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Review

Beyond the Red Itch: Understanding Complex Occupational Skin Conditions †

Moazzam Zaidi * and Aiggan Tamene Kitila

Department of Public Health, Dunedin School of Medicine, University of Otago, P.O. Box 56, Dunedin 9054, New Zealand
* Correspondence: moazzamzaidi772@gmail.com
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Abstract: Occupational skin diseases (OSDs) represent one of the most prevalent categories of work-related illness, affecting workers across a broad spectrum of industries globally. Despite their high incidence, OSD remains frequently underdiagnosed and inadequately managed. This narrative review synthesises current knowledge on the anatomy and physiology of skin as the body's primary biological barrier, the spectrum of occupational exposures responsible for skin pathology, and the clinical entities that arise from such exposures. The paper examines high-risk occupational groups, describes common diagnostic approaches, and outlines evidence-based management strategies. Emerging issues including the rising burden of acrylate sensitisation in the beauty industry, nanoparticle exposures, and pandemic-driven overuse of disinfectants are also discussed. The review concludes by outlining preventive strategies in line with the hierarchy of controls.

Keywords: occupational skin disease; contact dermatitis; irritant; allergen; patch testing; skin absorption; chemical exposure; occupational medicine

1. Introduction

The skin is the human body's largest organ and its foremost physical barrier against the external environment. One square inch of skin contains approximately 19 million skin cells, 1000 nerve endings, 20 blood vessels, and 60,000 melanocytes [1]. Its structural integrity is essential not only for local defence but for systemic homeostasis: when the skin barrier is breached by chemical irritants, allergens, thermal insults, or mechanical trauma, the consequences extend well beyond superficial discomfort. Systemic toxicant absorption, secondary infection, chronic disability, and in some cases carcinogenesis may all ensue [2].

Occupational skin disease (OSD) constitutes one of the most prevalent and economically significant categories of work-related illness worldwide. Prevalence estimates vary by region and occupation but generally range from about 0.7 to 5 cases per 1000 workers annually, with considerably higher rates in high-risk industries such as healthcare, manufacturing, agriculture, and construction [3,4]. Despite this burden, OSDs remain persistently underreported and frequently misdiagnosed, in part because their clinical presentation overlaps with non-occupational skin conditions, and in part because the link between workplace exposure and skin disease is not always apparent to either the worker or the clinician. A contributing factor is that clinicians do not always enquire about a patient's occupation and job duties during clinical assessment. Where the occupational cause is not identified, treatment is directed at symptoms rather than the source of exposure, and the condition may persist or worsen despite ongoing management.

This review was originally presented at the 3rd International Commission on Occupational Health (ICOH) Scientific Committee on Occupational Medicine Conference in Lima, Peru (December 2025). It provides a comprehensive account of occupational skin conditions organised around four themes: skin anatomy and physiology as the biological foundation, the principal categories of occupational exposure, the clinical entities that

result, and the evidence base for prevention. Illustrative cases from clinical practice are integrated into the relevant sections. The review concludes by examining emerging challenges that are reshaping the occupational dermatology landscape.

2. Methods

This is a narrative review of the literature on occupational skin disease. The narrative format was selected because the aim was to synthesise a broad, clinically oriented body of evidence spanning multiple disciplines including occupational medicine, dermatology, toxicology, and epidemiology, rather than to answer a single focused clinical question amenable to systematic pooling.

2.1. Search Strategy

A structured literature search was conducted in PubMed/MEDLINE, Embase, and CINAHL from database inception to November 2025. The search combined free-text terms and controlled vocabulary (MeSH and Emtree) across three concept domains: (1) occupational context ("occupational disease", "occupational exposure", "workplace"); (2) skin disease ("dermatitis", "contact dermatitis", "skin disease", "eczema", "chemical burn", "urticaria", "skin cancer", "leukoderma"); and (3) clinical and preventive focus ("diagnosis", "patch test", "prevention", "control measures", "hierarchy of controls"). Boolean operators (AND, OR) were used to combine terms within and across domains. No language restrictions were applied, though non-English articles were included only where an English abstract was available. The reference lists of key review articles and textbook chapters were hand-searched to identify additional relevant sources.

2.2. Inclusion and Exclusion Criteria

Studies and sources were considered for inclusion if they addressed any aspect of occupational skin disease in adult working populations, including epidemiology, pathophysiology, toxicology**,** clinical presentation, diagnosis, management, or prevention. Both primary research and review articles were eligible. Guidelines and position statements from authoritative bodies including the International Contact Dermatitis Research Group, the European Society of Contact Dermatitis, and relevant national occupational health agencies were also included. Sources were excluded if they addressed skin disease in non-occupational contexts without relevance to work-related exposure, if they related exclusively to paediatric populations, or if full-text retrieval was not possible.

