A comparison of DNA repair and survival of *Escherichia coli* O157:H7 following exposure to both low- and medium-pressure UV irradiation

J. L. Zimmer-Thomas, R. M. Slawson and P. M. Huck

ABSTRACT

DNA repair and survival of pathogenic *E. coli* O157:H7 was investigated following exposure to ultraviolet (UV) radiation from both low-pressure (LP) and medium-pressure (MP) lamps. This study included irradiation at UV doses used in drinking water treatment and lower doses indicative of potential treatment problems. Immediately following UV exposure, an average log inactivation of 4.5 or greater was observed following all tested doses of LP (5, 8, 20 and 40 mJ/cm^2) or MP UV (5 and 8 mJ/cm^2) indicating the sensitivity of *E. coli* O157:H7 to UV irradiation. Following conditions conducive to repair, maximum photo repair occurred rapidly within 30 minutes after low doses (5 and 8 mJ/cm^2) of LP UV. The rate of repair was much higher than reported previously in non-pathogenic *E. coli* (which occurred within 2 hours). In contrast to LP UV, limited photo repair of *E. coli* O157:H7 was observed following MP UV exposure at reduced doses (5 and 8 mJ/cm^2). At these lower doses, low levels of light independant repair were observed following LP UV, but not following exposure of MP UV irradiation. This study indicates that MP UV may enhance UV disinfection of *E. coli* O157:H7 by reducing the ability to repair following non-ideal treatment conditions. Following doses used in drinking water treatment (20 and 40 mJ/cm^2), low levels of photo repair following LP UV were evident.

Key words | DNA repair, *E. coli* O157:H7, low-pressure UV, medium-pressure UV, photoreaction, UV irradiation

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INTRODUCTION

Ultraviolet (UV) radiation is a technology that is gaining acceptance as a viable drinking water treatment strategy in North America. Recent attention is primarily due to the known ability of UV to effectively inactivate the pathogenic protozoan *Cryptosporidium* at very low doses (Bukhari *et al.* 1999; Clancy *et al.* 2000; Mofidi *et al.* 2001; Zimmer *et al.* 2003).

The two UV sources predominantly used in water treatment are low-pressure (LP) and medium-pressure (MP) mercury lamps. LP UV lamps have traditionally been used in drinking water and wastewater treatment and emit almost monochromatically at a wavelength of 254 nm (Bolton 2001). More recently developed MP UV lamps emit over a broader range of wavelengths from far UV (185 nm) to infrared wavelengths (1367 nm) (Linden & Mofidi 1999).

The primary mechanism responsible for cell injury and loss of viability by UV irradiation is damage to the structure and function of DNA (Friedberg *et al.* 1995). To a lesser extent, UV radiation can affect cellular proteins, lipids and membranes (Harm 1980; Tevini 1993).

To reduce DNA damage, microorganisms may possess several processes that can allow for cell survival or repair following UV exposure (Friedberg *et al.* 1995; Thoma 1999). UV damage can be 'repaired' by direct reversal or removed and in some instances UV damage can be tolerated by the cell. Some microorganisms have the ability to directly reverse DNA damage through processes such as photoreactivation.

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This repair process involves the use of an enzyme called DNA photolyase that requires wavelengths between 300 and 500 nm (portion of UV-B and visible range) to directly reverse DNA damage (Friedberg *et al.* 1995). In contrast to photoreactivation, there are numerous light independent repair and damage tolerant processes that can allow for survival following UV exposure. For example, nucleotide excision repair is a complex repair process that involves the coordination of numerous enzymes to remove DNA damage (Friedberg *et al.* 1995). Depending on the level of damage, cells can also tolerate UV damage until repair can occur (Friedberg *et al.* 1995).

The subject of DNA repair in microorganisms following UV irradiation has been well recognized and studied since its discovery over half a century ago (reviewed in Sancar 2000). However, with the increased use of UV as a technology in drinking water treatment this subject has become a renewed area of interest, specifically with regard to potential DNA repair of environmental and waterborne pathogens. Recognizing and understanding DNA repair in pathogens, as well as indicator organisms, can help to optimize UV treatment by eliminating conditions under which repair may occur.

