Sk'n deep

SKINdeep 2: e173515 (2026)
DOI: 10.1553/skindeep.2026.173515

Perspectives

Far-UVC in human skin: safety evidence and therapeutic potential in dysbiosis-driven dermatoses

Maximilian Zarfl[1], Peter Wolf[1]

1 *Medical University of Graz, Department of Dermatology and Venereology, Research Unit for Photodermatology, Graz, Austria*
Corresponding author: Peter Wolf (peter.wolf@medunigraz.at)

Abstract

Far-UVC exhibits potent antimicrobial activity while limiting penetration into viable epidermis. Unlike conventional 254 nm UV-C, far-UVC induces DNA photodamage only in superficial keratinocytes, with no evidence of basal cell involvement or photocarcinogenesis in preclinical and pilot human studies. These features have established far-UVC as a promising tool for infection control in occupied environments. Beyond disinfection, far-UVC may also represent a novel therapeutic modality in dermatology. Several dermatoses, including atopic dermatitis, cutaneous T-cell lymphoma, and polymorphic light eruption, are characterized by cutaneous dysbiosis that drive inflammation. Far-UVC's selective action on surface-associated pathogens could modulate microbial imbalance while sparing deeper commensals and host tissue. Early data suggest potential for ecological rebalancing and immune modulation. Translational studies are now needed to test whether controlled far-UVC exposures can reshape microbial communities and improve disease outcomes in dysbiosis-driven dermatoses.

Key words: Far-UVC, Skin safety, Microbiome modulation, Dysbiosis, Atopic dermatitis, Cutaneous T-cell lymphoma, Polymorphic light eruption

OPEN ACCESS

Subject editor: Johann W. Bauer
Received: 29 September 2025
Accepted: 1 December 2025
Published: 26 January 2025

Citation: Zarfl M, Wolf P (2026) Far-UVC in human skin: safety evidence and therapeutic potential in dysbiosis-driven dermatoses. SKINdeep 2: e173515. https://doi.org/10.1553/skindeep.2026.173515

Introduction

The search for safe and effective antimicrobial strategies has intensified in recent years, driven both by global viral outbreaks and the growing burden of antimicrobial resistance. Ultraviolet radiation is a well-established germicidal modality. Conventional germicidal UV-C at 254 nm causes profound DNA photodamage and photocarcinogenesis, precluding its use in occupied environments. In contrast, far-UVC within the 200–235 nm range demonstrates a markedly different biological profile. Because of its strong absorption by proteins and the stratum corneum [9], far-UVC penetrates only the outermost non-viable cell layers, while retaining potent antimicrobial efficacy against bacteria, fungi, and viruses [1–3, 16]. This distinctive profile—potent germicidal action combined with restricted skin penetration—has established far-UVC as a candidate for safe disinfection in occupied spaces. Importantly, its limited biological reach also raises the possibility of dermatological applications. Given the role of microbial dysbiosis in conditions such as atopic dermatitis (AD), cutaneous T-cell lymphoma (CTCL), and polymorphic light eruption (PLE), far-UVC may offer a novel approach to targeted microbiome modulation.

Antimicrobial activity

Robust inactivation of gram-positive and gram-negative bacteria, fungi, and viruses has been consistently reported at 222 nm and 233 nm. Far-UVC is effective against multidrug-resistant organisms such as methicillin-resistant *Staphylococcus aureus* (MRSA), extended-spectrum β-lactamase producing *Escherichia coli* (ESBL), and opportunistic pathogens including *Candida* species [2, 4, 5]. Interestingly, antimicrobial action appears to be independent of resistance phenotype, with equal susceptibility demonstrated in resistant and non-resistant strains [4, 5]. Inactivation is rapid: 233 nm LED systems achieved 5-log reductions within clinically relevant doses (20–60 mJ/cm²), while 222 nm excimer lamps eliminated airborne and surface pathogens with comparable efficiency [2, 7]. Nevertheless, efficacy is reduced in protein- and mucin-rich matrices, highlighting the need for contextual dose adjustments [7, 16].

