

RESEARCH BRIEFS

Patient Room Decontamination against Carbapenem-Resistant *Enterobacteriaceae* and Methicillin-Resistant *Staphylococcus aureus* Using a Fixed Cycle-Time Ultraviolet-C Device and Two Different Radiation Designs

The contaminated surface environment of hospital rooms has been demonstrated to be a source of patient-to-patient transmission of multidrug-resistant organisms (MDROs), including methicillin-resistant *Staphylococcus aureus* (MRSA).^{1,2} MDROs can survive on surfaces for days to weeks (for months in some cases).³ Following terminal room cleaning and disinfection, frequent contamination by these pathogens has been reported.^{1,2} Patients admitted to a room where the previous occupant was also colonized or infected with an MDRO are at substantial risk for acquisition of this MDRO.⁴ For these reasons, “no-touch” methods of room disinfection have been evaluated, including devices that emit ultraviolet (UV) light or produce hydrogen peroxide vapor.⁵ Use of UV devices has been demonstrated to reduce healthcare-associated pathogens.⁵

Carbapenem-resistant *Enterobacteriaceae* (CRE) are a growing problem in the United States and worldwide.⁶ CRE infections are difficult to treat and have been associated with substantial mortality. Frequent contamination of room surfaces near CRE carriers has been described.⁷ For this reason, we assessed the efficacy of a UV-C device to inactivate MRSA and CRE. We also assessed whether 2 cycles of the device (in which 1 cycle was completed on the right side of the bed and 1 cycle on the left side of the bed) were superior to a single cycle (in which the device was placed at the foot of the bed).

In this study, we evaluated the Optimum UV (Clorox, Pleasanton, CA), a fixed cycle-time UV-C (254 nm) device with four 157-cm lamps in an unoccupied patient room (24.76 m²). To assess the efficacy of 2 different decontamination methods, the UV-C device was placed either at the end of the bed in the center of room in a single cycle (design A) or at both sides of the bed in the center of room with a single cycle at the right side and another cycle at left side (design B). The cycle time of the device for room decontamination was fixed at 5 minutes. Each design was tested in triplicate. Locations where test Formica sheets were placed were categorized as the UV-C direct group (laser point visible on site) or the UV-C indirect group (laser point not visible on site). Testing was performed using clinical isolates of MRSA and carbapenem-resistant *Klebsiella pneumoniae* (CRKP). A 10–15 µL inoculum containing ~10⁶–10⁷ test organisms per replicate organism

detection and counting (Rodac) template was spread separately on each Formica sheet using a sterile glass hockey loop. Inoculated Formica sheets were placed and attached with tape in the following 10 locations throughout the room for both designs: (1) the back of the computer (indirect/indirect); (2) the side of the sink counter (indirect/direct); (3) the vertical end of the foot of the bed (direct/indirect); (4) the horizontal top of the bed (direct/direct); (5) the vertical back of the visitor’s chair (indirect/indirect); (6) the horizontal counter in front of the refrigerator (direct/direct); (7) the vertical end of the head of the bed (indirect/indirect); (8) the vertical side of the bedside table facing the window (indirect/indirect); (9) the horizontal top of the toilet rim in the bathroom (indirect/indirect); and (10) the horizontal top of the couch table (direct/direct). After UV-C cycle completion, each Formica sheet was cultured using Rodac plates containing Day-Engley (DE) neutralizing agar. These plates were incubated aerobically at 37°C for 48 hours for CRKP and MRSA. The colony-forming units (CFU) of the test organisms on each plate were then counted. When Rodac plates had confluent growth, the number of CFUs per plate was set at 300 for analysis. Statistical significance was determined by paired *t* test; *P* < .05 was considered significant.

Overall, in an unoccupied patient room, we observed a 5.01-log₁₀ reduction using design A and a 5.87-log₁₀ reduction using design B for decontamination of CRKP (*P* = .0003). We observed a 4.61-log₁₀ reduction and a 5.06-log₁₀ reduction for MRSA (*P* = .0418) (Table 1). At UV-C direct sites, we measured an additional 0.87-log₁₀ reduction using design B for CRKP (*P* = .0028) and an additional 0.55-log₁₀ reduction for MRSA (*P* > .05) compared with design A. When we assessed the correlation of log₁₀ reduction (in direct line of sight) to distance, there was a strong correlation (*r* = –0.826, *P* = .0009 for CRKP and *r* = –0.842, *P* = .0006 for MRSA); however, we detected a ≥ 4.89-log₁₀ reduction for CRKP and a ≥ 4.19-log₁₀ reduction for MRSA at the farthest distance (2.69 m). At UV-C indirect sites, we measured an additional 0.86-log₁₀ reduction using design B for CRKP (*P* = .0002) and an additional 0.38-log₁₀ reduction for MRSA (*P* = .0113) compared to design A. The frequency of too-numerous-to-count (TNTC) cultures was as follows: MRSA 42% (25 of 60) and CRKP 32% (19 of 60). Data for microbial inactivation without TNTC results are shown in the Supplemental Material.

