Evaluation of solar light inactivation on multidrug-resistant *Escherichia coli* CGMCC 1.1595
Xiu-Feng Yin, Na Shi, Ting Meng and Ying-Xue Sun

**ABSTRACT**

This study investigated the simulated solar light disinfection of *Escherichia coli* CGMCC 1.1595, a multidrug-resistant (MDR) strain resistant to tetracycline and ampicillin. With the increase of light intensity, the maximum inactivation efficiency reached 0.74 log in 60 min following visible light irradiation with an intensity of 115.8 mW/cm² and following UVA–visible light irradiation, using a 98% UVA-ray contribution at 6.5 mW/cm² and 95% contribution at 20.0 mW/cm², the inactivation efficiency was up to 6.09 log. The inactivated MDR *E. coli* did not regrow after light irradiation or in the dark after 24 or 48 h after visible light disinfection, demonstrating that visible light disinfection can prevent MDR *E. coli* self-repair. The MDR *E. coli* plasmid electrophoresis band gradually went dark with increase of the light irradiation time and could be completely eliminated by high UVA light intensity treatment, however, simulated sunlight irradiation had minimal influence on both tetracycline and ampicillin resistance of the MDR *E. coli* strain.

**Key words** | inactivation, multidrug resistant *E. coli* strain, plasmid elimination, solar light radiation, tetracycline resistance shift

**HIGHLIGHTS**

- Simulated solar light can inactivate multidrug resistant (MDR) *Escherichia coli* CGMCC 1.1595.
- The inactivation efficiency of MDR *E. coli* reached 0.74 log in 60 min under visible light irradiation.
- The inactivation efficiency of MDR *E. coli* was up to 6.09 log under UVA–visible light irradiation.
- Visible light disinfection can prevent MDR *E. coli* self-repair.
- Simulated sunlight irradiation had minimal impact on the tetracycline and ampicillin resistance of MDR *E. coli*.

**INTRODUCTION**

The global increase in antibiotic consumption has resulted in large quantities of pharmaceutical origin being found in sewage and wastewater treatment plants (Rizzo *et al.* 2013; Kraemer *et al.* 2019). One of the most severe consequences of antibiotic pollution is the rise in antibiotic resistance (Kümmerer 2009). Antibiotic-resistant bacteria (ARB) and antibiotic-resistance genes (ARGs) are extensively detected in wastewater effluents and aquatic environments worldwide and pose a serious threat to aquatic ecosystems and human health (Ouyang *et al.* 2015; Xu *et al.* 2016; Gao *et al.* 2018). The aquatic environment is considered to be a particularly vast reservoir of ARGs and is also where ARB can emerge following the transfer of ARGs between autochthonous and allochthonous bacteria (Shao *et al.* 2018; Almakki *et al.* 2019). A few chromosomal ARGs transmit to their offspring through vertical gene transfer and most plasmid ARGs may transfer between bacterial strains via horizontal gene transfer (Forbes & Schaberg 1985; Rasmussen & Sørensen 1998; Gao *et al.* 2018).
The spread of ARBs in aquatic environments is affected by many factors including sunlight radiation, temperature, and dissolved oxygen, and among these sunlight radiation is of significance (García-Fernández et al. 2015; Giannakis et al. 2018a). Solar light can inactivate microorganisms in surface waters (Boehm et al. 2009). Giannakis et al. (2018b) found that a 6 log inactivation of a streptomycin-resistant strain could be achieved after 2 h irradiation with a simulated solar light intensity of 1,200 W/m². The mechanism of solar light inactivation involves direct photolysis of photo-oxidation and indirect photolysis of photosensitivity reactions through which the absorption of solar radiation by photosensitizers in the water results in the formation of active intermediate action cells (Silverman et al. 2013).