More utilities are considering incorporation of UV in treatment strategies for reasons other than inactivation of *Cryptosporidium* alone, such as decreasing the emphasis on chemical disinfection and increasing multiple barrier treatment processes (e.g. Protasowicki & Malley 2002). Therefore, parameter optimization can be addressed by further assessing the ability of UV to inactivate other pathogens and determine their potential to repair DNA following treatment.

Over the past few decades *E. coli* O157:H7 has emerged as a significant human pathogen. This organism has been involved in numerous foodborne outbreaks worldwide (Meng & Doyle 1998) and more recently implicated in waterborne outbreaks (Geldreich *et al.* 1992; O'Connor 2002; Olsen *et al.* 2002). A recent drinking water outbreak of *E. coli* O157:H7 in Walkerton, Ontario, Canada resulted in more than 2,300 people becoming ill and 7 deaths (O'Connor 2002). The presence of this pathogen in water identifies the need to further understand its potential tolerance to treatment. It should be noted that *E. coli* O157:H7 is not differentiated or detected in typical coliform or *E. coli* tests used for water analysis due to physiological and metabolic differences when compared to other *E. coli* strains (Geldreich *et al.* 1992; *Standard Methods* 1998; Blatchley *et al.* 2001). It is unlikely that *E. coli* O157:H7 will be identified after drinking water is treated, unless there is a specific concern related to contamination.

Research is required in order to more conclusively elucidate DNA repair and survival of the pathogen *E. coli* O157:H7 following UV irradiation. To date, no studies have investigated DNA repair in *E. coli* O157:H7 following MP UV and only limited conflicting studies have evaluated repair following LP UV exposure in water (Tosa & Hirata 1999; Sommer *et al.* 2000; Mofidi *et al.* 2002). Recent studies performed using non-pathogenic *E. coli* have indicated differences in the repair following low doses of MP and LP UV irradiation (Oguma *et al.* 2002; Zimmer & Slawson 2002). These studies showed that LP and MP UV demonstrated similar levels of inactivation, however, following MP UV exposure DNA repair was greatly reduced. Due to the previously noted physiological and metabolic differences observed between non-pathogenic *E. coli* strains and *E. coli* O157:H7, potential differences in repair ability following UV exposure need to be further evaluated.

The purpose of this study was to compare the DNA repair potential and survival of *E. coli* O157:H7 following both MP and LP UV exposure. Repair was investigated following low delivered UV doses indicative of compromised treatment (5 and 8 mJ/cm^2), as well as following higher UV doses (20 and 40 mJ/cm^2) more commonly used in drinking water treatment. DNA repair of *E. coli* O157:H7 was also compared to that of a non-pathogenic *E. coli* strain previously studied.

METHODS

E. coli O157:H7

A strain of *E. coli* O157:H7 (ATCC 43895, Manassas, VA) originally implicated in a hemorrhagic colitis outbreak was used in this study. This particular strain produces both Shiga toxin 1 and Shiga toxin 2.

To ensure sufficient cell density, *E. coli* O157:H7 were grown under optimal conditions in trypticase soy broth

(VWR Canada, Mississauga, ON) at 37°C in an oscillating waterbath shaker (Gyrotory Water Bath Shaker, Model G76, New Brunswick Scientific, Edison, NJ). A 20–22 hour culture in stationary phase was used for experimental purposes to represent the growth phase most typically observed in the environment and the most resistant stage of growth (Mofidi *et al.* 2002).

