



RESEARCH ARTICLE

Germicidal potential and skin compatibility of an innovative UVC phototherapy device emitting at 234 nm

Zheng Tang¹ | David Welch¹ | Manuela Buonanno¹ | Mark Gerber² | David J. Brenner¹

¹Center for Radiological Research, Columbia University Medical Center, New York, New York, USA

²Spectrum Medical Technologies, Vero Beach, FL, USA

Correspondence

David Welch, Center for Radiological Research, Columbia University Irving Medical Center, 630 West 168th Street, New York, NY 10032, USA.

Email: dw2600@cumc.columbia.edu

Funding information

Open Philanthropy / Good Ventures Foundation; Empire State Development; Far UV Foundation; Columbia University Urban Tech Award; National Institute for Occupational Safety and Health, Grant/Award Number: R01OH012312

Abstract

Chronic wounds are a major healthcare issue affecting more than 10 million Americans each year, with a 5-year survival similar to cancer and costing the healthcare system billions of dollars annually. Current solutions, such as antiseptics and antibiotics, can be toxic to cells or contribute to the development of antibiotic-resistant strains of bacteria. Exposure to germicidal ultraviolet radiation (GUV) at 254 nm has been reported as an effective method for chronic wound management. However, concerns about the health hazards from exposure to 254 nm radiation have limited its use for wound management applications. In contrast, wavelengths of ultraviolet radiation in the range of 200–235 nm have exhibited similar germicidal ability but with a lower penetration range in tissue, potentially making those wavelengths better suited for chronic wound disinfection. In this study, a novel phototherapy device emitting principally at 234 nm was used to assess the killing efficacy against Methicillin-resistant *Staphylococcus aureus* (MRSA). Potential health hazards from exposure using the device were evaluated using a 3D human skin model. The ultraviolet exposure device tested in this study shows promise for effective decontamination of chronic nonhealing wounds without associated health hazards.

KEYWORDS

antimicrobial, cyclobutane pyrimidine dimers, far-UVC, LED, MRSA, surgical site infection, ultraviolet radiation

INTRODUCTION

Surgical site infections (SSI) occur at the skin and tissue around an incision, usually involving bacterial infection

and leading to a chronic wound if wound management is ineffective. Patients with vascular diseases, which can result from diabetes, autoimmune diseases, and trauma, are especially at risk. A wound is classified as chronic

Abbreviations: CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*; CFU, colony formation units; CPD, cyclobutane pyrimidine dimers; DNA, deoxyribonucleic acid; GUV, germicidal ultraviolet radiation; HA-MRSA, hospital-acquired methicillin-resistant *Staphylococcus aureus*; H_{LEL} , lamp exposure limit; LED, light emitting diode; Lp Hg, low-pressure mercury lamp; MRSA, methicillin-resistant *Staphylococcus aureus*; OD₆₀₀, optical density at 600 nm; PBS, phosphate-buffered saline; RIP, RNAPIII inhibiting peptide; SSI, surgical site infections; TSA, tryptic soy agar; UVC, ultraviolet-C.

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if it has a loss of anatomical and functional integrity 90 days after its onset.¹ Chronic wounds are considered a global healthcare issue and represent a significant burden to both patients and healthcare providers. In the United States, chronic wounds affect more than 10 million people each year, with a 5-year survival similar to cancer,² and the estimated treatment cost is \$25 billion annually.^{3,4}

The standard therapeutic options for chronic wound management include wound dressings (hydrogels, alginates), external devices (negative pressure), drugs/pharmaceuticals (antibiotics, anti-inflammatory agents), and biological approaches (recombinant growth factors)⁵ and surgery. Antibiotics and antiseptics are the most prominent treatment methods for the management of chronic wounds.^{5,6} Systemic antibiotic use is usually combined with local antiseptics to prevent wounds from being infected. However, the use of local antibiotics may not be fully effective due to their limited concentrations in situ.⁶ Furthermore, antibiotic treatments may contribute to the establishment of resistant strains, such as methicillin-resistant *Staphylococcus aureus* (MRSA).⁶ For example, the use of β -lactam antibiotics and quinolones has been associated with an increase in MRSA transmission.⁷