2.3. Screening and Study Selection

Titles and abstracts were screened by the authors against the inclusion criteria. Full texts were obtained for all potentially eligible sources. Given the narrative format and broad scope of the review, a formal PRISMA flow was not applied; however, selection decisions were guided consistently by relevance to the four thematic areas of the review. Where multiple sources addressed the same topic, priority was given to the most recent, highest-quality, or most cited evidence. Older foundational references were retained where they remained the primary source for an established concept.

2.4. Data Synthesis

Data were synthesised narratively. Evidence was not formally graded, but the strength and consistency of findings across sources are noted where relevant. Clinical cases included in this review are drawn from the primary author's own practice and are presented to illustrate key diagnostic and management principles. All cases have been de-identified.

3. Skin Anatomy and Physiology Relevant to Occupational Exposure

Understanding why the skin is both the first line of defence and a vulnerable target requires familiarity with its layered architecture and the factors that govern its permeability. The skin comprises three principal layers, each with distinct functions, and its capacity to exclude or admit substances depends on a set of well-characterised physicochemical and physiological variables.

3.1. The Epidermis

The epidermis is the outermost layer and the primary physical and chemical barrier. Its outermost sublayer, the stratum corneum, consists of tightly packed, keratinised dead cells surrounded by a lipid-rich matrix. This architecture resists penetration by most water-soluble substances [5]. The epidermis also houses melanocytes responsible for Ultraviolet (UV) protection, Langerhans cells that serve as the skin's sentinel antigen-presenting cells and are critical in allergic sensitisation, and Merkel cells for mechanoreception [6].

3.2. The Dermis

The dermis constitutes approximately 90% of skin thickness and is composed largely of collagen and elastin fibres that confer tensile strength and elasticity [7]. It contains hair follicles, sebaceous glands producing protective sebum, eccrine sweat glands involved in thermoregulation and the excretion of some chemicals, sensory nerve endings, and a rich vascular supply. Compromise of the dermis through deep chemical burns or chronic inflammatory processes results in scarring and permanent functional loss.

3.3. The Hypodermis

The hypodermis consists primarily of adipose tissue. It cushions underlying musculoskeletal structures, assists with temperature regulation, and provides conduits for cutaneous nerves and blood vessels. In severe burns or dermatoses, involvement of the hypodermis indicates full-thickness injury.

3.4. Determinants of Skin Absorption

The capacity of the intact skin barrier to exclude or admit substances depends on several intersecting factors. Lipid solubility and molecular size are central: small, lipid-soluble molecules penetrate the stratum corneum readily, while large or water-soluble molecules are substantially blocked under intact conditions [8]. Any compromise of barrier integrity dramatically increases permeability. Damaged, inflamed, or eczematous skin allows substantially greater absorption, as does dry and cracked skin [9]. Elevated skin temperature and occlusion under gloves or boots further increase absorption by promoting vasodilation and hydration of the stratum corneum. Higher chemical concentrations and longer or repeated contacts amplify the absorbed dose. Skin site also matters considerably: the face, scrotum, and axillae are far more permeable than the palms and soles [10]. At the regulatory level, the European Food Safety Authority (EFSA) has established a guidance framework for assessing dermal absorption of chemicals and pesticides, providing standardised methods for estimating how substances penetrate human skin to protect both workers and consumers [11].

4. Categories of Occupational Skin Exposure

Occupational exposures that affect the skin can be broadly grouped into thermal, chemical, biological, and physical categories. These are not mutually exclusive: many occupational settings involve simultaneous exposures across several of them, and the cumulative effect is often greater than any single exposure would predict.

4.1. Thermal Exposures

Heat and cold are significant occupational skin hazards, particularly for outdoor, manufacturing, and industrial workers. Heat exposure can produce a range of skin conditions from miliaria (heat rash caused by sweat duct obstruction) to full-thickness burns [12]. Most outdoor heat fatalities occur within the first few days of hot-environment work, before physiological adaptation is established, and young male workers in physically demanding sectors such as construction and agriculture are especially vulnerable [13]. Cold exposure can cause frostbite and non-freezing cold injuries such as perniosis (chilblains) and immersion foot, typically affecting exposed acral regions; industries such as fishing and transport carry the highest cold-related burden.

4.2. Chemical Exposures

Chemical exposures represent the dominant cause of occupational skin disease [3]. They are most usefully classified into three mechanistic categories: irritants, allergens, and systemically harmful chemicals. Irritants cause direct, non-immunological cellular damage to the skin at sufficient concentrations. Key examples include acids, alkalis, detergents, degreasers, organic solvents, disinfectants, and wet cement, which is highly alkaline [14]. Because irritation does not require prior sensitisation, any worker with sufficient exposure concentration or duration is at risk.