Prior to each experiment, a suspension of *E. coli* O157:H7 was centrifuged (Sorvall FA-Micro, DuPont Canada, Mississauga, ON) at 400 × g for 8 minutes and the supernatant was aseptically drawn off. The pellet was resuspended in 0.01 M phosphate buffered saline (PBS) to obtain a whole cell concentration ranging from 10^7 to 10^9 cells/ml. Every effort was made to ensure consistency in starting concentrations. When replicate experiments could not be performed from the same starting cell batch, it is noted in the text. The sample was vortexed (Fisher Genie 2, VWR Canada, Mississauga, Canada) and 5 ml were added directly into a 50 mm plastic Petri dish (VWR Canada, Mississauga, ON). Prior to irradiation, a portion of the *E. coli* O157:H7 suspension was removed, serially diluted and plated in triplicate to determine the initial cell concentration (expressed as colony forming units (cfu) per ml). All samples were processed using the standard plate count technique (*Standard Methods* 1998).

UV unit and dose calculations

A bench-scale collimated beam apparatus (Calgon Carbon Corp., Pittsburgh, PA) with an interchangeable LP (12 W) or MP (1 kW) mercury UV lamp was used to irradiate the cells in this study. A polyvinyl chloride (PVC) collimating tube (93 cm) which aids in focusing the UV beam on the sample was used in all experiments. Samples to be irradiated were placed on a magnetic stir plate directly below the collimating tube for irradiation.

The dose calculation for LP and MP UV irradiation was carried out as previously described (Zimmer & Slawson 2002; Zimmer *et al.* 2003). Briefly, the UV dose (mJ/cm^2) was determined by multiplying the average irradiance (mW/cm^2) in the sample liquid by the irradiation time (s). These doses were calculated using software provided by Bolton Photosciences (Ayr, Ontario, Canada). Irradiance for both lamp types was measured using a radiometer (International Light, Model IL 1700, equipped with a SED 240 UV detector, Newburyport, MA).

Numerous factors must be included to calculate an accurate dosage for both LP and MP lamps. For the polychromatic MP UV lamp, factors included in the dose calculation were: the variation in the irradiance across the Petri dish (Petri factor), the attenuation of the beam within the liquid containing the organism (water factor), the reflection of UV at the liquid surface (reflection factor = 0.975), and the variation in the sensor sensitivity to wavelength (sensor factor = 1.206). Also a germicidal (weighted) factor was applied to the MP dose calculation (Bolton 2000). This "weighted" factor accounts for the relative DNA absorbance efficiency (being 1 at 260 nm) of the polychromatic MP UV lamp emissions. The weighted doses are approximately 25% lower than the unweighted doses. Weighting the MP dosage is thought to allow for better comparison to emissions from LP UV lamps. It should be noted that most studies use unweighted doses for MP UV irradiation. Both weighted and unweighted doses are shown in Table 1, however, only weighted doses will be discussed throughout.

For LP UV lamps, factors incorporated into the dosage calculation included: a Petri factor, reflection factor and water factor.

E. coli O157:H7 UV irradiation

The *E. coli* O157:H7 suspension in the Petri dish was placed under the collimating tube of the UV unit. With the Petri dish lid removed and while slowly mixing, the sample was exposed to UV radiation for selected time periods to

Table 1 | Log inactivation of E. coli O157:H7 following LP and MP UV irradiation

	UV dose (mJ/cm^2)			
UV lamp source	Unweighted	Weighted	No. of expts	Log inactivation (± st dev)
LP	5	N/A[a]	3	4.5 (± 0.2)
	8	N/A	5	5.1 (± 0.3)
MP	6.7	5	6	6.4 (± 0.2)
	10.6	8	5	6.6 (± 0.4)

[a]N/A – not applicable, LP doses are not weighted.

yield the desired UV dose. All irradiation experiments were carried out at room temperature.

In this study samples were irradiated at selected doses of 5, 8, 20 and 40 mJ/cm^2 from LP UV lamps and weighted doses of 5 and 8 mJ/cm^2 from MP UV lamps. The 5 and 8 mJ/cm^2 doses are lower than typically used in water treatment and were selected to favor conditions for repair. The higher doses of 20 and 40 mJ/cm^2 from LP UV were used to reflect a range of treatment doses used in some jurisdictions (DVGW 1997; NIPH 2002).