The common disinfectants for presurgery skin disinfection usually include antiseptics (e.g., 1% povidone-iodine, 0.25% acetic acid, 3% hydrogen peroxide, and 0.5% sodium hypochlorite)⁸ and chlorhexidine. However, it has been reported that antiseptics may have potential toxicity to fibroblasts,^{8,9} and chlorhexidine might delay the wound healing process.¹⁰ Additionally, the use of disinfectants cannot avoid the possibility of infections caused by pathogens brought by air flow and alighting into the surgical incision site. Likewise, the commonly used disinfection treatments for chronic wound infections, like antibiotic drugs, are not adequate as they compromise the vascular system, which leads to decreased blood flow.^{6,11}

Hospital-acquired methicillin-resistant *Staphylococcus aureus* (HA-MRSA) is one of the most common bacteria found in patients with open wounds or taking multiple antibiotics. The major types of infection include bacteremia, pneumonias, and wound infection.¹² The lower sensitivity of HA-MRSA to antibiotics compared to the community-acquired methicillin-resistant *S. aureus* (CA-MRSA) makes it more difficult to design an effective treatment. Silver products are widely used as topical agents, and MRSA is sensitive to silver. However, the role of silver in preventing MRSA reservoir growth remains unknown.¹² In addition, biofilm can develop in chronic wounds and usually exists as a combination of bacteria and fungi, which impede the healing process and the

treatment.¹³ It was reported that well-developed biofilm can be formed in chronic wounds within 10 hours and exist while the wound stays exposed.^{14–16} After pathogenic bacteria such as MRSA enter the open wound, the bacteria disrupt the balance of microflora of the skin and replace the commensal species.^{14,17,18} The microenvironments created by biofilm also limit the penetration of antibiotics and antiseptics.¹⁴ According to a recent review, bacteriophages, metal nanoparticles, (RIP), synthesized RIP derivatives, proteinase K, and hamamelitannin were reported as promising therapies to eradicate MRSA.¹⁹ However, there is a need for a direct approach that would disinfect the wound at its inception.

Germicidal ultraviolet radiation (GUV) is widely used to kill or inactivate pathogens. Traditional germicidal applications of GUV utilized low-pressure mercury lamps, which principally emit 254 nm light. At exposure doses typically required for disinfection with 254 nm light, these GUV sources are a human health hazard, causing damage to the skin (erythema) or the eyes (photokeratitis). An emerging technique for GUV is the use of far-UVC, defined as wavelengths from 200 to 235 nm.^{20,21} Most of the currently available far-UVC devices are KrCl excimer lamps, which emit primarily at 222 nm and are equipped with an optical filter to reduce longer wavelength emissions that are characteristic of this excimer. 222 nm emitting devices are currently available as both overhead fixtures to expose entire room volumes^{22–24} and as handheld devices.²⁵ KrCl excimer lamps have previously been tested for disinfection of SSI.^{26–28}

Gas discharge lamps such as mercury lamps, xenon lamps, or KrCl excimer lamps are common sources for GUV applications because of their high light intensity, large illumination area, and wide wavelength range.^{29–32} However, compared to light-emitting diode (LED) lamps, they are bulkier and easier to break, require high voltages to operate, can release large amounts of heat during operation, and are limited in their output spectrum.^{31,32} Furthermore, the release of mercury vapor from a broken lamp could cause potential damage to the environment and to the human immune and reproductive systems after long-term exposure.^{32–34} LEDs have many advantages as light sources, such as a compact design, simpler electronic operation, no requirement for warm-up time, and a long lifespan. Furthermore, LEDs can be produced to emit specific wavelengths, making them a competitive alternative light source for GUV.^{31,32,34} The comparison of emission spectrum and light intensity between GUV LEDs and traditional GUV light sources was completed in previous studies, which supported the potential for LEDs to be used as an alternative light source for generating GUV.^{32,35–38}

Like conventional GUV at 254 nm, the inactivation mechanisms for far-UVC on bacteria are believed to be through direct DNA as well as protein damage.^{21,39} In the case of UV-sensitive tissues such as skin and eyes, the high absorption of far-UVC wavelength by the proteins in the uppermost layers of the tissues^{40–42} significantly limits their penetration into the tissue.⁴⁰ Therefore, far-UVC devices could potentially be used on skin wounds to inactivate pathogens while being minimally damaging for the skin.

