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MICROBIOLOGICAL EFFECTS OF ULTRAVIOLET-C IRRADIATION ON PATHOGENS OF PURULENT WOUNDS: EXPERIMENTAL EVIDENCE, ANTIMICROBIAL MECHANISMS, AND THERAPEUTIC PROSPECTS

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Abstract

Background: Purulent wound infections remain a major clinical problem because of polymicrobial contamination, biofilm formation, delayed tissue repair, and the increasing prevalence of antimicrobial-resistant microorganisms. Staphylococcus aureus, methicillin-resistant S. aureus (MRSA), Pseudomonas aeruginosa, Acinetobacter baumannii, members of Enterobacterales, and Candida spp. are among the microorganisms frequently associated with infected wounds. Ultraviolet-C (UVC) irradiation has re-emerged as a non-pharmacological antimicrobial strategy because it can inactivate microorganisms independently of conventional antibiotic-resistance mechanisms.

Objective: To critically analyze the microbiological effects of ultraviolet irradiation on pathogens associated with purulent and infected wounds, with particular emphasis on wavelength-dependent activity, bacterial susceptibility, biofilm response, experimental wound models, tissue safety, and the translational potential of conventional 254-nm UVC, filtered 222-nm far-UVC, and UVC light-emitting diode systems.

Methods: Published experimental and clinical studies concerning UVC-mediated inactivation of wound-associated microorganisms were reviewed, with emphasis on S. aureus, MRSA, P. aeruginosa, A. baumannii, Escherichia coli, and Candida albicans. In vitro investigations, animal wound models, human wound studies, and recent clinical trials were evaluated according to wavelength, radiant exposure, microbial reduction, effects on wound healing, and tissue safety. The literature assessment was updated through September 2026.

Results: Conventional 254-nm UVC demonstrates rapid bactericidal activity against major wound pathogens but can also cause DNA photolesions in mammalian cells. In mouse wounds contaminated with P. aeruginosa or S. aureus, a single 254-nm UVC exposure significantly decreased bacterial burden, improved survival in P. aeruginosa infection, and accelerated healing of S. aureus-infected wounds. Filtered 222-nm far-UVC retains substantial antimicrobial activity while showing shallower penetration into mammalian tissues. Experimental MRSA wound models demonstrated bacterial reductions without the impairment of keratinocyte migration observed with conventional 254-nm irradiation. Human pressure-ulcer studies have additionally reported decreased microbial burden following 222-nm UVC treatment. UVC-LED irradiation around 275 nm has also demonstrated activity against bacterial and fungal wound pathogens, although its safety window

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requires further definition. Biofilm-associated organisms generally require greater radiant exposures than planktonic cells.

Conclusion: UVC irradiation represents a promising adjunctive strategy for superficial infected and purulent wounds, particularly when multidrug-resistant organisms are present. The microbiological response is strongly influenced by wavelength, radiant exposure, microbial species, biofilm organization, wound geometry, and tissue shielding. Filtered 222-nm far-UVC appears particularly promising because it may provide a more favorable antimicrobial-to-tissue toxicity ratio than conventional UVC. However, standardized dosing, device spectral purity, long-term tissue safety, and large controlled clinical studies are required before routine therapeutic use can be recommended.

Keywords: ultraviolet-C; UVC; far-UVC; purulent wound; wound infection; Staphylococcus aureus; MRSA; Pseudomonas aeruginosa; biofilm; antimicrobial resistance; wound healing.

Introduction

Purulent wound infection develops through a complex interaction between microorganisms, damaged tissue, host defense mechanisms, local perfusion, wound exudate, and therapeutic interventions. Bacterial contamination does not necessarily indicate invasive infection; however, progressive microbial proliferation, biofilm formation, inflammatory activation, tissue destruction, and impaired re-epithelialization can convert colonization into clinically important infection. Chronic wounds frequently contain Staphylococcus aureus, Pseudomonas aeruginosa, Enterobacterales and, in healthcare-associated settings, resistant organisms such as MRSA and multidrug-resistant Acinetobacter baumannii. Conventional antimicrobial treatment is complicated by increasing resistance, impaired antibiotic penetration into biofilms, local ischemia, repeated contamination, and the toxicity of some antiseptic agents to regenerating cells. These problems have stimulated renewed interest in physical antimicrobial technologies capable of reducing microbial burden without relying on conventional biochemical antibiotic targets.