Immediately following UV exposure, the entire sample volume was collected and placed into a sterile test tube covered with foil to prevent any visible light penetration. A portion of the irradiated sample was removed, serially diluted in foil covered test tubes and plated in triplicate on trypticase soy agar (VWR Canada, Mississauga, Canada) to determine the levels of viable organisms immediately following exposure. These plates were incubated in the dark at 37°C for 24 hours.

All log inactivations were determined as the difference between the log of the initial concentration (cfu/ml) of *E. coli* O157:H7 and the log of the concentration (cfu/ml) immediately following UV exposure.

DNA repair conditions

The repair conditions were as described in Zimmer *et al.* (2003). Briefly, the remaining UV-irradiated suspension was divided and transferred into two separate plastic Petri dishes (Phoenix Biomedical Products Inc., Mississauga, Canada). One of the two dishes was covered with foil to prevent light exposure and one was exposed to photo-reactivating light at 37°C (Zimmer & Slawson 2002). A non-pathogenic strain of *E. coli* (ATCC 11229), which has previously demonstrated photoreactivation, was used as a positive control for repair (Zimmer *et al.* 2003). A control was also run to ensure that *E. coli* O157:H7 did not replicate or die-off within the test media. A minimum of three experiments were performed at each UV dose and under each set of repair conditions.

RESULTS AND DISCUSSION

E. coli O157:H7 inactivation

The average log inactivation of *E. coli* O157:H7 immediately following LP UV irradiation at doses of 5 and 8 mJ/cm^2 and weighted MP doses of 5 and 8 mJ/cm^2 are presented in Table 1. The number of trials and averages with standard deviations are shown. From Table 1 it can be observed that MP UV irradiation resulted in higher levels of inactivation when compared to LP UV irradiation at the same doses. An average of 1.5 to 2 log higher levels of inactivation were observed following irradiation with MP UV when compared to inactivation data following LP UV. The difference in inactivation between MP UV and LP UV at the same dose was significant ($P < 0.0002$).

Various differences between LP and MP UV irradiation, including the weighted dose calculation (shown in Table 1) may have contributed to higher levels of DNA damage. Further detail has been reported previously (Kalisvaart 2001; Zimmer & Slawson 2002). Following both LP and MP UV exposure distinct tailing in the level of inactivation was observed as the dose increased. This phenomenon has been observed by other researchers (Bukhari *et al.* 1999; Hassen *et al.* 2000; Blatchley *et al.* 2001; Mofidi *et al.* 2002) and may be related to factors such as a higher initial concentration of organisms irradiated and subsequent shielding effects.

The levels of inactivation demonstrated by *E. coli* O157:H7 are higher than previously reported with non-pathogenic *E. coli* under the same conditions. For example, at a dose of 8 mJ/cm^2 non-pathogenic *E. coli* demonstrated 4.2 and 5.0 log inactivation following LP and MP (Zimmer & Slawson 2002), respectively, however, in this study pathogenic *E. coli* O157:H7 demonstrated a 5.1 and 6.6 log inactivation following LP and MP, respectively. Sommer *et al.* (2000) and others (Mofidi *et al.* 2002) have shown that no relationship could be assumed between UV resistance and serotype.

E. coli O157:H7 DNA repair potential

As mentioned previously, few conflicting studies have examined *E. coli* O157:H7 repair and survival following LP UV exposure (Tosa & Hirata 1999; Sommer *et al.* 2000; Mofidi *et al.* 2002). However, to date, no studies have examined the potential for *E. coli* O157:H7 to repair DNA damage following MP UV exposure. Previous work carried out by Zimmer & Slawson (2002) and Oguma *et al.* (2002) using a non-pathogenic strain of *E. coli*, demonstrated that

exposure to MP UV significantly reduced DNA repair ability compared to LP UV irradiation.