Allergens elicit a type IV delayed-type hypersensitivity immune response in susceptible, previously sensitised individuals. Common occupational allergens include nickel, chromates in cement, epoxy resins, rubber accelerators, acrylates, fragrances, and preservatives [15]. Unlike irritants, allergens can provoke disease at trace concentrations once sensitisation is established, making complete avoidance the only reliable management strategy. A subset of occupational chemicals can also penetrate intact or compromised skin and enter the systemic circulation in toxicologically relevant quantities, causing organ damage remote from the site of contact. Organophosphate and carbamate pesticides, aromatic amines, phenols, and polycyclic aromatic hydrocarbons are important examples [16].

4.3. Biological and Physical Exposures

Biological hazards affecting the skin include infectious microorganisms (bacteria, fungi, viruses, and parasites), plant-derived irritants and allergens such as urushiol from poison ivy and furocoumarins that cause phytophotodermatitis, and protein allergens that cause contact urticaria, notably natural rubber latex and food proteins in food handlers [17]. Mechanical friction, pressure, vibration, and ultraviolet radiation also contribute to the occupational skin disease burden. UV radiation from outdoor solar exposure is the predominant cause of occupational skin cancer, particularly basal cell and squamous cell carcinoma, while Polycyclic aromatic hydrocarbons (PAHs), ionising radiation, and arsenic are additional carcinogens with established skin cancer risk [18].

5. Occupational Groups at Risk and Clinical Entities

The interplay between exposure type and clinical outcome varies considerably across occupational settings. Table 1 summarises the principal occupational groups, their key skin hazards, and the conditions they most commonly develop. The sections below address each major clinical entity in turn, with illustrative cases drawn from clinical practice.

Table 1. Occupational groups with significant skin exposure hazards.

Occupation Group	Key Exposure Risks	Common Chemicals	Skin Disease/Injury Risk	Example Roles
Cleaners and Hygiene Workers	Wet work, strong irritants, glove occlusion	Detergents, bleach, disinfectants	Irritant contact dermatitis, hand eczema, fissures	Commercial cleaners, hospitality workers
Healthcare and Aged Care	Chronic wet work, disinfectants, maceration	Alcohol-based hand rubs, chlorhexidine, iodine	Irritant and allergic contact dermatitis, glove eczema	Nurses, dental staff
Construction and Trades	Strong irritants, potent sensitisers	Chromate-containing cement, solvents, epoxy resins	Allergic Contact dermatitis (CD) from chromates, irritant CD, chemical burns, occupational skin cancer (UV)	Painters, carpenters, plasterers, tilers
Automotive and Mechanical	Hydrocarbons, oils, solvents	Fuels, lubricants, brake fluids, degreasers	Oil acne, folliculitis, contact dermatitis, chemical burns	Mechanics, panel beaters, automotive painters
Agriculture and Pesticides	Pesticides, fuels, fertilisers	Organophosphates, carbamates, pyrethroids	Systemic toxicity, irritant CD, chloracne, chemical burns, occupational skin cancer (UV)	Farmers, sprayers, horticulture workers
Hairdressing and Beauty	High allergen load	Paraphenylenediamine hair dyes, bleach, acrylates/methacrylates	Allergic CD, hand eczema, nail dystrophy	Hairdressers, beauty therapists, nail technicians
Healthcare Laboratories	Potent sensitisers, corrosives	Formaldehyde, solvents, acids	Allergic CD, chemical burns, irritant CD	Laboratory technicians, pathology workers

5.1. Irritant Contact Dermatitis

Irritant contact dermatitis (ICD) is the most common occupational skin disease, accounting for approximately 60 to 80% of occupational contact dermatitis cases [4]. Because it results from direct chemical or physical injury to skin cells without immunological sensitisation, it can develop in any exposed worker given sufficient

concentration and duration of contact. Wet work, defined as skin contact with water, wet clothing, or occlusive gloves for more than two hours per working day, is the single most important occupational risk factor, and is ubiquitous in healthcare, food handling, hairdressing, cleaning, and catering [9]. Water itself is the most common single irritant in occupational practice; combined with soaps, detergents, or glove occlusion, its effect is greatly amplified. Acute ICD presents with erythema, vesiculation, oedema, and stinging or burning at the exposure site, with a sharp and well-defined border. Chronic ICD, more common in occupational settings due to repeated lower-level exposures, typically manifests as lichenification, fissuring, scaling, and dull pain or itch [19].

Note on case studies: All clinical cases presented in this review are illustrative and drawn from routine clinical practice. All cases have been fully de-identified; no identifying information has been retained.