Ultraviolet irradiation, particularly within the UVC spectrum, possesses broad antimicrobial activity. UVC primarily comprises wavelengths between approximately 200 and 280 nm and can disrupt microbial nucleic acids as well as, depending on wavelength, proteins and cellular membranes. Conventional germicidal lamps commonly emit near 254 nm, whereas newer technologies include 222-nm krypton-chloride excimer sources and UVC light-emitting diodes operating approximately between 260 and 280 nm.

The therapeutic application of UVC to wounds is fundamentally different from environmental surface disinfection. A clinically useful treatment must not only reduce microbial viability but also preserve keratinocytes, fibroblasts, vascular structures and inflammatory cells necessary for tissue repair. Consequently, the most important question is not simply whether ultraviolet radiation kills microorganisms, but whether a therapeutic

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window exists in which microorganisms are preferentially inactivated while wound healing remains intact.

This review examines the microbiological characteristics of ultraviolet irradiation against pathogens associated with purulent wounds, with particular emphasis on experimental evidence, differential susceptibility, biofilms, tissue safety and newer far-UVC approaches.

2. Materials and Methods

2.1. Literature search strategy and evidence selection

A targeted narrative literature assessment was performed using PubMed/MEDLINE and peer-reviewed primary publications concerning ultraviolet irradiation and infected wounds. Search concepts included combinations of ultraviolet C, UVC, far-UVC, 222 nm, 254 nm, 275 nm, wound infection, purulent wound, MRSA, Staphylococcus aureus, Pseudomonas aeruginosa, Acinetobacter baumannii, Candida albicans, biofilm, and wound healing.

Priority was given to studies directly assessing microbial viability, colony-forming units, bacterial bioluminescence, wound bacterial burden, histological repair, keratinocyte migration, or clinically measured wound outcomes. In vitro studies were interpreted separately from animal wound experiments and human clinical evidence because irradiance, tissue architecture, microbial shielding, and host immune responses can substantially modify UVC effectiveness in vivo.

Particular attention was paid to wavelength because the biological effects of 222-, 254-, and 275-nm radiation cannot be assumed to be equivalent.

3. Results

3.1. Microbiological profile of purulent wounds

The microbiology of purulent wounds is heterogeneous. Acute traumatic wounds may initially contain environmental or skin-derived organisms, whereas chronic ulcers and healthcare-associated wounds frequently develop complex microbial communities.

Staphylococcus aureus is particularly important because it can colonize damaged tissue, generate multiple virulence factors and form biofilms. MRSA adds the problem of reduced susceptibility to multiple conventional antibacterial agents.

Pseudomonas aeruginosa is a highly adaptable Gram-negative pathogen with a strong tendency to establish biofilms in moist wound environments. Its extracellular polymeric matrix, metabolic heterogeneity, efflux systems and intrinsic antimicrobial resistance contribute to persistence.

A. baumannii is particularly relevant to traumatic, military and hospital-associated wounds, where multidrug-resistant strains may complicate treatment. Experimental work has specifically investigated UVC decontamination of wounds infected with multidrug-resistant A. baumannii.

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Other Gram-negative bacteria, including E. coli, Klebsiella pneumoniae and Proteus spp., may occur in polymicrobial wounds. Fungal colonization or infection, particularly by C. albicans, may also become clinically relevant in burns and severely compromised tissue.

This diversity is important because microbial susceptibility to ultraviolet radiation is not uniform.