Due to the fact that treated water is transported through dark distribution systems photoreactivation is typically of less concern in drinking water treatment. However, photoreactivation may be a potential issue if UV treatment, due to space constraints, occurs prior to a process unit that allows light exposure (e.g. UV exposure prior to filtration, when filters are located in areas illuminated with fluorescent lights or windows allowing penetration of sunlight). Also, photoreactivation cannot be ruled out when water is exposed to light following distribution (e.g. consumers storing UV treated water in light exposed areas). There is also the potential issue of UV irradiation used in preparing bottled water, since it is typically exposed to light, often for extended periods prior to consumption.

Following UV irradiation, the repair potential of *E. coli* O157:H7 was assessed. Figure 1 shows the change in *E. coli* O157:H7 concentration (expressed as colony forming units (cfu) per ml) following low dose LP UV irradiation. Following lower doses of 5 and 8 mJ/cm^2, DNA photo repair in *E. coli* O157:H7 began almost immediately after light exposure and reached a maximum level of repair within 30 minutes. The maximum levels of cell repair achieved were different following each UV dose, however the time to reach maximum levels of photo repair was similar.

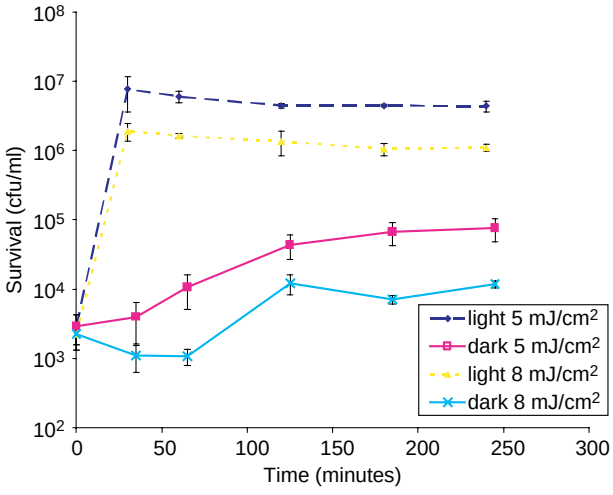

Figure 1 | Repair of *E. coli* O157:H7 following LP UV irradiation at low doses of 5 and 8 mJ/cm^2. Averages with standard deviations at 5 mJ/cm^2 are based on three separate experiments and at 8 mJ/cm^2 are based on five separate experiments.

The time to reach maximum levels of repair in *E. coli* O157:H7 was more rapid compared to a non-pathogenic strain of *E. coli* previously examined (Zimmer & Slawson 2002). An example of this comparison between *E. coli* O157:H7 and non-pathogenic *E. coli* (ATCC 11229) repair can be seen in Figure 2. Non-pathogenic *E. coli* reached maximum levels of repair in 2 hours after exposure to light compared to less than 30 minutes for pathogenic *E. coli* O157:H7.

Rapid repair of pathogenic *E. coli* O157:H7 following LP UV exposure may indicate that monitoring for an indicator organism, such as non-pathogenic *E. coli*, following UV disinfection may not be a representative indicator of the activity of other pathogens present. A recent study by Oguma *et al.* (2004) on DNA repair of *Legionella* following UV exposure highlighted a similar issue.

After experiments involving the irradiation of *E. coli* O157:H7 with LP UV, experiments were conducted exposing the same strain of *E. coli* O157:H7 to UV from MP lamps. These experiments were carried out using weighted UV doses of 5 and 8 mJ/cm^2 under the same incubation conditions. Figure 3 shows the potential repair and survival of *E. coli* O157:H7 following MP UV exposure. It can be observed in Figure 3 that following MP UV irradiation the levels of *E. coli* O157:H7 after incubation in photoreactivating light increased only slightly over the tested time period.