Clinical case: Oil and gas worker with irritant contact dermatitis.

A 35-year-old male oil and gas worker presented with an acute inflammatory rash over the forearms and hands. He was applying an SDS (sodium dodecyl sulphate)-containing protein-based emulsion to pipelines as a corrosion inhibitor, alongside exposure to multiple other workshop chemicals. The distribution and temporal pattern were consistent with irritant contact dermatitis attributable to the protein-SDS emulsion. Upon cessation of emulsion use, his condition resolved, confirming the causal relationship. The case illustrates a recurring challenge in ICD diagnosis: the worker was simultaneously exposed to several potential irritants, making attribution non-trivial. Meticulous exposure mapping and a test-of-time assessment, observing whether symptoms improved on removal of the suspected agent, were essential to establishing the cause.

5.2. Allergic Contact Dermatitis

Allergic contact dermatitis (ACD) is a type IV delayed hypersensitivity reaction requiring two phases: an initial sensitisation phase during which dendritic cells present hapten-protein complexes to naive T lymphocytes and establish immunological memory, followed by an elicitation phase upon re-exposure to even minute quantities of the same antigen [20]. ACD accounts for approximately 20 to 30% of occupational contact dermatitis [3]. In contrast to ICD, the clinical presentation typically appears 12 to 72 h after re-exposure, may extend beyond the primary exposure site, and can be triggered by concentrations far below those required to cause irritation in an unsensitised individual [21]. Table 2 summarises the key distinguishing features of ICD and ACD.

Table 2. Distinguishing features of irritant versus allergic contact dermatitis.

Feature	Irritant Contact Dermatitis (ICD)	Allergic Contact Dermatitis (ACD)
Acute symptoms	Stinging, smarting → itching	Itching → pain
Chronic symptoms	Itching/pain	Itching/pain
Acute lesions	Erythema → vesicles → erosions → crusts → scaling	Erythema → papules → vesicles → erosions → crust → scaling
Margination	Sharp, strictly confined to exposure site	Sharp at site; may spread peripherally or generalise
Onset (acute)	Within hours of exposure	12 to 72 h after exposure
Concentration dependence	Dependent on concentration and barrier state	Relatively independent; trace amounts sufficient in sensitised individuals
Incidence	Any exposed worker	Sensitised individuals only

The distinction matters both clinically and occupationally. Once sensitisation to a workplace allergen is established, the worker must avoid that allergen completely and indefinitely. Substitution of glove material, reformulation of the product, or in some cases redeployment becomes necessary. The construction industry illustrates this well: chromate allergy from cement exposure is a well-established cause of chronic ACD that frequently ends careers [22]. Regulatory interventions can, however, meaningfully reduce the burden of ACD. In Europe and North America, mandatory limits on water-soluble chromate content in cement have been associated with a measurable decline in the prevalence of chromate-related ACD among construction workers, demonstrating that exposure reduction at source translates directly into disease prevention [23].

Clinical case: Factory maintenance worker with allergic contact dermatitis to tea tree oil.

A factory maintenance worker performing varied tasks presented with rashes at multiple skin sites that did not correspond to a single workplace chemical exposure. Systematic exposure mapping failed to identify an obvious occupational source. Further history revealed that the worker had been habitually applying tea tree oil (Melaleuca alternifolia) to minor skin cuts and abrasions after each work shift as a personal remedy. Patch testing using the Australian Baseline Series, which includes tea tree oil, yielded a strongly positive reaction. The diagnosis was allergic contact dermatitis attributable to a personal habit rather than a primary occupational exposure. This

case is a reminder that not all skin disease in workers is occupationally caused. A thorough history covering all sources of skin contact, occupational and non-occupational alike, is essential before attributing causation.

5.3. Chemical Burns

Strong acids, strong alkalis including wet cement and concentrated cleaning agents, certain solvents, and oxidising agents can cause full-thickness skin injury within seconds of contact [14]. Chemical burns differ importantly from thermal burns in that the damaging chemical reaction continues until the agent is fully diluted or neutralised, meaning delayed or inadequate irrigation allows the burn to progress [24]. Immediate and copious irrigation with water is the critical first-aid intervention for any chemical skin contact, regardless of the specific agent. In construction and manufacturing, wet cement is a particular hazard, causing alkaline burns that may not be initially painful and are therefore often treated inadequately.

5.4. Contact Urticaria

Contact urticaria presents as a transient wheal-and-flare reaction developing within 10 to 60 min of skin contact with the causative agent and resolving within 24 h [25]. It may be immunological, IgE-mediated as with natural rubber latex or food proteins in food handlers, or non-immunological, through direct pharmacological mast cell activation as with certain preservatives and vehicles. The distinction has important management implications: immunological contact urticaria can progress to anaphylaxis, and affected workers should carry a self-administered adrenaline auto-injector. Antihistamines are first-line pharmacotherapy for both forms.