3.2. Antimicrobial mechanisms of UVC irradiation

The classical antimicrobial action of UVC is associated with absorption of ultraviolet photons by microbial nucleic acids. This produces photochemical lesions, particularly cyclobutane pyrimidine dimers and 6-4 photoproducts, which interfere with DNA replication and transcription. If the accumulated damage exceeds the microorganism's capacity for repair, replication stops and the cell loses viability.

The biological mechanism depends partly on wavelength. At approximately 254 nm, nucleic acids represent a major target. Some bacteria possess photoreactivation and nucleotide-excision mechanisms capable of repairing portions of UV-induced DNA damage. Experimental observations have demonstrated photoreactivation in P. aeruginosa following UVC exposure under selected conditions.

Far-UVC near 222 nm appears to produce a more complex pattern of microbial injury. Because proteins absorb strongly at this wavelength, 222-nm irradiation can damage membrane and structural proteins in addition to inducing nucleic-acid lesions. Recent mechanistic work suggests that degradation of membrane proteins and peptidoglycan, increased membrane permeability, leakage of intracellular material and DNA injury may jointly contribute to bacterial inactivation.

These differences may be particularly important for wound treatment because protein-rich exudate, necrotic material, fibrin, biofilm matrix and irregular tissue surfaces can absorb or scatter ultraviolet photons before they reach bacterial cells.

3.3. Conventional 254-nm UVC: experimental activity and pathogen susceptibility

The bactericidal effect of conventional 254-nm UVC has been demonstrated against several important wound microorganisms.

Dai et al. developed murine partial-thickness wound models infected with bioluminescent P. aeruginosa or S. aureus. Approximately 10^7 bacterial cells were inoculated into cutaneous abrasions, and UVC was applied 30 minutes after contamination. A single radiant exposure of 2.59 J/cm² reduced bacterial burden by approximately one order of magnitude compared with untreated wounds. In the P. aeruginosa model, UVC improved survival by 58.3%, while in S. aureus-infected wounds the wound-healing rate increased by approximately 31.2%.

These findings are microbiologically important because P. aeruginosa infection in this experimental system was capable of progressing toward lethal systemic disease. Early reduction of local microbial burden therefore affected not only wound colonization but also the biological course of infection.

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The same investigators examined multidrug-resistant A. baumannii. UVC exposures of 3.24 J/cm² in abrasion wounds and 2.59 J/cm² in burns reduced bacterial burden by approximately tenfold relative to untreated controls.

Experimental 254-nm irradiation has also been evaluated against C. albicans in infected burn models, demonstrating that the antimicrobial spectrum is not restricted to bacteria.

The principal disadvantage of conventional UVC is host-cell phototoxicity. In the murine study by Dai et al., UVC-induced DNA lesions were detectable in wound tissue immediately after treatment, although substantial repair occurred over the subsequent 48 hours. This observation illustrates the central therapeutic dilemma: the radiation dose needed for microbial control must remain compatible with tissue regeneration.

The response of wound microorganisms to UVC depends on cell structure, physiological state, pigmentation, DNA-repair capacity, extracellular matrix, and exposure conditions.

In some experimental systems, P. aeruginosa has shown greater sensitivity to direct UVC exposure than MRSA in planktonic culture. However, this relationship can change dramatically once biofilms form. P. aeruginosa produces abundant extracellular polymeric substances that can attenuate ultraviolet penetration and shield deeper bacterial populations.

Therefore, a statement such as “Gram-negative organisms are more UV-sensitive than Gram-positive organisms” is too simplistic for wound microbiology. Planktonic susceptibility does not necessarily predict susceptibility inside a mature purulent wound.

Distance from the light source, radiation angle, irradiance, exposure time, wound exudate and surface irregularities also influence the effective dose reaching individual microorganisms.

3.4. Biofilm response to UVC irradiation

Biofilm formation is one of the major reasons why chronic purulent wounds can persist despite antimicrobial therapy. Within a biofilm, bacteria are embedded in an extracellular matrix composed of polysaccharides, extracellular DNA, proteins, lipids and host-derived material.