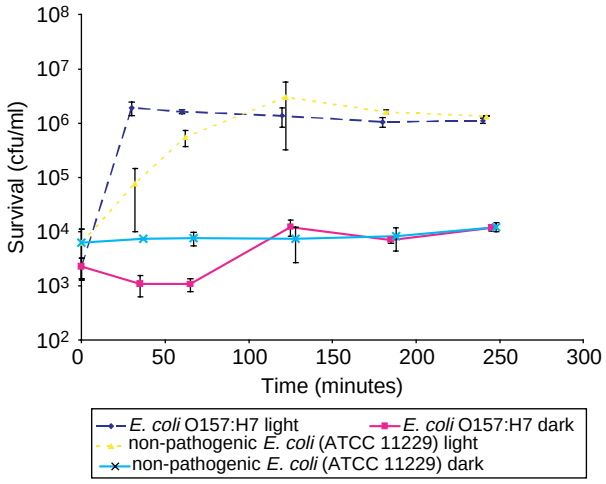

Figure 2 | Comparison of repair potential between *E. coli* O157:H7 and non-pathogenic *E. coli* (ATCC 11229) following LP UV irradiation at 8 mJ/cm^2. Averages with standard deviations are shown.

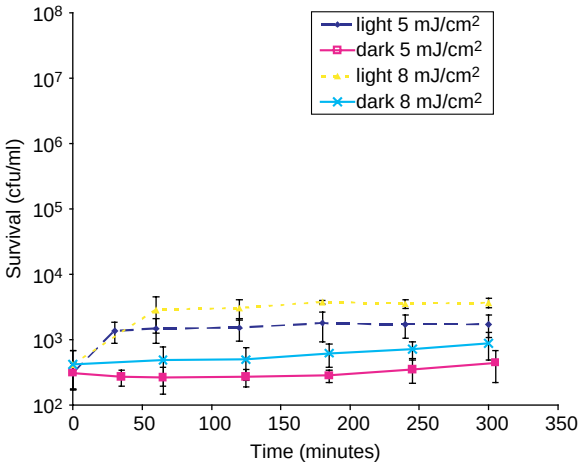

Figure 3 | Repair potential of *E. coli* O157:H7 following MP UV irradiation at weighted doses of 5 and 8 mJ/cm^2. Averages with standard deviations at 5 mJ/cm^2 are based on four separate experiments and at 8 mJ/cm^2 are based on three separate experiments.

As previously discussed (Zimmer & Slawson 2002) the reduced ability to repair following MP UV exposure may be due to inhibition from specific wavelengths that are present from MP lamps but not from LP lamps. One wavelength or a combination of wavelengths may result in irreversible physiological changes involved in the repair process or damage may be occurring to the repair enzymes themselves (Zimmer & Slawson 2002). Proteins have shown to readily absorb wavelengths below 240 nm (Harm 1980), consequently, these wavelengths emitted from MP lamps and not LP lamps may inhibit repair enzymes themselves. Recently, Oguma *et al.* (2005) indicated that repair repression following MP UV was not attributed to emissions at 230 nm, 254 nm or 300 nm alone, but rather simultaneous exposure to broad wavelengths may be involved.

The results of the present study also demonstrated that some level of light independent repair of *E. coli* O157:H7 occurred after incubation in the dark following low doses (5 and 8 mJ/cm^2) of LP UV exposure (Figure 1). No increases in light independent survival were observed in the dark following MP UV exposure at the same doses (Figure 3). Following LP UV irradiation, the levels of repair in the dark were much lower than those observed following exposure to photoreactivating light. However, as shown in Figure 2, these levels of repair are higher than observed previously in non-pathogenic *E. coli* that were exposed to identical conditions (Zimmer & Slawson 2002).