5.5. Occupational Skin Cancer

Prolonged occupational UV radiation exposure is the dominant cause of work-related skin cancer, predominantly basal cell carcinoma and squamous cell carcinoma [26]. Outdoor workers in construction, agriculture, and maritime industries face the highest UV burden over their careers. Additional occupational carcinogens with proven skin cancer risk include PAHs, ionising radiation, and inorganic arsenic compounds, all classified by the International Agency for Research on Cancer (IARC) as Group 1 human carcinogens. Surveillance programmes including regular skin inspection are warranted in high-risk cohorts, and sun protection behaviours need to be actively promoted rather than assumed.

Clinical case: Automotive mechanic with chemical leukoderma and acneiform eruption.

A 40-year-old male automotive mechanic with 20 years of occupational exposure presented with progressive depigmentation of the dorsal hands and acneiform lesions on the face. His work involved regular replacement of brake pads and clutch components, entailing sustained dermal contact with phenolic resins. Hand hygiene practices were inconsistent. Examination revealed irregular hypopigmented macules on the hands consistent with chemical leukoderma, and comedonal and inflammatory papules on the face suggesting oil acne secondary to phenolic resin exposure. Management combined exposure reduction with clinical treatment: thorough workplace exposure assessment, substitution of phenolic resin-containing products where technically feasible, mandatory nitrile gloves, enforced hand hygiene protocols, and topical retinoid therapy for the acneiform component. This case illustrates that chemical exposures beyond simple irritants and allergens can produce a broad spectrum of skin pathology, and that acneiform and dyspigmentation conditions in workers should prompt inquiry into specific chemical exposures rather than default attribution to idiopathic causes.

6. Diagnostic Framework for Occupational Skin Disease

Diagnosing OSD requires more than pattern recognition of the rash itself; it requires systematic reconstruction of exposure history, meticulous clinical examination, and targeted investigation. A common pitfall is treating the skin findings in isolation without anchoring the diagnosis in a confirmed occupational exposure. Identifying the causative exposure is central to diagnosis and cannot always be achieved by the clinician alone. Collaboration with an occupational hygienist is often essential, as the occupational hygienist brings specialist expertise in workplace exposure assessment that the clinician typically does not possess. This collaboration enables accurate characterisation of what the worker was exposed to, at what concentration, and for how long, information that is indispensable for confirming occupational causation. Table 3 outlines the recommended stepwise approach.

Table 3. Stepwise diagnostic framework for occupational skin disease.

Step	Component	Key Actions
1	Occupational history	Document current and past job titles, specific tasks, materials handled, duration and frequency of skin contact, PPE use, and temporal relationship between work and symptom onset or improvement during time off.
2	Clinical examination	Document anatomical distribution of lesions, morphology, and evolution over time; note whether distribution corresponds to a plausible exposure site.
3	Exposure mapping	Identify all chemicals, biological agents, and physical hazards at the workplace; document the frequency and duration of exposure to each hazard; review Safety Data Sheets for every product handled
4	Patch testing	Gold standard for ACD; allergens applied to upper back for 48 h; readings at 48 and 96 to 120 h. Diagnosis requires consistent clinical history, positive test, and confirmed exposure.
5	Prick testing	Used where immunological contact urticaria is suspected; identifies IgE-mediated sensitisation.
6	Additional investigations	Bacterial and fungal cultures, skin biopsy, and serological assays may be required to exclude differential diagnoses.

Patch testing deserves particular emphasis. It remains the gold standard for identifying allergic sensitisation, but a positive patch test in isolation does not diagnose ACD: the clinical picture must be consistent with the allergen, and a plausible route of workplace exposure must be confirmed [27]. Conversely, a negative result does not exclude ACD if the relevant allergen was not included in the series tested. Occupational dermatology increasingly relies on expanded industry-specific panels, such as those for dental, construction, and cosmetics workers, to improve diagnostic yield.

7. Prevention Strategies

OSD is, to a substantial degree, preventable. The hierarchy of controls provides the organising principle for prevention, prioritising measures that remove or reduce the hazard at source over those that rely on individual worker behaviour. Accurate quantification of dermal exposure is an important prerequisite for selecting appropriate controls. Tools such as IH SkinPerm™, based on the SkinPerm model developed by toxicologist Wil ten Berge and made available through the American Industrial Hygiene Association (AIHA), can estimate dermal absorption from liquid and vapour exposures, supporting evidence-based decisions about the level of control required [28]. Table 4 outlines prevention strategies at each level of the hierarchy of controls.