Solar light is mainly composed of ultraviolet A (UVA) (320–400 nm), ultraviolet B (UVB) (290–320 nm), ultraviolet C (UVC) (200–290 nm), visible (400–700 nm) and infrared light (Silverman et al. 2013). UVB/C can be directly absorbed by DNA, leading to the formation of lesions in pyrimidine and purine bases (Lian et al. 2018), however, UVC cannot reach the Earth’s surface because of the ozone layer in the stratosphere and most UVB quickly decays in natural water (Kadir & Nelson 2014; Lian et al. 2018). UVA’s effects on DNA include direct and UVA oxidative damage. Direct damage is the destruction of DNA by the formation of the cyclobutane pyrimidine dimer (Giannakis et al. 2016). In a visible light disinfection system, microbacteria can use endogenous photosensitizers such as NADH and cytochromes to absorb photons (Lavi et al. 2004) at 400–500 nm which can produce reactive oxygen species (ROS) that attack cell membranes and cause cell damage (Garza et al. 2018). UVA rays make up approximately 95% of total UV radiation but only 10% of the solar energy that reaches the Earth’s surface, while visible light accounts for 40% of the solar energy. It is therefore important to understand microbial inactivation in surface waters by UVA rays and visible light.

Jiménez-Tototzintle et al. (2018) found that the efficiency of P. aeruginosa inactivation increased along with the UVA light dose, with a maximum of 6 log achieved after 3 h irradiation with 7.74 kJ/L of accumulated UVA intensity. Lui et al. (2016) investigated the ability of semi-commercial LED arrays (270–740 nm) to inactivate Escherichia coli K12 ATCC W3110 and Enterococcus faecalis ATCC 19433, and found that inactivations of 5 log and greater were consistently achieved after 6 h irradiation with the 270, 365, 385 and 405 nm arrays. Visible light is not as effective at microbial disinfection as UVA rays. Li et al. (2017) found that the efficiency of Escherichia coli DH5α inactivation was less than 1 log after 6 h irradiation using a xenon lamp (300 W) with λ > 420 nm.

High visible light intensity can promote the absorption of more photons by endogenous cellular photosensitizers, leading to the generation of high levels of ROS, which will damage bacteria (Lubart et al. 2011), however, it is difficult for solar light disinfection to eliminate antibiotic resistances in E. coli. Giannakis et al. (2018b) found that the blaCTX-M-9 gene in E. coli ESBL 8543 was not eliminated after 4 h solar light disinfection and Rizzo et al. (2012) reported that while solar light radiation can reduce ciprofloxacin resistance (MIC decreased by 33% after 3 h irradiation) in an MDR E. coli isolate from a wastewater treatment plant, it did not impact its resistance to amoxicillin (MIC > 256 µg/mL) and sulfamethoxazole (MIC > 1,024 µg/mL).

In this paper, the effect of light irradiation on tetracycline and ampicillin resistance in Escherichia coli CGMCC 1.1595, an MDR, was studied using simulated solar light. The inactivation efficiency of different light intensity conditions was analyzed and the photoreactivation and dark repair effects were also investigated. Furthermore, the inactivation mechanism was examined using a plasmid elimination test and observing changes in antibiotic susceptibility.

MATERIALS AND METHODS

Microorganism

The MDR E. coli strain (E. coli CGMCC 1.1595) was provided by the Institute of Microbiology of the Chinese Academy of Sciences. This strain harbors the plasmid pBR322 which confers resistance to tetracycline (TET) and ampicillin (AMP) (Huang et al. 2013; Pang et al. 2016).

Sample preparation

The MDR E. coli was cultured by removing a single colony from plates, subculturing in nutrient broth (g/L) (peptone:
10.0, beef extract: 3.0, NaCl: 5.0, pH: 7.2) with 16 mg/L TET and 32 mg/L AMP at 37 °C overnight in a rotary shaker (200 rpm). The cells were collected by centrifugation (5 min at 12,000 rpm at 4 °C), washed twice with sterile physiological solution, and resuspended in sterile physiological solution at a concentration of approximately 10^6 CFU/mL.

**Light irradiation experiment**

Light irradiation experiments were performed using an XPA-7 photochemical reactor (Xujiang Electromechanical Plant, China) equipped with a constant-temperature water tank. The bacterial suspension was continuously stirred in the reactor while light irradiation was provided by 100, 300, 500 W mercury and 1,000 W xenon lamps equipped with 300 nm or 400 nm optical filters to simulate UVA–visible light (λ > 300 nm) and visible light (λ > 400 nm). During light irradiation disinfection, light intensities with wavelengths of 300–400 nm and 400–760 nm were measured with UV-A and FZ-A irradiance meters, respectively. The light dose (D) was the product of the light irradiation time (T) and light intensity (I).