This matrix affects UVC treatment through several mechanisms. It can absorb and scatter radiation, physically shield cells located below the surface, create microbial subpopulations with different metabolic states, and preserve dead biomass after organisms are inactivated.

Tingpej et al. demonstrated that ultraviolet irradiation could inactivate multiple nosocomial organisms growing in biofilms, but exposure did not necessarily remove the extracellular biofilm matrix. P. aeruginosa biofilm was particularly difficult to eradicate in that experimental system.

More recently, Palma et al. evaluated 254-nm UVC against preformed biofilms of MSSA, MRSA, methicillin-resistant S. epidermidis, E. coli, P. aeruginosa and C. albicans. At 946.7 mJ/cm², reported log10 reductions for staphylococcal biofilms were

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approximately 4.3–4.9, whereas C. albicans showed lower susceptibility. Gram-negative bacterial biofilms were strongly reduced at lower tested doses in that experimental setting.

These findings have an important clinical implication: microbial killing does not necessarily equal physical biofilm removal. Debridement may therefore remain necessary even when UVC substantially decreases viable organisms.

3.5. Filtered 222-nm far-UVC

Filtered far-UVC around 222 nm has attracted considerable interest because its penetration through mammalian tissue is markedly lower than that of conventional germicidal UVC.

The proposed mechanism is based on strong absorption by proteins. Bacterial dimensions are small enough for far-UVC photons to reach essential intracellular and membrane targets, whereas the same wavelength is strongly attenuated within superficial protein-rich layers of mammalian tissue. Experimental studies using appropriately filtered 222-nm sources have therefore reported efficient bacterial killing with substantially less deep tissue injury than conventional 254-nm lamps.

Narita et al. directly investigated 222-nm UVC in MRSA-infected mouse wounds. Immediate bacterial counts were approximately 1–1.5 log lower than untreated wounds at several tested radiant exposures. Importantly, histological evaluation demonstrated that keratinocyte migration was impaired following conventional 254-nm exposure in the experimental model, whereas 222-nm UVC did not produce the same impairment.

Far-UVC has also demonstrated activity against a broad spectrum of organisms including S. aureus, P. aeruginosa, E. coli, C. albicans and bacterial spores. However, fungal spores and hyphae may be less susceptible to 222-nm radiation than vegetative bacterial cells.

This differential susceptibility suggests that the microbial composition of a wound should be considered when defining a treatment protocol.

3.6. Comparative evidence across UVC wavelengths

The comparative evidence indicates that wavelength selection changes both antimicrobial effects and tissue response.

Table 1. Selected experimental and clinical evidence relevant to UVC treatment of infected wounds

Study	Wavelength	Model / microorganisms	Major microbiological finding	Wound/tissue finding	Main limitation

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Study	Wavelength	Model / microorganisms	Major microbiological finding	Wound/tissue finding	Main limitation
Dai et al., 2012	254 nm	Mouse wounds; P. aeruginosa, S. aureus	2.59 J/cm² produced approximately 10-fold reduction in bacterial burden	Increased survival in P. aeruginosa infection and faster healing in S. aureus wounds	Conventional UVC produced DNA lesions in host tissue
Dai et al., 2012	254 nm	Mouse wounds; MDR A. baumannii	Approximately 10-fold reduction following UVC	Demonstrated feasibility of traumatic-wound decontamination	Animal model; high radiant exposures
Narita et al., 2018	222 vs 254 nm	MRSA-infected mouse wounds	222 nm significantly reduced MRSA burden	Keratinocyte migration preserved better than after 254 nm	Small animal groups
Buonanno et al., 2017	222 vs 254 nm	MRSA; skin models and mice	Efficient MRSA killing	Filtered 222 nm caused substantially less mammalian skin damage	Not a purulent-wound clinical study
Goh et al., 2021	222 nm	Human pressure ulcers	Median CFU reduction 78.9%; complete eradication in 29.4% of sessions	No reported irradiation-related adverse events	Pilot study, 16 patients
Sheikh et al., 2021	275 nm LED	P. aeruginosa, S. aureus + fibroblasts	Strong antibacterial activity	Substantial fibroblast viability retained under tested conditions	In vitro system