Cutaneous safety and technical considerations

The central concern is whether repeated far-UVC exposure induces genotoxicity or photocarcinogenesis. Multiple preclinical models, including reconstructed 3D skin models, ex vivo studies, and murine experiments, consistently show that 222 nm and 233 nm irradiation at doses up to 100 mJ/cm² induces only superficial and transient cyclobutane pyrimidine dimers (CPDs) without basal keratinocyte involvement [1, 2, 9, 10]. Long-term murine experiments demonstrate absence of photocarcinogenesis after chronic 222 nm far-UVC exposure, in contrast to conventional UVC or narrowband-UVB [8]. An extreme human self-exposure pilot study up to 18 000 mJ/cm² found no erythema or basal DNA lesions [11]. Importantly, interindividual factors influence susceptibility. Recent investigations demonstrate that phototypes with low melanin content exhibit slightly higher CPD levels after far-UVC exposure, although still markedly below those induced by UVB [6, 14]. The thickness of the stratum corneum has been identified as the principal protective factor, and demographic variables such as age and sex do not significantly alter barrier thickness [14]. However, safety is also critically dependent on spectral purity. Early devices with residual >230 nm emission induced DNA damage [12]. Bandpass filtering eliminates this hazard [12, 15], though inter-device variability remains [15]. Recently, the concept of the lamp exposure limit (HLEL) has been proposed as a practical safety metric, enabling calculation of safe exposure times and installation parameters [13]. Advances in LED-based systems at 233 nm may further improve spectral control, efficacy, and integration flexibility [2, 7]. These data collectively support a favorable safety margin when devices are adequately filtered.

Far-UVC and dysbiosis-driven dermatoses

Dysbiosis is central to several inflammatory skin diseases. In AD, *S. aureus* dominates lesional skin and exacerbates inflammation via superantigens and proteases; targeted microbial modulation improves disease control [17]. In CTCL, *S. aureus* colonization is linked to disease progression through toxin-driven proliferation of malignant T cells [18]. In PLE, altered microbial composition has been reported, with significant shifts in bacterial communities after ultraviolet exposure [19]. Recent mechanistic data show that eradication of the skin microbiota restores cytokine production in PLE, highlighting a causal link between dysbiosis and abnormal immune responses [20]. Current antimicrobial strategies rely on systemic or topical antibiotics, with attendant risks of resistance and collateral disruption of commensals. Far-UVC irradiation, by contrast, penetrates only superficial epidermis, enabling selective suppression of surface-associated pathogens while sparing deeper commensal reservoirs [2, 10]. Experimental studies demonstrate effective reduction of Candida and bacterial pathogens on skin models without compromising tissue viability [2, 5]. This raises the hypothesis that far-UVC could be developed as a microbiome-modulating therapy. Controlled, local exposures might transiently suppress dominant pathogens such as *S. aureus*, permitting restoration of diversity and barrier function (Fig. 1).