Our study demonstrated that the fixed-time UV-C device evaluated in this study was effective in 5–10 minutes in eliminating >5-log₁₀ MRSA and CRKP when the surfaces were in direct line of sight of the UV-C device and >4-log₁₀ MRSA and CRKP when the surfaces were in indirect line of sight. These results are similar to those we have previously published.⁸ While the use of 2 cycles of the device with placement at either side of the bed for 1 cycle each often

TABLE 1. Microbial Reduction in Formica Surfaces Experimentally Contaminated with Multidrug-Resistant Pathogens Using a Fixed Cycle-Time Ultraviolet-C (UV-C) Device and 2 Different Radiation Designs

	Carbapenem-Resistant <i>K. pneumoniae</i> ^a		Methicillin-Resistant <i>S. aureus</i>	
	Design A	Design B	Design A	Design B
UV-C direct sites (n = 24)	5.74 (5.24–6.23) (n = 12)	6.61 (6.33–6.88) (n = 12)	5.27 (4.71–5.83) (n = 12)	5.82 (5.38–6.25) (n = 12)
UV-C indirect sites (n = 36)	4.53 (4.37–4.69) (n = 18)	5.39 (4.99–5.78) (n = 18)	4.17 (4.17–4.17) (n = 18)	4.55 (4.25–4.85) (n = 18)
Total (n = 60)	5.01 (4.71–5.31) (n = 30)	5.87 (5.54–6.21) (n = 30)	4.61 (4.32–4.90) (n = 30)	5.06 (4.73–5.39) (n = 30)

^aValues are shown in mean log₁₀ reductions (95% confidence interval) (No. of samples). Quantitated inoculum was 6.93-log₁₀ per Rodac template for carbapenem-resistant *K. pneumoniae* and 6.65-log₁₀ per Rodac template for methicillin-resistant *S. aureus*.

Microbial reduction was calculated by subtracting the number (log₁₀) of test organisms measured on UV-C cycle completion from quantitated inoculum (log₁₀). For design A, the UV-C device was placed at the end of the bed in the center of room with a single cycle (total exposure time: 5 min). For design B, the UV-C device was placed at both sides of the bed in the center of room with a single cycle at the right side and another cycle at left side (total exposure time: 10 min).

resulted in significantly improved inactivation of the vegetative MDROs studied (ie, MRSA, CRKP), a single cycle was highly effective, and the difference is likely not clinically meaningful. Given the frequency of TNTC cultures, our level of microbial kill should be considered a maximum level.

We are aware of several limitations of this study. First, we only inoculated Formica sheets. Our previous study revealed that CRE survived poorly not only on fabrics but also on hard surfaces such as Formica and steel.⁹ Second, the size of our study limited our ability to perform subgroup analyses such as inactivation in direct versus indirect line of sight.

In this study, we tested a clinical CRE strain because of the global concern regarding its morbidity and mortality. Importantly, the present study demonstrated that UV-C room decontamination was effective against CRE. The role of hospital environmental surfaces in CRE transmission remains controversial: CRE was infrequently (8%) isolated from environmental surfaces in rooms housing CRE colonized/infected patients in our previous study,⁹ while CRE contamination of hospital environment was frequently (88%) identified at surrounding sites among patients with this organism by an Israeli study.⁷

In conclusion, the present data and our previous findings have demonstrated that UV-C devices can effectively decontaminate hospital surfaces contaminated with MDROs, including CRE. In this experiment, 2 cycles of a UV-C device were often statistically superior to a single cycle, but both designs are likely to be clinically effective (>2-log₁₀ reduction) because epidemiologically important pathogens measured in hospital room surfaces were <100 CFU/Rodac (<2-log₁₀).¹⁰