In this paper, we describe a novel UVC phototherapy platform that uses LEDs with a peak emission at 234 nm. We assessed the device's germicidal efficacy against MRSA in solution and its safety in terms of induction of DNA damage in an in vitro skin tissue model.

MATERIALS AND METHODS

LED exposure system, characterization, and dosimetry

The phototherapy platform features 92 LEDs (SSL™ prototype 2024.1), which were manufactured by Crystal IS (Green Island, NY) and emit with a center wavelength of 235 nm, an optical bandpass filter with a peak transmission at 234 nm, a power supply with rechargeable lithium-ion batteries, an optical head unit (R&D Technologies North Kingston, RI), and a 3D-printed base, including a base ring (R&D Technologies North Kingston, RI). After the addition of filtration (Edmund Optics, Barrington, NJ), the center wavelength of the emission spectrum of the device is 234 nm. The emission spectrum of the device was measured using a BTS2048-UV spectroradiometer (Gigahertz-Optik, Inc., Amesbury, MA) and is

plotted in Figure 1. Optical power measurements used a Hamamatsu C9536 power meter with an H9535-222 sensor head (Hamamatsu Corporation, Bridgewater, NJ). The lamp exposure limit, H_{LEL} , was determined based on the measured spectrum.⁴³ The H_{LEL} of the filtered LEDs using the ACGIH skin limit is 115 mJ/cm², the H_{LEL} using the ACGIH eye limit is 34.8 mJ/cm², and the H_{LEL} using the ICNIRP exposure limits is 15.3 mJ/cm².^{44,45}

Also used for the skin safety tests was a low-pressure mercury lamp (Mineralight XX-15S, UVP, Upland, CA), which principally emits at 254 nm.

Exposures using the LED were performed with the LEDs 2.5 cm away from the target. The average irradiance during an exposure was 40.5 μ W/cm². Exposures using the 254 nm lamp were performed with an irradiance of 500 μ W/cm².

Survival of MRSA

MRSA (USA300, multilocus sequence type 8, clonal complex 8, staphylococcal cassette chromosome mec type IV) was kindly provided by Dr. Anne-Catrin Uhlemann at Columbia University in New York. USA300 isolates have quickly developed resistance to antimicrobial agents, thereby becoming the cause of community- and hospital-acquired invasive diseases such as bacteremia, endocarditis, and pneumonia.⁴⁶ Fresh colonies of MRSA were obtained by picking a single colony from a stock plate and mixing it well in a glass bottle containing 40 mL tryptic soy broth (TSB). After being cultured overnight, shaking at 140 rpm at 35°C, the MRSA stock was collected in a 50 mL tube, then centrifuged at 3374g for 12 min. The supernatant was discarded, and 1 mL phosphate-buffered saline (PBS) was added to resuspend the collected pellet. After