**Bacterial count**

Bacterial counts were performed using the spread plate method. Briefly, small amounts of bacterial suspension were diluted according to the expected number of colonies; 100 μL of diluted bacterial suspension was spread onto a nutrient agar plate (g/L) (peptone: 10, beef extract: 3, NaCl: 5, agar: 15, pH: 7.2) and incubated at 37 °C for 24 h. Measurements were made in triplicate and the average values and standard deviation were plotted as CFU/mL. The inactivation efficiency of the sample was expressed as logarithmic inactivation efficiency (logN₀/Nᵣ, where N₀ and Nᵣ represent the concentration of the bacterial suspension before and after light irradiation disinfection).

**Photoreactivation and dark repair**

After light irradiation, bacterial suspension samples were transferred to Petri dishes for photoreactivation and dark repair tests. For the photoreactivation capacity, dishes were placed on magnetic stirrers, exposed to light irradiation for 48 h and bacterial concentration counted at 24 h intervals. To assess dark repair capacity, dishes were placed on magnetic stirrers in the dark for 48 h and bacterial concentration counted at 24 h intervals. The photoreactivation/dark repair efficiency was calculated using Equation (1):

\[
\text{Reactivation efficiency} = \frac{N_r - N_t}{N_0 - N_t} \times 100\% \quad (1)
\]

where \(N_0\) and \(N_t\) represent bacterial concentration before and after light irradiation disinfection; \(N_r\) represents hatchability of the inactivated microbe exposed to light (photoreactivated) or dark (dark repair).

**Plasmid elimination test**

The plasmid elimination test was conducted with 100, 300, and 500 W mercury and 1,000 W xenon lamps. Strains were inoculated in nutrient broth supplemented with 16 mg/L TET and 32 mg/L AMP at 37 °C and 200 rpm for 14 h, then centrifuged at 12,000 g for 5 min, and washed twice with sterile water to remove medium and antibiotics. Bacteria were resuspended in sterile water and added into a tube to eliminate the plasmid. The plasmid was extracted using the Takara Minibest Purification Plasmid Kit ver.4.0 (Takara Plasmid, China).

**Antibiotic resistance assay**

Bacterial antibiotic resistance before, and after, plasmid elimination was assessed with the Kirby–Bauer method according to Clinical and Laboratory Standards Institute (2013). After plasmid elimination, colonies were transferred into 10 mL of physiological solution to achieve a concentration of 10^8 CFU/mL (0.5 McFarland). Bacterial suspensions were then spread onto Mueller–Hinton agar (g/L) (casein hydrolysate: 17.5, starch: 1.5, beef extract: 5, agar: 12.5, pH: 7.2) using a sterile cotton swab. Antibiotic discs of TET (30 mg) and AMP (30 mg) (Hang Zhou Microbial Reagent Co., Ltd, China) were placed on the surface of each inoculated plate. After incubation for 16–18 h at...
35 ± 2 °C, the diameters of the growth inhibition zones were measured.

**Stable inheritance of antibiotic resistance after plasmid elimination**

After irradiation with either 100, 300, and 500 W mercury or 1,000 W xenon lamps, the MDR *E. coli* was subcultured in nutrient broth containing either no antibiotic, 16 mg/L TET, or 32 mg/L AMP (37 °C, 200 rpm, 14 h). Antibiotic resistance was assessed with the Kirby–Bauer method every two generations to track the variation tendency.

**RESULTS AND DISCUSSION**

**MDR *E. coli* inactivation**

Inactivation of MDR *E. coli* using simulated solar light was performed using various light intensities. The light intensities of visible light (λ > 400 nm) generated by 100, 300, and 500 W mercury and 1,000 W xenon lamps were 18.9, 27.3, 40.2, and 115.8 mW/cm², respectively. During the visible light disinfection, the inactivation efficiency increased with irradiation time and reached 0.09, 0.14, 0.28 and 0.74 log at 1 h with light intensities of 18.9, 27.3, 40.2, and 115.8 mW/cm² (Figure 1(a)). Furthermore, the inactivation efficiency increased with light intensity and dose at certain irradiation times (Table S1, Supporting Information). Such results might be caused by bacteria using endogenous photosensitizers to produce ROS, which can attack bacteria, disrupt bacterial defense and also prevent bacterial photoreactivation under high visible light intensity (Rincón & Pulgarin 2003).