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Study	Wavelength	Model / microorganisms	Major microbiological finding	Wound/tissue finding	Main limitation
Song et al., 2022	275 nm LED	Mouse SSTI models; MRSA and C. albicans	Dose-dependent antimicrobial activity	Shortened healing time in infected wounds	Translation of mouse dose to humans uncertain
Palma et al., 2025	254 nm	Preformed bacterial/fungal biofilms	>3-log reductions achievable at high doses; species-dependent susceptibility	Not a wound model	Surface biofilm study
Tsai et al., 2026	222 nm	Randomized study, pressure injuries	Greater bacterial reduction compared with standard care at follow-up	Improved wound scores and area reduction	Single center, modest sample size

3.7. UVC-LED systems near 275 nm

Development of compact UVC-LED devices has expanded the range of possible wound-treatment systems. LEDs allow precise wavelength selection, rapid on/off operation, lower device size and potentially improved dose control compared with traditional mercury lamps.

Sheikh et al. investigated a 275-nm UVC-LED against P. aeruginosa and S. aureus. Both organisms showed substantial inhibition under the tested exposure conditions. Scanning electron microscopy demonstrated structural changes in bacterial cells after irradiation, while human skin fibroblast viability remained relatively high across the tested experimental parameters.

Song et al. subsequently studied 275-nm UVC-LED irradiation in vitro and in infected mouse skin. MRSA, E. coli, P. aeruginosa, and C. albicans were among the susceptible organisms. In mouse models of MRSA- and C. albicans-associated skin and soft-tissue infection, UVC-LED treatment decreased microbial burden and shortened wound-healing time relative to untreated infected controls.

The findings are promising, but 275 nm penetrates biological tissue differently from 222 nm. Consequently, safety results obtained with filtered far-UVC cannot simply be transferred to UVC-LED devices operating near 275 nm.

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3.8. Human clinical evidence

Translation from laboratory culture to an infected human wound is difficult because pus, necrotic tissue, fibrin, exudate, wound depth and complex geometry can shield microorganisms.

Early clinical studies nevertheless suggest that superficially accessible microorganisms can be reduced by UVC.

Thai et al. examined 22 chronic ulcers with signs of infection or critical colonization. A single 180-second 254-nm UVC exposure produced a statistically significant reduction in semiquantitative bacterial burden. Particularly marked reductions were observed in wounds containing P. aeruginosa, and significant decreases were also reported among ulcers containing MRSA.

A preceding case series involving chronic MRSA-positive ulcers also reported reduction in microbial burden and improvement in wound closure, although the uncontrolled design prevents firm causal conclusions.

Goh et al. subsequently evaluated 222-nm UVC in patients with stage 2 or 3 sacral or gluteal pressure ulcers. Across 68 irradiation sessions in 16 patients, 87% of sessions showed a decrease in CFU counts, the median reduction was 78.9%, and complete eradication was observed in 29.4% of sessions. Cultured organisms included MRSA, P. aeruginosa and Klebsiella pneumoniae. No adverse events were reported during the sessions.

More recently, Tsai et al. conducted a randomized study involving 70 ICU patients with pressure injuries. Patients assigned to 222-nm UVC received daily 60-second exposures for seven days before dressing changes. Sixty-five participants remained in the final analysis. Compared with standard care, the irradiation group demonstrated greater improvement in wound scores, bacterial counts and wound area at selected follow-up points. The authors appropriately emphasized the need for larger multicenter confirmation.

These newer human data strengthen the rationale for further clinical investigation of 222-nm technology.

3.9. Effects on wound healing and tissue safety

Reduction of microbial burden can indirectly facilitate healing by decreasing inflammatory stimulation, protease production, bacterial toxins and competition for tissue nutrients. This may partly explain the faster wound closure observed in several experimental infection models.

However, UVC itself can affect host cells.