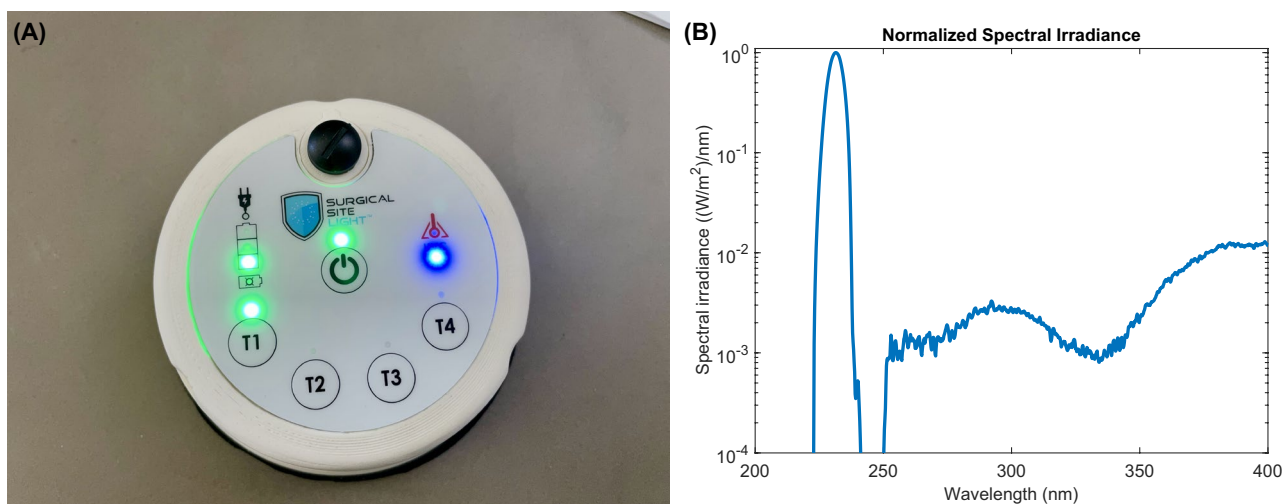


FIGURE 1 (A) A photograph of the tested device with indicator LEDs. (B) Spectrum emitted from the device tested in this study. The center wavelength of the emission spectrum of the device is 234 nm.

measuring the optical density at 600 nm (OD_{600}), PBS was used to adjust the OD_{600} of the resuspended MRSA to 0.6. 1 mL of MRSA stock at $OD_{600}=0.6$ was added into a 30 mm petri plate and exposed to 0, 4, 6, 10, or 20 mJ/cm² of far-UVC exposure using the LED device. After exposure, the MRSA solution was serially diluted, and 15 μ L of each dilution was plated on 1.5% tryptic soy agar (TSA) by spreading. Plates were incubated at 37°C in the incubator for 24 h before counting colonies for assessment of the colony formation units (CFU/mL).

The survival fraction (S) of MRSA exposed in solution to 234 nm LEDs was defined by calculating the ratio of colonies of irradiated cells (CFU/mL at each UV dose) to colonies in zero dose control (CFU_{controls}): $S = \text{CFU}_{\text{UV}} / \text{CFU}_{\text{controls}}$. Survival values were calculated for each repeat experiment and natural log ($\ln$) transformed to bring the error distribution closer to normal.⁴⁷ Using this approach, MRSA survival [S] was described by first-order kinetics according to the equation: $\ln [S] = -k \times D$, where k is the UV inactivation rate constant or susceptibility factor (cm²/mJ) and D is the dose. The regression was performed with the intercept term set to zero, representing the definition of 100% relative survival at zero UV dose. The data at zero dose, which by definition represent $\ln [S] = 0$, were not included in the regression.

UV-associated premutagenic DNA lesions in a 3D human skin model

We used a 3D human skin model, EpiDerm-FT (MatTek Corp., Ashland, MA), consisting of stratum corneum and 8–12 human cell layers to reproduce human epidermis and dermis.⁴⁸ We measured induction of the most abundant premutagenic DNA lesions in the epidermis, cyclobutane pyrimidine dimers (CPD), in 3D human skin constructs 30 min after exposure to the $D_{99.9}$ determined for MRSA using this LED system, using the immunohistochemical approach previously described.⁴⁹

RESULTS

Survival of MRSA

Results for the inactivation of MRSA in PBS solution by the LED device are plotted in Figure 2. The MRSA inactivation susceptibility constant was $k = 0.43 \text{ cm}^2/\text{mJ}$ (95% Confidence Interval: 0.40, 0.45), and the D_{90} , which is the UV dose that inactivates 90% of the exposed bacteria, was calculated as $D_{90} = -\ln [1 - 0.90] / k = 5.38 \text{ mJ}/\text{cm}^2$. Similarly, $D_{99.9}$, or the dose that results in a 3-log reduction of survival, was calculated as $16.14 \text{ mJ}/\text{cm}^2$.