Table 4. Prevention strategies for OSD by hierarchy of controls.

Level of Control	OSD-Focused Examples
Elimination	Remove a sensitiser or irritant from the process; stop unnecessary wet work; automate tasks involving direct skin contact.
Substitution	Replace a strong allergen or irritant with less sensitising formulations or alternative technologies.
Engineering controls	Install splash guards, local exhaust ventilation, dosing devices, and no-touch tools; redesign Personal protective equipment (PPE) to reduce friction and occlusion.
Administrative controls	Limit duration and frequency of exposure; introduce job rotation and mandatory rest breaks; establish standard operating procedures; screen workers and refer early.
PPE and personal skin protection	Select nitrile or other non-latex gloves and clothing appropriate to the specific hazard; provide emollients and skin-friendly cleansers for skin care and recovery; train workers and monitor compliance. Barrier creams are not recommended as a substitute for appropriate gloves, as hazardous substances can penetrate the cream and be absorbed through the skin [29].

Effective OSD prevention relies on layered controls rather than any single intervention. Elimination or substitution of the most hazardous agents should be the first aspiration, though it is not always technically feasible; engineering and administrative controls then carry the next greatest burden. Personal protective equipment including correctly selected gloves, barrier creams, and emollients is important but functions as the last line of defence. A recurring finding in OSD literature is that over-reliance on gloves without attention to glove selection, fit, and occlusion effects can itself contribute to ICD through maceration [30].

Education and pre-placement health assessments are important adjuncts. Workers with pre-existing atopic skin conditions are at substantially elevated risk for ICD and may benefit from modified duty arrangements,

particularly in high wet-work environments. Early recognition and management, before sensitisation or chronicisation occurs, substantially improves long-term prognosis.

8. Emerging Issues in Occupational Skin Disease

The landscape of occupational dermatology continues to evolve as new materials, working practices, and global events introduce novel exposure risks alongside established ones. Acrylate sensitisation has emerged as one of the most significant growing concerns, particularly in the beauty industry. Methyl methacrylate and other acrylates used in artificial nail products and UV-cured gel systems are potent sensitisers, and rates of acrylate ACD among nail technicians and beauty therapists have risen sharply over the past decade [31]. A particularly serious downstream consequence of occupational acrylate sensitisation is the subsequent loss of tolerance to acrylate-containing medical devices including skin-adhesive continuous glucose monitors, with significant implications for the personal healthcare of affected workers [32]. This intersection of occupational and personal medicine is not widely appreciated and warrants greater clinical awareness.

The proliferating use of engineered nanoparticles, including titanium dioxide, zinc oxide, silver nanoparticles, and carbon nanotubes, in industrial, cosmetic, and medical products raises growing concerns about dermal penetration, particularly through disrupted skin barriers [33]. Evidence on the toxicological consequences of dermal nanoparticle exposure remains limited, and the regulatory and epidemiological evidence base is developing more slowly than the technologies themselves. This lag presents a precautionary challenge for occupational health practitioners who must manage risk in advance of definitive guidance.

The COVID-19 pandemic dramatically increased occupational exposure to alcohol-based hand rubs and quaternary ammonium disinfectants, precipitating epidemic-level hand dermatitis particularly among healthcare workers [34]. Post-pandemic, elevated baseline rates of disinfectant and wet-work exposure have persisted across many sectors, sustaining a higher background burden of ICD than existed before 2020. More broadly, climate change, evolving industrial processes, and the introduction of bio-based and green chemistry products are continuously adding new sensitisers to the occupational environment, requiring ongoing vigilance in surveillance and reporting systems. Registration guidelines for occupational dermatoses, such as those developed by the Dutch Centre for Occupational Diseases (NCvB), provide a useful framework for standardising how cases are identified, classified, and reported across sectors [35].

9. Limitations of the Review

This review has several limitations inherent to narrative approaches. The literature search and study selection were not guided by a pre-registered protocol, and formal quality appraisal or evidence grading was not conducted. While these factors may introduce some risk of selection bias, the review provides a broad overview of the available evidence, and recommendations should be considered in light of the varying strength of supporting studies.

10. Conclusions

Occupational skin diseases remain among the most common work-related health conditions worldwide and continue to impose a significant burden on workers and industries alike. The skin's integrity as a barrier is central to protection from the chemical, biological, thermal, and physical hazards of many occupational settings, yet that barrier is readily compromised, and the consequences of its failure extend from local inflammation to systemic toxicity and malignancy.