Keratinocytes must proliferate and migrate across the wound surface during re-epithelialization. Fibroblasts generate extracellular matrix and participate in wound contraction. Endothelial cells are required for neovascularization. Excessive ultraviolet injury to any of these compartments could counteract antimicrobial benefit.

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The Narita MRSA wound model is particularly informative in this context. Conventional 254-nm irradiation impaired epidermal-cell migration, whereas 222-nm-treated wounds showed migration patterns closer to untreated wounds.

Thus, optimal wound phototherapy should be evaluated using a combined endpoint: microbial reduction + preservation of regenerative tissue, rather than microbial killing alone.

3.9.1. Safety considerations

Conventional 254-nm UVC can induce cyclobutane pyrimidine dimers and other DNA lesions in mammalian cells. Repeated or excessive exposure therefore raises concerns regarding mutagenicity, delayed repair and carcinogenesis.

Filtered 222-nm systems may reduce this risk because the radiation has limited penetration through protein-rich superficial tissue. Experimental skin models and human investigations generally support a wider safety margin for properly filtered 222-nm light.

Nevertheless, the word filtered is critical. Krypton-chloride excimer lamps can emit small amounts of longer-wavelength ultraviolet radiation. Optical filtering substantially alters biological safety, and unfiltered devices should not be assumed to share the safety characteristics reported for filtered systems.

Furthermore, recent experimental literature demonstrates that even far-UVC biology is more complex than a simple “safe versus harmful” dichotomy. Cellular responses depend on wavelength purity, dose, tissue model and biological endpoint.

Accordingly, therapeutic wound irradiation should use calibrated medical-grade equipment rather than generic germicidal lamps.

3.10. Factors determining treatment efficacy and antibiotic-resistance considerations

The effectiveness of ultraviolet irradiation in purulent wounds can be conceptualized as an interaction among microbial susceptibility, optical delivery and wound architecture.

Planktonic organisms located directly on an exposed wound surface receive the greatest dose. Bacteria located in crypts, hair follicles, deeper tissue, thick exudate or mature biofilm may receive substantially less irradiation.

Purulent material itself can therefore become a physical protective layer.

This provides a microbiological rationale for wound cleansing and debridement before light-based treatment. Removing pus, necrotic debris and devitalized tissue may increase photon access to viable microorganisms.

The timing of irradiation may also matter. Experimental prophylactic treatment applied shortly after bacterial inoculation has often been more successful than treatment of an established deep infection. In the Dai experiments, UVC was applied only 30 minutes after contamination.

Therefore, the impressive efficacy of prophylactic animal experiments should not automatically be extrapolated to long-standing human abscesses or deeply infected wounds.

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3.10.1. Antibiotic resistance and UVC susceptibility

One of the most attractive features of UVC is that conventional antibiotic-resistance mechanisms do not directly prevent photon-mediated cellular injury.

Methicillin resistance, beta-lactamase production, altered penicillin-binding proteins and many drug-efflux mechanisms do not intrinsically protect DNA or cellular proteins from sufficiently intense UVC exposure. This explains why MRSA remains susceptible to far-UVC in multiple experimental systems.

Similarly, recent work has demonstrated substantial 222-nm activity against multiple antibiotic-resistant healthcare-associated organisms, including resistant Staphylococcus, Enterococcus, Enterobacterales, A. baumannii and P. aeruginosa.

However, resistance to antibiotics should not be confused with inability to develop altered UV tolerance. Microorganisms vary naturally in pigment production, DNA repair mechanisms, sporulation, aggregation and biofilm formation, all of which can modify irradiation response.

Therefore, UVC should be considered a complementary antimicrobial modality rather than a guaranteed “resistance-proof” treatment.

4. Discussion

The accumulated experimental evidence demonstrates that ultraviolet irradiation can substantially decrease the viability of major pathogens associated with purulent wounds. The clearest data concern S. aureus, MRSA and P. aeruginosa, although meaningful activity has also been reported against multidrug-resistant A. baumannii, Enterobacterales and C. albicans.