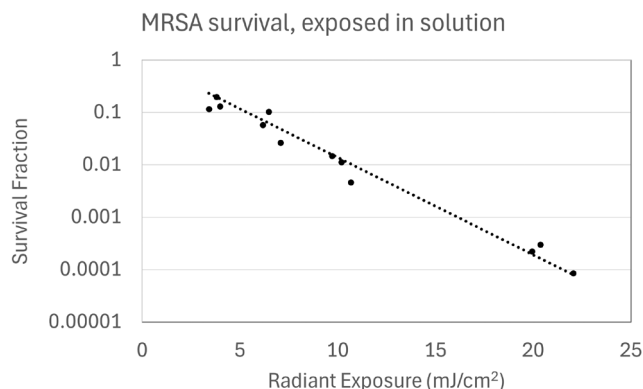


FIGURE 2 Survival of methicillin-resistant *S. aureus* (MRSA) exposed in solution to different doses of 234 nm LEDs. Fitting the results to an exponential model estimates a susceptibility k value of $0.43 \text{ cm}^2/\text{mJ}$.

DNA damage in a 3D human skin model

To assess induction of damage on skin from the 234 nm LED device, we exposed 3D human skin tissue models to the dose that killed 99.9% of the MRSA ($D_{99.9} = 16.14 \text{ mJ}/\text{cm}^2$). Nuclei positive for CPD typically appear black or brown in color using this assay.^{50,51} However, samples that had been exposed to $16.14 \text{ mJ}/\text{cm}^2$ and fixed 30 min after exposure (Figure 3, middle panel) appeared similar to unirradiated controls (Figure 3, left panel), indicating that at this dose the 234 nm LED device did not induce CPDs within this skin tissue model (Figure 3). In contrast, CPDs were detected in epidermal keratinocytes (appearing as black nuclei) of skin models exposed to $20 \text{ mJ}/\text{cm}^2$ from a low-pressure mercury lamp (Figure 3, right panel).

DISCUSSION

Our findings suggest that 234 nm induces a 90% reduction in MRSA survival with a radiant exposure dose of $5.38 \text{ mJ}/\text{cm}^2$ and a 99.9% reduction with a radiant exposure dose of $16.14 \text{ mJ}/\text{cm}^2$. This antimicrobial dose of far-UVC did not produce DNA photodamage within a human skin tissue model.

It was suggested that UVC can eradicate pathogens selectively and help with the wound healing process with the appropriate doses, without damaging the viability of mammalian host cells.⁵² However, the studies on the application of UVC in curing chronic wounds are limited to in vitro and ex vivo models. The lower penetration range and better absorption in the tissues of 234 nm light make it a promising candidate for chronic wound decontamination.⁴¹ Our findings suggested that 234 nm light has germicidal ability against MRSA.