The simulated solar light of λ > 300 nm was produced using the 100, 300, and 500 W mercury and 1,000 W xenon lamps combined with 300 nm optical filters. The visible light intensities were 18.9, 27.3, 40.2, and 115.8 mW/cm², while the UVA light intensities were 6.5, 10.0, 20.0 and 2.83 mW/cm², respectively. Figure 1(b) shows the inactivation efficiency of light radiation of λ > 300 nm. During this light radiation disinfection, the inactivation efficiency increased with the irradiation time, reaching 6.09, 1.17, 6.09 and 1.16 log at 1 h (Figure 1(b)). While the inactivation efficiency was up to 6.09 log under λ > 300 nm light radiation, UVA–visible light disinfection efficiency was higher.

With λ > 300 nm light radiation for 1 h, the contributions of UVA rays to the inactivation rate were approximately 98%, 88%, 95% and 36%, where the light intensities were 6.5, 10.0, 20.0 and 2.83 mW/cm², respectively. In the UVA–visible light disinfection system the inactivation efficiency was controlled by the UVA light intensity and dose (Table S2). UVA can lead the photosensitizer to absorb UVA photons and generate ROS, which attacks DNA and causes oxidative damage, resulting in bacterial damage (Baier et al. 2006; Ito et al. 2007). Moreover, high light intensity may obtain a high flow of photons, and...
increased photons can directly attack bacteria to increase the inactivation efficiency (Xiong & Hu 2013). The inactivation increased rapidly with the light radiation of $\lambda > 300$ nm within 50 min irradiation time, and then exhibited a lag phase, which may be due to the accumulation of UVA oxidation-related injury (Giannakis et al. 2016).

**Photoreactivation and dark repair**

The photoreactivation and dark repair percentages of the MDR *E. coli* varied between the visible light and UVA-visible inactivation groups (Figure 2). The photoreactivation percentages of MDR *E. coli* inactivated by 1 h exposure to the visible light intensities of 18.9, 27.3, 40.2 and 115.8 mW/cm$^2$ were $-355\%$, $-231\%$, $-64\%$ and $-22\%$ after 24 h continuous light irradiation and $-346\%$, $-229\%$, $-77\%$ and $-22\%$, respectively after 48 h (Figure 2(a)). Although the inactivation efficiency of visible light disinfection with 115.8 mW/cm$^2$ was lower compared with UVA-visible, the inactivated MDR *E. coli* did not regrow after light irradiation for 24 h and 48 h. This might be due to the high flow of photons that a high light intensity obtains, which can prevent bacterial reactivation (under visible light) (Xiong & Hu 2013).

There was no photoreactivation of MDR *E. coli* inactivated by 1 h of UVA-visible light simulated by the 100 W (with UVA and visible light intensity of 6.5 and 25.4 mW/cm$^2$, respectively) and 500 W mercury lamps (with UVA and visible light intensity of 20.0 and 61.2 mW/cm$^2$, respectively). The photoreactivation percentages of the MDR *E. coli* inactivated by 1 h of UVA-visible light simulated by the 200 W mercury (with UVA and visible light intensity of 10.0 and 27.3 mW/cm$^2$, respectively) and 1,000 W xenon lamps (with UVA and visible light intensity of 2.83 and 115.8 mW/cm$^2$, respectively) were 24% and 24.8% after 24 h continuous light irradiation, and 93% and 39.5% after 48 h. The photoreactivation ability of an organism depends on a number of factors including light dose, wavelength, light intensity, and exposure time (Benabbou et al. 2007). Xiao et al. (2018) found that *E. coli* ATCC 15597 and 700891 resurrected after 4 h irradiation under an LED lamp (10 klx) after UVA-visible pre-radiation and that the effect of a low dose of UVA-visible light ($1.08 \times 10^5$ J/cm$^2$) on ROS production was not enough to cause damage to the bacteria. Photoreactivation may rely on pyrimidine dimers that were not completely damaged by light irradiation and the photolyase was still active after binding to the dimer (Wen et al. 2019).