Conventional 254-nm UVC established the principle that light-based decontamination can modify the biological outcome of infected wounds. In mouse models, reducing P. aeruginosa burden was associated with improved survival, while reduction of S. aureus burden was associated with accelerated wound healing. These are important findings because they move the concept beyond simple agar-plate disinfection.

However, conventional UVC exposes host tissue to the same fundamental photochemistry responsible for microbial inactivation. This limits the acceptable therapeutic window.

Far-UVC represents a potentially important advance because the relationship between microbial dimensions and tissue penetration creates an opportunity for selective antimicrobial action. The 222-nm MRSA experiments demonstrated that significant bacterial reduction can occur without the same impairment of re-epithelialization produced by 254-nm irradiation. Human pressure-ulcer data now provide preliminary translational support for this concept.

Nevertheless, several limitations prevent premature clinical adoption.

First, dose reporting is not yet standardized. Studies use different units, irradiance levels, distances, exposure times and devices.

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Second, superficial bacterial counts may underestimate organisms present in deeper tissue.

Third, wound microbiology is often polymicrobial, whereas most laboratory experiments evaluate a single species.

Fourth, laboratory strains may behave differently from clinical isolates embedded in mature host-derived biofilms.

Fifth, a decrease in CFU does not necessarily demonstrate eradication of infection.

Finally, long-term safety data for repeated therapeutic exposure of damaged human tissue remain limited.

The recent randomized clinical evidence with 222-nm irradiation is encouraging because it suggests that microbiological reduction may translate into measurable improvement in wound characteristics. However, a single-center trial cannot establish optimal dosing across diabetic wounds, burns, surgical-site infections, pressure injuries and traumatic purulent wounds.

The most rational future role of UVC may therefore be as an adjunct to established wound management: debridement, drainage when required, appropriate systemic or topical antimicrobial therapy, restoration of perfusion, glycemic management, pressure relief and modern wound dressing.

4.1. Future research directions

Future studies should standardize wavelength, spectral purity, irradiance, radiant exposure, source-to-wound distance and treatment frequency. Microbiological assessment should include quantitative CFU measurements before and after irradiation, species identification, antimicrobial susceptibility profiles and biofilm status.

For experimental purulent-wound models, particularly useful endpoints would include bacterial CFU/g tissue, wound-surface CFU, histological inflammatory score, neutrophil infiltration, re-epithelialization, granulation tissue thickness, angiogenesis and time to wound closure.

Comparative experiments between 222-, 254- and 275-nm irradiation under identical microbiological conditions would be particularly valuable.

Another priority is to investigate whether UVC treatment modifies microbial virulence, antibiotic susceptibility or subsequent biofilm formation among surviving organisms.

5. Conclusion

Ultraviolet irradiation possesses substantial antimicrobial activity against key microorganisms involved in purulent wound infection, including Staphylococcus aureus, MRSA, Pseudomonas aeruginosa, multidrug-resistant Acinetobacter baumannii and selected fungal pathogens.

Conventional 254-nm UVC can significantly reduce bacterial burden and improve outcomes in experimental infected wounds, but host-cell DNA injury limits repeated

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clinical exposure. UVC-LED systems near 275 nm demonstrate strong antimicrobial potential but similarly require careful tissue-safety evaluation.

Filtered 222-nm far-UVC currently represents the most promising wavelength for direct therapeutic investigation because it combines broad microbial inactivation with relatively limited penetration into mammalian tissue. Experimental MRSA models and emerging human pressure-injury studies support this concept.

Nevertheless, UVC should not currently be considered a replacement for surgical debridement, drainage or appropriate antimicrobial therapy in deep purulent infection. Its greatest potential is as a locally delivered adjunct for reducing superficial microbial burden, particularly in chronic or multidrug-resistant wound infection.

Future well-designed experimental and clinical studies are required to define microorganism-specific doses, optimize treatment frequency, evaluate effects on biofilms and establish long-term tissue safety.

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