A portion of this study was intended to investigate repair and survival under ideal LP UV disinfection conditions at doses used or proposed for use in drinking water treatment (DVGW 1997; NIPH 2002). A minimum of three replicate experiments were carried out at each dose, however due to varying starting concentrations, Figure 4 shows a representative experiment at each dose. All replicate experiments demonstrated similar levels of initial inactivation and repair (normalized data for comparison are shown in Table 2). As shown in Figure 4, low levels of photo repair were evident following irradiation at these higher doses. However these levels were much lower than those observed at the lower LP UV doses. This indicates that if conditions for repair were present following these doses some minimal level of repair in *E. coli* O157:H7 may take place. Following doses of 20 and 40 mJ/cm^2 no light independent repair was observed. This indicates that repair

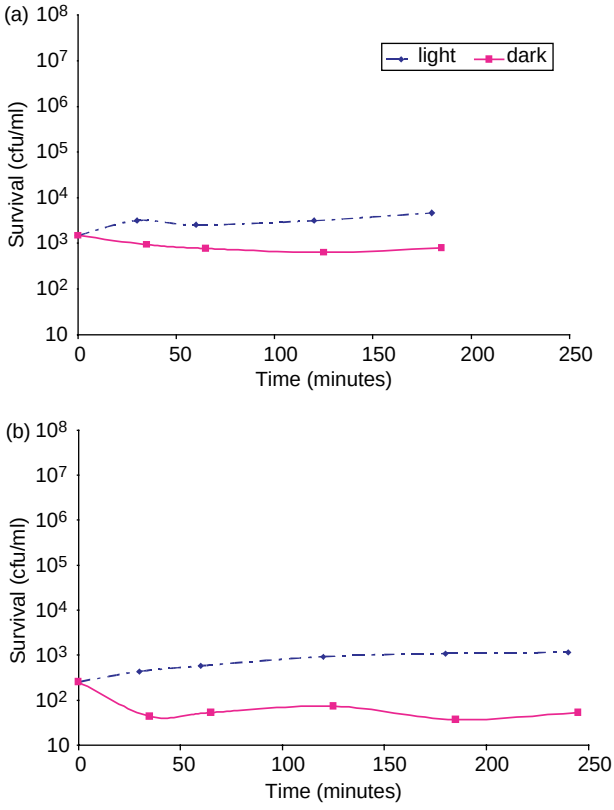

Figure 4 | Repair potential of *E. coli* O157:H7 following LP UV irradiation at (a) 20 and (b) 40 mJ/cm^2. Representative experiments are shown.

Table 2 | Comparison of initial log inactivation and effective log repair of *E. coli* O157:H7 following LP and MP UV exposure after light and dark incubation

UV lamp source	UV Dose (mJ/cm^2)[a]	Average initial log inactivation	Repair conditions	Average effective log repair[b]	Actual log inactivation with repair[c]
LP	5	4.5 (±0.2)	Light	3.4 (±0.1)	1.0
			Dark	1.4 (±0.0)	3.1
	8	5.1 (±0.3)	Light	2.9 (±0.2)	2.2
			Dark	0.7 (±0.1)	4.4
	20	5.1 (±0.1)	Light	0.4 (±0.0)	4.7
			Dark	−0.2 (±0.2)[d]	5.3
	40	5.3 (±0.1)	Light	0.5 (±0.2)	4.8
			Dark	−0.3 (±0.3)[d]	5.6
MP	5	6.4 (±0.2)	Light	0.7 (±0.1)	5.7
			Dark	0.1 (±0.1)	6.3
	8	6.6 (±0.4)	Light	0.9 (±0.2)	5.7
			Dark	0.3 (±0.3)	6.3

[a]MP UV doses are weighted.
[b]Effective log repair = log CFU/ml at the maximum repair level − log CFU/ml immediately following UV exposure.
[c]Actual log inactivation with repair = initial log inactivation−effective log repair.
[d]Negative values reflect mortality following exposure.

is less likely to occur during light restricted distribution following treatment.

A comparison of repair following both LP and MP UV irradiation, under light and dark conditions, at each tested dose is shown in Table 2. These data are presented as average effective log repair. Effective log repair was calculated as the difference between the log CFU/ml at the maximum repair level and log CFU/ml immediately following UV exposure. Determining the effective log repair allows for comparison with initial log inactivation data. Examining the difference between the initial inactivation levels (initial log inactivation) and the level following repair (effective log repair) can aid in determining the "actual" levels of inactivation when repair is taken into account. Actual log inactivations following LP and MP UV irradiation are included in Table 2.