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SUPPLEMENTARY MATERIAL

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UV-C Light Disinfection of Carbapenem-Resistant *Enterobacteriaceae* from High-Touch Surfaces in a Patient Room and Bathroom

Carbapenem-resistant *Enterobacteriaceae* (CRE) are associated with mortality and morbidity.¹ Cleaning of high-touch surfaces (HTSs) in the near-patient environment is often suboptimal.^{2,3} Significant interest has been directed toward novel “no touch” disinfection mechanisms such as ultraviolet (UV) light for removal of multidrug-resistant organisms from patient rooms.⁴ To our knowledge, UV light as a mechanism for removal of CRE from a patient room has not been investigated. In this study, we assessed the efficacy of UV light treatment at reducing CRE in a patient room, and we sought to ascertain whether there is a difference in CRE reduction based on organism (ie, *K. pneumoniae* vs *E. coli* vs *E. cloacae*).

This study was performed in an empty patient room of The Johns Hopkins Hospital in September 2015. Using methods similar to those previously described,⁵ a template of a single contact plate (Becton Dickinson, San Jose, CA) was drawn on Formica swatches 12.7 cm × 17.8 cm (5 × 7 inches). A 10 µL aliquot of a 0.5 × 10⁷ inoculum of a single CRE type was spread onto the contact plate template using a sterile, glass,

hockey-stick-shaped spreader. The inoculated swatches were attached to a minimum of 17 high-touch surfaces (HTSs) in the patient room and bathroom. HTSs included those defined by the Centers for Disease Control and Prevention: bed rail, IV pump, vitals monitor, keyboard, over-bed tray table, call box, sink, shower curtain, and toilet seat.⁶ Each HTS was within ~8 feet of the UV device during at least 1 cycle.

The UV-C Clorox Healthcare Optimum-UV device (Clorox, Oakland CA) emits UV-C light at a wavelength of 254 nm via 157.5-cm (62-inch) maximum-output mercury lamps. This equipment was activated for 5 minutes on each side of the patient bed and 5 minutes in the bathroom (ie, a total of 15 minutes). A contact plate was then pressed onto each swatch. Plates were incubated at 37°C for 48 hours. Total colony-forming units (CFUs) were counted, and CREs were identified. This cycle was repeated for each CRE organism. Color change from yellow to dark green on device dose indicators were used to verify that UV light adequate to kill *Clostridium difficile* and methicillin-resistant *Staphylococcus aureus* was emitted. Negative controls (ie, inoculated swatches placed in a box to prevent exposure to UV) were used for all cycles. Proportions of plates with no growth of CRE (or no growth of any organism) post exposure to UV were compared using Fisher’s exact test.

A total of 133 HTSs were sampled: 34 with *E. coli*, 47 with *K. pneumoniae*, and 52 with *E. cloacae*. All negative control swatches grew organisms too numerous to count (TNTC), indicating that any reduction on other swatches was due to UV-C exposure. Only 2 of the plates (1.5%) grew any CRE after exposure to UV light: both *E. cloacae* plates grew cultures; 3 plates grew CFUs from a bedrail; and a plate from a shower curtain grew CRE TNTC. The other 131 plates did not grow any CRE, demonstrating a 6 log reduction in CRE. Seven plates (5.3%) grew non-pathogenic organisms (eg, filamentous *Bacillus* organisms).

These results show that UV light treatment was highly effective at killing CRE (*K. pneumoniae*, *E. coli*, and *E. cloacae*) on HTSs in a patient room and bathroom with 15 minutes of exposure. We found complete reduction of CRE from 25 different HTSs in the patient room and bathroom, including the IV pump, vitals monitor, call bell, keyboard, and toilet seat. The only 2 sites where we found growth of CRE (*E. cloacae*) after UV treatment were a shower curtain and a bed rail. The shower curtain swatch, which grew CRE TNTC, was between 2 curtain folds, and we hypothesize that this area was not exposed to UV light. The privacy curtain in the patient room, which was not folded had complete reduction of CRE, highlights the importance of setting the HTSs to maximize surface exposure to UV, such as straightening the curtains. We found no difference in reduction based on organism. A recent study has suggested that *K. pneumoniae* may persist in the near-patient environment longer than *E. coli* or other Gram-negative organisms.⁷ Our study shows that UV light treatment removes *K. pneumoniae* from HTSs as effectively as it removes other Gram-negative organisms.