Irritant contact dermatitis dominates the epidemiological picture, but the full spectrum of OSD demands equally broad clinical awareness. Diagnosis depends on a disciplined combination of occupational history, clinical examination, exposure mapping, and appropriate investigation. The case studies in this review illustrate that both over-attribution and under-attribution of skin disease to occupational causes are real risks in clinical practice.

Prevention is achievable. The hierarchy of controls provides a structured and effective framework, with the greatest gains coming from eliminating or substituting hazardous agents at source, supported by engineering controls, administrative measures, and appropriate personal protective equipment. As the nature of work continues to change through new materials, pandemic-driven practice shifts, and the emerging challenges of climate change and nanotechnology, continued surveillance, research, and education will be essential to protect worker skin health.

Author Contributions

M.Z.: conceptualization, methodology, data curation, investigation, writing (original draft preparation), writing (reviewing and editing). A.T.K.: data curation, investigation, writing (reviewing and editing). Both authors have read and agreed to the published version of the manuscript.

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Informed Consent Statement

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Data Availability Statement

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Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

During the preparation of this manuscript, Microsoft Copilot was used for grammar, language refinement, and general structuring of material derived from a conference presentation. All content has been reviewed, edited, and verified by the authors, who take full responsibility for the accuracy and integrity of the published work.

References

1. Wong, R.; Geyer, S.; Weninger, W.; et al. The dynamic anatomy and patterning of skin. *Exp. Dermatol.* **2016**, *25*, 92–98.
2. Baker, P.; Huang, C.; Radi, R.; et al. Skin Barrier Function: The Interplay of Physical, Chemical, and Immunologic Properties. *Cells* **2023**, *12*, 2745. https://doi.org/10.3390/cells12232745.
3. AlEdani, E.M.; Gurramkonda, J.; Chaudhri, S.; et al. COVID-19-Related Risk Factors for Developing Occupational Contact Dermatitis Along with Its Incidence, Prevention, and Management: A Systematic Review. *Cureus* **2024**, *16*, e68441.
4. Milam, E.C.; Nassau, S.; Banta, E.; et al. Occupational Contact Dermatitis: An Update. *J. Allergy Clin. Immunol. Pract.* **2020**, *8*, 3283–3293.
5. De Szalay, S.; Wertz, P. Protective Barriers Provided by the Epidermis. *Int. J. Mol. Sci.* **2023**, *24*, 3145. https://doi.org/10.3390/ijms24043145.
6. Baroni, A.; Buommino, E.; De Gregorio, V.; et al. Structure and function of the epidermis related to barrier properties. *Clin. Dermatol.* **2012**, *30*, 257–262.
7. Quan, T. Molecular insights of human skin epidermal and dermal aging. *J. Dermatol. Sci.* **2023**, *112*, 48–53. https://doi.org/10.1016/j.jdermsci.2023.08.006.
8. Shahinfar, S.; Maibach, H. In vitro percutaneous penetration test overview. *Front. Pharmacol.* **2023**, *14*, 1102433. https://doi.org/10.3389/fphar.2023.1102433.
9. Fluhr, J.; Moore, D.; Lane, M.; et al. Epidermal barrier function in dry, flaky and sensitive skin: A narrative review. *J. Eur. Acad. Dermatol. Venereol.* **2024**, *38*, 812–820.
10. Kapitány, A.; Medgyesi, B.; Jenei, A.; et al. Regional Differences in the Permeability Barrier of the Skin—Implications in Acantholytic Skin Diseases. *Int. J. Mol. Sci.* **2021**, *22*, 10428. https://doi.org/10.3390/ijms221910428.