As shown in Table 2, when DNA photo repair of *E. coli* O157:H7 is taken into consideration, the initial levels of inactivation observed following UV treatment may be reduced if conditions are appropriate. This difference is more pronounced following lower doses of LP UV than following MP UV exposure. For example, at a LP UV dose of 5 mJ/cm^2 a 4.5 log inactivation of *E. coli* O157:H7 occurs initially, however with light exposure the actual inactivation of this organism is reduced to 1 log as a result of repair opportunity. Following MP UV at the same dose the initial log inactivation is 6.4, after light exposure the inactivation is reduced slightly to 5.6 log inactivation. Similar differences were observed following a higher dose of 8 mJ/cm^2. Following incubation under dark conditions, LP UV irradiated samples were reduced by up to 1.4 log units, with the greatest difference again following the lowest tested dose of 5 mJ/cm^2. No effective repair was observed following MP UV doses after incubation under dark conditions. Following LP UV at higher doses of 20 and 40 mJ/cm^2 the actual log inactivations were reduced only slightly in light (average 0.4 to 0.5 log units), and no reduction was observed following incubation in the dark.

As can be observed in Figure 4 and shown in Table 2 (expressed as negative average effective log repair) survival decreased under dark conditions following irradiation at these higher doses.

When properly operated and maintained, UV technology can be an effective drinking water disinfection technology. With well maintained UV reactors delivering appropriate UV doses ($\geq 40\ mJ/cm^2$), followed by suitable chemical disinfection residuals, it is unlikely that opportunity for repair of *E. coli* O157:H7 of significance to affect public health would arise following either LP or MP UV irradiation. It is at times when the delivered UV dose is reduced that repair may become a concern following UV irradiation. A reduced delivered UV dose can occur as a result of improper hydraulic conditions within the UV reactor, inorganic or organic fouling on the UV lamp, lamp outage, aging lamps, power failures, and increases in turbidity or reduced transmittance of the water (USEPA 1999; NWRI & AWWARF 2000). These issues may arise more often with smaller water suppliers and in-home UV units, where there may be limited continuous monitoring of the system.

Ensuring conditions that are unfavourable for repair should be part of the UV treatment strategy, and DNA repair should be taken into consideration when justifying minimum dose regulations. The maintenance of a chemical disinfectant residual (e.g. chlorine) in water following UV treatment will minimize the opportunity for repair.

CONCLUSIONS

Pathogenic *E. coli* O157:H7 has the ability to photo repair rapidly (< 30 minutes) following low doses (5 and $8\ mJ/cm^2$) of LP UV radiation. In comparison to LP UV, following MP UV irradiation photoreactivation was substantially lower at comparable doses (5 and $8\ mJ/cm^2$). At lower doses light independent repair of *E. coli* O157:H7 was observed following LP UV irradiation, but was minimal compared to photoreativation. Following higher LP UV doses of 20 and $40\ mJ/cm^2$, similar to those used in drinking water treatment, photoreativation of *E. coli* O157:H7 was limited. Repair was not observed following these higher doses of LP irradiation after incubation under dark conditions. These data demonstrate that DNA repair in *E. coli* O157:H7 is unlikely under ideal treatment conditions. Under non-ideal treatment conditions, this study indicates that *E. coli* O157:H7 may photo repair rapidly following exposure to LP UV, however, irradiation with MP UV under these non-ideal conditions may result in limited ability to repair.

It is recommended that DNA repair and survival of *E. coli* O157:H7 be evaluated using natural waters and environmental isolates. Additional studies need to be carried out to further elucidate the specific type of UV damage and repair processes that are taking place in *E. coli* O157:H7 following exposure to UV irradiation.

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