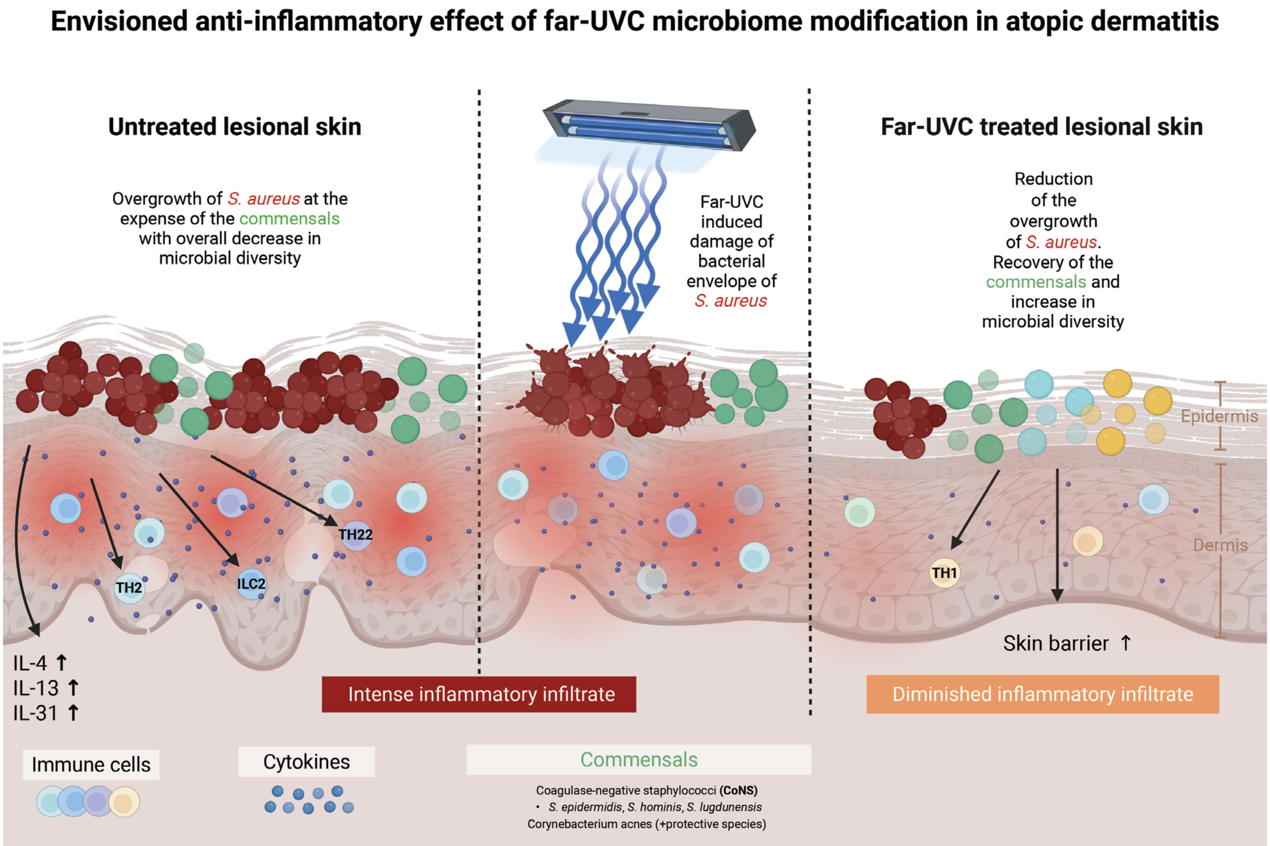

Figure 1. Envisioned anti-inflammatory effect of far-UVC microbiome modulation in atopic dermatitis. In lesional skin of atopic dermatitis (left), *S. aureus* overgrowth occurs at the expense of commensals, driving Th2 cytokine release (IL-4, IL-13), dense inflammatory infiltrates. Far-UVC irradiation (middle) reduces *S. aureus* burden at the skin surface. In treated lesional skin (right), restoration of microbial diversity is accompanied by attenuation of the inflammatory infiltrate, a shift towards more balanced Th1 responses, and reduction of epidermal thickening. These changes illustrate the potential of far-UVC not only to suppress pathogenic bacteria but also to improve barrier function in disease. The figure was created in BioRender. Zarfl, M. (2025) https://BioRender.com/vfsl2ky.

Controversies and research gaps

Despite current data on far-UVC are promising in terms of safety, several controversies remain, due to strong industrial involvement, underscoring the need for independent long-term studies [16]. Ocular safety is insufficiently characterized, with limited human data available. Likewise, chronic low-dose exposure in diverse skin phototypes warrants further investigation. Translation into clinical dermatology—such as targeted use for wound antisepsis and beyond—will require controlled clinical trials. In AD, endpoints could include microbial diversity, barrier recovery, and clinical severity scores. In CTCL, lesion-directed far-UVC could be tested for effects on microbial load and malignant T-cell activation. In PLE, prophylactic modulation of microbial composition may influence disease onset.

Conclusion

Far-UVC offers a compelling balance between broad antimicrobial efficacy and dermatological safety. Current data indicate minimal risk of basal DNA damage or carcinogenesis within exposure limits when properly filtered, though long-term human data remain unknown. Beyond antisepsis, a transformative possibility is the use of far-UVC as a targeted microbiome-modulating approach in dysbiosis-driven dermatoses such as AD, CTCL, and PLE [17–20]. Realizing this potential will require dermatology-led, independent clinical trials to define optimal doses, microbial and host outcomes, and long-term safety. If successful, far-UVC could inaugurate a new therapeutic class in dermatology, positioned at the intersection of photobiology and microbiome science.

Acknowledgements

The authors would like to thank Aaroh Anand Johsi, Medical University of Graz, for discussion and technical support in creating the BioRender figure.

Additional information

Conflict of interest

The authors have declared that no competing interests exist.

Use of AI

Artificial intelligence (AI) tools were used in the preparation of this manuscript exclusively to improve the fluency of English language expression and grammar. The scientific concept, literature review, data interpretation, and conclusions were conceived, analyzed, and written by the authors. No AI system was involved in the development of the research idea, evaluation of the evidence base, or generation of scientific conclusions. All content was critically reviewed, validated, and approved by the authors to ensure accuracy, originality, and compliance with the standards of the journal.

Funding

No funding was reported.

Author contributions

Conceptualization, MZ, PW; Visualization, MZ; Validation PW; Methodology, MZ, PW; Writing – original draft, MZ; Writing – Review & Editing, PW; Project administration, MZ, PW; Supervision and Funding acquisition, PW.

Author ORCIDs

Maximilian Zarfl https://orcid.org/0009-0005-3023-5577
Peter Wolf https://orcid.org/0000-0001-7777-9444

Data availability

All of the data that support the findings of this study are available in the main text.

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