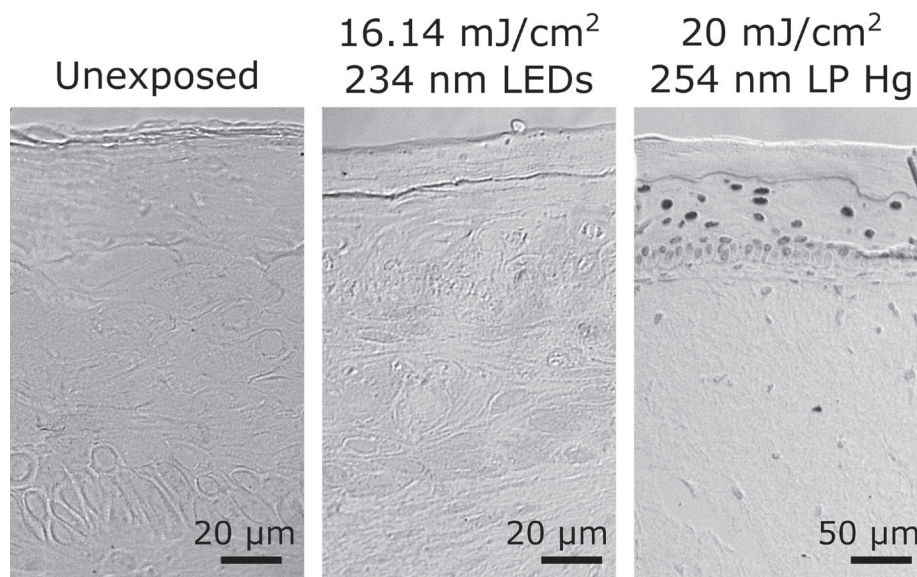


FIGURE 3 No cyclobutene pyrimidine dimers (CPD) were detected in 3D human skin tissue models 30 min after exposure to the dose that kills 99.9% of the MRSA bacteria ($D_{99.9} = 16.14 \text{ mJ/cm}^2$). On the other hand, CPD was detected in epidermal keratinocytes (appearing as black nuclei) of skin tissue models exposed to a similar dose (20 mJ/cm^2) from the low-pressure mercury lamp (254 nm Lp Hg).

The penetration range does vary slightly among far-UVC wavelengths. For example, at a given dose, 234 nm can penetrate further in the epidermis than 222 nm.⁴¹ The safety for exposed skin and the potential for reducing pathogens make the 234 nm light a promising candidate for skin disinfection during surgery and within chronic wounds. Previous studies have explored the germicidal potential of 233 nm light. In sodium chloride solution, 233 nm can cause a similar reduction of a $\log_{10}$ reduction (LR) of MRSA DSM 11822 in comparison to 254 nm at the same dose.³⁰ In the consideration of skin safety, compared to the commonly used germicidal 254 nm, 233 nm light is more highly absorbed before reaching the basal cell layers of the skin. The CPD damage is only observed at the superficial layer of the epidermis.³⁰ Although our study provides valuable insights into the potential of 234 nm in chronic wound management, there are some limitations that can be improved in the future. A current limitation with the tested device is the operational runtime. The device heats while operating and automatically shuts off after about 7 min of continuous usage. We found that a dose of about 20 mJ/cm^2 could be delivered within this operational window, which is well above the dose required for a 3-log reduction in MRSA in solution. The exposure window area is another limitation. The device exposes an 80 mm diameter circular area, which may restrict the application of this device on larger chronic wounds. Additional evaluation of the performance of this device against biofilms associated with MRSA is also needed. The complicated structure and components of the biofilm may limit the

penetration and absorption of 234 nm and thus its overall germicidal capacity within that environment. Finally, in vivo safety evaluation of this 234 nm emitting device should be performed to assess its potential damage to skin cells and tissues.

Future studies with this device could include additional evaluation of battery life, heating performance, compatibility with different chronic wound sizes, and further evaluation of the safety of 234 nm exposure. Additional studies can provide improved guidance for the application of this innovative UVC phototherapy platform for the decontamination of chronic nonhealing wounds.

ACKNOWLEDGMENTS

This work has been supported in part by the Open Philanthropy/Good Ventures Foundation, the Empire State Biodefense Commercialization Fund, the Far UV Foundation, a Columbia University Urban Tech Award, and CDC NIOSH R01OH012312.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

David Welch  <https://orcid.org/0000-0003-2784-496X>

Manuela Buonanno  <https://orcid.org/0000-0002-3455-3602>

[org/0000-0002-3455-3602](https://orcid.org/0000-0002-3455-3602)

David J. Brenner  <https://orcid.org/0000-0002-0926-1210>

[org/0000-0002-0926-1210](https://orcid.org/0000-0002-0926-1210)

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How to cite this article: Tang Z, Welch D, Buonanno M, Gerber M, Brenner DJ. Germicidal potential and skin compatibility of an innovative UVC phototherapy device emitting at 234 nm. *Photochem Photobiol.* 2026;102:411-417. doi:[10.1111/php.70016](https://doi.org/10.1111/php.70016)