, many other substances for which no published data on efficacy are available are nevertheless used and recommended for the treatment of dermatitis. The same is true for antihistamines.

Systemic treatment

Systemic treatment may become necessary in cases where local treatment is insufficiently effective. It is essential to take the specific side-effects profile of the agents used into consideration.

Short-term systemic corticosteroid therapy (from 3 days up to 2 weeks) may be indicated, particularly for extensive contact dermatitis in acute, severe, and/or therapy-refractory cases, frequently in the case of systemic contact dermatitis (hematogenous contact dermatitis). The usual rules on systemic administration of corticosteroids apply here. Systemic administration of alitretinoin may be helpful in chronic hand eczema [117, 118, 119, 120]. In this regard, the reader is referred to the guidelines on hand dermatitis [1]. Insufficient data is available to date on the long-term effects [119]. Cyclosporine is currently the drug of first choice in the treatment of severe, therapy-resistant atopic dermatitis in adults, an indication for which it is approved. Long-term oral administration of cyclosporine A can be helpful in patients with therapy-resistant hand dermatitis [121, 122]. Other immunomodulators, such as azathioprine, mycophenolate mofetil, or methotrexate are also used for atopic dermatitis off-label (but only if cyclosporine is ineffective or contraindicated), and can also be considered for contact dermatitis [94, 123, 124].

Basic therapy and skin protection

Follow-up treatment with basic moisturizing agents to promote skin barrier regeneration and protect against recurrence, combined with the use of skin protection creams, is beneficial when individually tailored to skin status and skin exposure [125, 126, 127]. On the other hand, preparations containing unsuitable levels of water and fat or allergenic components may delay the resolution of dermatitis or even intensify the effect of substances harmful to the skin [127]. Although skin protection training is beneficial in the case of hazardous occupational ex-

posure [128], the effectiveness of skin protection creams alone under working conditions has not be unequivocally proven [129]. Complete restoration of barrier function is not expected until several weeks after the clinical resolution of contact dermatitis. However, the beneficial effect of moisturizers is measurable [130].

Evidence of therapeutic efficacy

Only a small number of prospective, randomized, double-blind, controlled studies meeting current criteria have proven the efficacy of the contact dermatitis treatments mentioned here in sufficiently large patient populations.

Relevant data supporting efficacy is only available for the use of topical corticosteroids and systemic administration of alitretinoin in hand dermatitis [84, 131, 118]. However, this does not mean by implication that the other treatment forms discussed here are ineffective. Although studies on conventional therapy methods in dermatitis may be lacking for many reasons, the long-term clinical experience of experts in terms of efficacy is undisputed.

Note: Individually tailored systemic therapy should be considered when topical therapy is either ineffective or unfeasible.

Reporting dermatitis

It is generally necessary to establish whether a case of contact dermatitis has been triggered by occupational exposure. Where work-related causality is possible, a dermatological report is drawn up – with the patient's consent – to the relevant statutory accident insurance (e.g., employer's liability insurance association) [132]. However, if the reasonable suspicion of an occupational disease has already been confirmed, i.e., ceasing the activity appears the only option once all avenues of prevention have been explored, it is legally required of the treating physician to report an occupational disease (in Germany, using form F6000) [133].

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Conflicts of interest
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