The dark repair percentages of MDR *E. coli* inactivated by 1 h of visible light intensities of 18.9, 27.3, 40.2 and 115.8 mW/cm$^2$, were $-455\%$, $-209.9\%$, 7.18% and $-22\%$ after 24 h in the dark, and $-380\%$, $-256.7\%$, $-80.1\%$ and $-22\%$ after 48 h (Figure 2(b)). Similar to the photoreactivation results, MDR *E. coli* inactivated by visible light with 115.8 mW/cm$^2$ intensity did not regrow after 24 and 48 h in the dark. It is possible that in the visible light disinfection system, ROS could persist and prevent the dark repair capacity of MDR *E. coli* (Xiong & Hu 2013). Furthermore, there was no dark repair observed in MDR *E. coli*
inactivated with 1 h of UVA–visible light simulated by the 100 and 500 W mercury lamps. The dark repair percentages of MDR *E. coli* inactivated by 1 h of UVA–visible light simulated by the 200 W mercury and 1,000 W xenon lamps were 32.8% and 29% after 24 h in the dark, and 80.4% and 45% after 48 h. This might be because the UVA light irradiation dose was lower than $1.8 \times 10^3$ J/cm² and could not therefore inhibit bacterial repair (Xiao et al. 2018). The completely inactivated MDR *E. coli* did not self-repair after 48 h in the dark, which is consistent with a previous study (Benabou et al. 2007). Visible light has a residual disinfection effect, which prevents MDR *E. coli* self-repair. UVA disinfection requires a high light dose to prevent MDR *E. coli* performing the dark repair process.

**Effect of light irradiation on plasmid elimination**

TET and AMP resistance of MDR *E. coli* did not change during 1 h light irradiation under the study conditions. Figure S1 (Supporting Information) illustrates the change in TET inhibition zone diameter under $\lambda > 400$ nm and $\lambda > 300$ nm light irradiation and shows that its diameter was less than 11 mm. MDR *E. coli* did not form an AMP inhibition zone in this light irradiation system. This suggests that the MDR *E. coli*'s TET and AMP resistance was not affected by $\lambda > 400$ nm and $\lambda > 300$ nm light radiation, which agrees with a previous study which showed that the *bla*<sub>ctx-m-9</sub> gene in *E. coli* ESBL 8543 was not eliminated after solar light disinfection for 4 h (Giannakis et al. 2018b).

To evaluate the effects of full wavelength light irradiation on the antibiotic resistance of MDR *E. coli*, the influence of light irradiation on plasmid elimination was explored. The plasmid was digested with HindIII and the reaction product separated on a 0.7% agarose gel by electrophoresis at 110 V (Figure 3). Quantitative analysis using ImageJ software to measure the gray value of the electrophoresis bands with the marker used as an internal reference is shown in Figure 4. With increasing light irradiation time the relative gray value of the plasmid electrophoresis band gradually decreased. Light irradiation dose was also an important factor that influenced plasmid elimination. The relative gray value of the plasmid band decreased from 0.58 to 0 during 10 min light irradiation time under the 500 W mercury lamp, which indicates that the plasmid can be completely eliminated by high UVA light intensity.

Xenon lamp irradiation (mainly visible light) had a lower impact on plasmid elimination than mercury lamp irradiation (mostly UVA). During visible light disinfection, endogenous cellular photosensitizer bacteria can absorb the visible light photons, generating ROS which cause bacterial damage (Lubart et al. 2011). UVA could penetrate the cell membrane and cytoplasm, and covalently bind two thymine bases adjacent to the bacterial DNA to form dimers, thus damaging the pyrimidines and purine bases (McGuigan et al. 2012) and the configuration of DNA and RNA, interfering with its normal replication (Horai et al. 2017).