11. European Food Safety Authority (EFSA); Buist, H.; Craig, P.; et al. Guidance on Dermal Absorption. *EFSA J.* **2017**, *15*, 4873. https://doi.org/10.2903/j.efsa.2017.4873.
12. Uter, W.; Kanerva, L. Physical Causes: Heat, Cold, and Other Atmospheric Factors. In *Kanerva's Occupational Dermatology*; John, S., Johansen, J., Rustemeyer, T.; et al., Eds.; Springer: Berlin/Heidelberg, Germany, 2020. https://doi.org/10.1007/978-3-319-68617-2_33.
13. Fatima, S.; Rothmore, P.; Giles, L.; et al. Extreme heat and occupational injuries in different climate zones: A systematic review and meta-analysis of epidemiological evidence. *Environ. Int.* **2021**, *148*, 106384.
14. Jacobsen, G.; Rasmussen, K.; Bregnhøj, A.; et al. Causes of irritant contact dermatitis after occupational skin exposure: A systematic review. *Int. Arch. Occup. Environ. Health* **2022**, *95*, 35–65.
15. Sasseville, D.; Belsito, D.V.; Zirwas, M.J.; et al. Occupational Contact Dermatitis in North American Production Workers Referred for Patch Testing: Retrospective Analysis of Cross-Sectional Data from the North American Contact Dermatitis Group 1998 to 2014. *Dermatitis* **2017**, *28*, 183–194.
16. Semple, S. Dermal exposure to chemicals in the workplace: Just how important is skin absorption? *Occup. Environ. Med.* **2004**, *61*, 376–382.
17. Doutre, M. Occupational contact urticaria and protein contact dermatitis. *Eur. J. Dermatol.* **2005**, *15*, 419–424.
18. Parikh, A.K.; Tan, I.J.; Cohen, B.A. Occupational Exposures and Skin Cancer: A Brief Report. *Skin Res. Technol.* **2024**, *30*, e70107.
19. Bains, S.; Nash, P.; Fonacier, L. Irritant Contact Dermatitis. *Clin. Rev. Allergy Immunol.* **2019**, *56*, 99–109.
20. Kostner, L.; Anzengruber, F.; Guillod, C.; et al. Allergic Contact Dermatitis. *Immunol. Allergy Clin. N. Am.* **2017**, *37*, 141–152.
21. Johansen, J.; Bonefeld, C.; Schwensen, J.; et al. Novel insights into contact dermatitis. *J. Allergy Clin. Immunol.* **2022**, *149*, 1162–1171. https://doi.org/10.1016/j.jaci.2022.02.002.
22. Kridin, K.; Bergman, R.; Khamaisi, M.; et al. Cement-Induced Chromate Occupational Allergic Contact Dermatitis. *Dermatitis* **2016**, *27*, 208–214.
23. Bregnbak, D.; Johansen, J.D.; Jellesen, M.S.; et al. Chromium allergy and dermatitis: Prevalence and main findings. *Contact Dermat.* **2015**, *73*, 261–280. https://doi.org/10.1111/cod.12436.
24. Palao, R.; Monge, I.; Ruiz, M.; et al. Chemical burns: Pathophysiology and treatment. *Burns* **2010**, *36*, 295–304.
25. Giménez-Arnau, A.; Pesqué, D.; Maibach, H. Contact Urticaria Syndrome: A Comprehensive Review. *Curr. Dermatol. Rep.* **2022**, *11*, 194–201.
26. Pega, F.; Momen, N.; Streicher, K.; et al. Global, regional and national burdens of non-melanoma skin cancer attributable to occupational exposure to solar ultraviolet radiation for 183 countries, 2000–2019: A systematic analysis from the WHO/ILO Joint Estimates of the Work-related Burden of Disease and Injury. *Environ. Int.* **2023**, *181*, 108226.
27. Burkemper, N.M. Contact Dermatitis, Patch Testing, and Allergen Avoidance. *Mo. Med.* **2015**, *112*, 296–300.
28. AIHA Exposure Assessment Strategies Committee. IH SkinPerm™. American Industrial Hygiene Association. Available online: https://www.aiha.org/public-resources/healthierworkplaces/workplace-hazards/apps-and-tools-resource-center/aiha-risk-assessment-tools/ihskinperm/ihskinperm-download (accessed on 17 June 2026).
29. Zhai, H.; Maibach, H.I. Barrier creams—Skin protectants: Can you protect skin? *Dermatitis* **2002**, *13*, 3–6.
30. Behroozy, A.; Keegel, T. Wet-work Exposure: A Main Risk Factor for Occupational Hand Dermatitis. *Saf. Health Work* **2014**, *5*, 175–180.
31. Voller, L.M.; Warshaw, E.M. Acrylates: New sources and new allergens. *Clin. Exp. Dermatol.* **2020**, *45*, 277–283. https://doi.org/10.1111/ced.14093.
32. Kucharczyk, M.; Słowik-Rylska, M.; Cyran-Stemplewska, S.; et al. Acrylates as a significant cause of allergic contact dermatitis: New sources of exposure. *Adv. Dermatol. Allergol.* **2021**, *38*, 555–560.
33. Filon, L.; Bello, D.; Cherrie, J.; et al. Occupational dermal exposure to nanoparticles and nano-enabled products: Part I—Factors affecting skin absorption. *Int. J. Hyg. Environ. Health* **2016**, *219*, 536–544.
34. Loi, A.; Aribou, Z.; Fong, Y. Improving Recovery of Irritant Hand Dermatitis in Healthcare Workers with Workplace Interventions During the COVID-19 Pandemic. *Front. Public Health* **2022**, *10*, 844269.
35. Dutch Centre for Occupational Diseases (NCvB). Registration Guidelines for Occupational Dermatoses. Available online: https://www.beroepsziekten.nl/registratierichtlijnen/beroepsdermatosen (accessed on 17 June 2026).