**Changes in antibiotic susceptibility of MDR *E. coli***

The Kirby–Bauer test was used to assess MDR *E. coli* resistance to TET (Figure 5). The resistance, mediation, and sensitivity of *E. coli* CGMCC 1.1595 to TET and AMP were judged according to the diameter of the inhibition zone. According to the standard of Clinical and Laboratory Standards Institute (2015), the TET tolerance of *Enterobacteriaceae* is determined by the inhibition zone diameter (D) as follows: D > 15 mm is sensitive, D between 12 and 14 mm is intermediary, and D < 11 mm is resistant. AMP tolerance of *Enterobacteriaceae* is determined by the inhibition zone diameter (D) as follows: D > 17 mm is sensitive, D between 14 and 16 mm is intermediary, D < 13 mm is resistant. In brief, the smaller the inhibition diameter, the higher the bacterial resistance to the antibiotic.

The average inhibition zone diameter for TET before light irradiation was 8.5–9.7 mm, and after 10 min light irradiation, the diameter was less than 11 mm (Figure 5). This indicates that MDR *E. coli* resistance to TET was not altered by solar light irradiation. MDR *E. coli* did not form an AMP inhibition zone after either mercury or xenon lamp irradiation. Giannakis et al. (2018b) reported that the *bla*<sub>ctx-m-9</sub> gene in *E. coli* ESBL 8543 was not eliminated after 4 h solar light disinfection and Rizzo et al. (2012) found that while solar light radiation can influence ciprofloxacin resistance (MIC decreased by 33% after 180 min of irradiation), it had no impact on amoxicillin (MIC > 256 μg/mL) and sulfamethoxazole (MIC > 1,024 μg/mL) resistances. From our results, we see that solar light
Figure 3 | Agarose gel electrophoretogram of the plasmid DNA after light irradiation stimulated by (a) 100 W, (b) 300 W, (c) 500 W mercury and (d) 1,000 W xenon lamps; 1-6 represent 0, 2, 4, 6, 8, 10 min, respectively.

Figure 4 | Relative gray value of the electrophoresis bands of plasmid DNA after light irradiation.

Figure 5 | Tetracycline (TET) inhibition zone diameter before and after plasmid elimination.
irradiation had no influence on AMP and TET resistance in the MDR *E. coli*.

After plasmid elimination by light irradiation, the MDR *E. coli* were cultured in nutrient broth containing no antibiotics, 16 mg/L TET, or 32 mg/L AMP for ten generations. During this process, MDR *E. coli* did not form an AMP inhibition zone (Table 1) and resistance to TET and AMP did not change as the number of passages increased. In addition, there was no difference in the change of TET resistance between the MDR *E. coli* cultured in nutrient broth containing no antibiotics, TET, or AMP. This suggests that the antibiotic itself might not have an inductive effect on antibiotic resistance.

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N/A, not available.

**CONCLUSION**

This study examined the inactivation of the MDR *E. coli* strain (*E. coli* CGMCC 1.1595) by simulated solar light. During the visible light disinfection, inactivation efficiency increased along with light intensities, reaching 0.74 log at 1 h irradiation time at a light intensity of 115.8 mW/cm². Under UVA-visible light irradiation, the inactivation efficiency reached 6.09 log, with UVA rays contributing 36% to 98% to this efficiency at UVA light intensities of 2.83 to 20.0 mW/cm². Although the inactivation efficiency of the visible light disinfection at an intensity of 115.8 mW/cm² was lower than that of the UVA-visible, the inactivated MDR *E. coli* did not regrow after either 24 or 48 h in light irradiation or in the dark, demonstrating that visible light disinfection can prevent MDR *E. coli* self-repair. As light irradiation time increased, the resistance plasmid electrophoresis band gradually went dark and the plasmid could be eliminated completely by high UVA light intensity. Overall, light irradiation had no influence on TET and AMP resistance of MDR *E. coli*. This may also show that the antibiotic itself may not have an obvious inductive effect on antibiotic resistance.

**ACKNOWLEDGEMENTS**

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**SUPPLEMENTARY MATERIAL**

The Supplementary Material for this paper is available online at https://dx.doi.org/10.2166/ws.2020.124.

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