The Bactericidal Effect of Ultraviolet and Visible Light on *Escherichia coli*

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**ABSTRACT:** The bactericidal radiation dosages at specific wavelengths in the ultraviolet (UV)–visible spectrum are not well documented. Such information is important for the development of new monochromatic bactericidal devices to be operated at different wavelengths. In this study, radiation dosages required to cause mortality of an *Escherichia coli* strain, ATCC 25922, at various wavelengths between 250 and 532 nm in the UV and visible spectrum were determined. Radiation at 265 nm in the UV region was most efficient in killing the *E. coli* cells and 100% mortality was achieved at a dose of 1.17 log mJ/cm². In the visible spectrum, the radiation dosages required for a one-log reduction of the *E. coli* cell density at 458 and 488 nm were 5.5 and 6.9 log mJ/cm², respectively. However, at 515 and 532 nm, significant killing was not observed at radiation dosage up to 7 log mJ/cm². Based on the cell survival data at various radiation dosages between 250 and 488 nm, a predictive equation for the survival of *E. coli* cells is derived, namely log(*S*/*S*₀) = −(1.089 × 10⁷ e⁻⁰.⁰₆₃₃ × L). The symbols, *S*₀, *S*, L, and D, represent initial cell density, cell density after irradiation, wavelength of the radiation and radiation dosage, respectively. The proportion of the surviving *E. coli* cells decreases exponentially with the increase in radiation dosage at a given wavelength. In addition, the radiation dose required for killing a certain fraction of the *E. coli* cells increases exponentially as the wavelength of radiation increases.


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**KEYWORDS:** UV radiation; visible laser light; bactericidal effect; *Escherichia coli*

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**Introduction**

Ultraviolet light (UV) has been used extensively for disinfection at waste and drinking water treatment and clinical and industrial facilities (Decho, 2000; Wilson, 1994). For most UV disinfection equipment, either the mercury 254 nm UV line or the UV–visible light bands from a xenon arc lamp is used as the UV source (Hockberger, 2002). However, with the creation of new UV diodes and lasers that emit radiation between 280 and 400 nm, applications of these devices to the production of bactericidal UV light systems can be anticipated once lethal dose information is available.

Visible light lasers have been used as bactericidal agents to remove bacterial biofilms (Nandakumar et al., 2006). Recently, Hamblin et al. (2005) also showed that high intensity visible light could kill *Heliobacter pylori* in the stomach of humans. Therefore, in some instances, the use of visible light as a bactericidal agent might prove to be cost effective and cause fewer side effects than anti-bacterial drugs. However, as in the case for UV, insufficient wavelength dependent dose information is available documenting the bactericidal effects of visible light.

*Escherichia coli* is an indigenous member of the intestinal flora of healthy humans and warm-blooded animals, and comprises about 1% of the total bacterial biomass (Leclerc et al., 2001). Although only some *E. coli* strains are pathogenic to humans, certain pathogenic serotypes, such as *E. coli* O157:H7, have caused numerous outbreaks associated with food and drinking water (Betts, 2000; Kuhnert et al., 2000; Rasmussen and Casey, 2001; Swerdlow et al., 1992). Because *E. coli* is ubiquitous in the fecal materials of humans and warm-blooded animals and the presence of fecal contamination correlates well with the presence of
many fecal-borne human pathogens (Leclerc et al., 2001), this bacterial species has been used as a standard fecal contamination indicator to assess the cleanliness and safety of drinking water and commercial food (Tallon et al., 2005; Van Houdt and Michiels, 2005). As UV disinfection is becoming more popular in drinking/waste water treatment as well as in clinical and industrial facilities, it is of interest to examine the bactericidal effects of UV and visible light on this bacterial species. The objectives of this study are: (1) to determine the survival of an E. coli strain, ATCC 25922, when it is exposed to various dosages of radiation ranging from 250 to 532 nm; and (2) to develop a predictive mathematic equation to estimate the survival of E. coli based on the wavelengths and dosage of the radiation.

Materials and Methods

Bacteria Preparation

The E. coli ATCC 25922 strain, a Gram-negative bacterium, was obtained from the American Type Culture Collection (ATCC). This bacterium is a non-diarrheagenic clinical strain and has been used as a standard E. coli strain for various microbiological tests. The bacterial culture was stored in 25% glycerol at −80°C and re-grown on Trypticase Soy Agar (TSA) (Difco, Detroit, MI) plates. The E. coli cells were cultured in Trypticase Soy broth (TSB) (Difco) for 15 h, in a shaking incubator at 200 rpm at 37°C. After incubation, the culture was washed in sterile distilled water and centrifuged for 10 min at 3,000g. The wash process was repeated three times. The washed cells were suspended in sterile distilled water to $7.85 \times 10^7$ CFU/mL prior to the UV and visible radiation treatments. This concentration was chosen because it provided the highest cell density possible for the radiation treatments and was still transparent enough to ensure that all cells received equal exposure dose during treatment in a shallow container.

UV Treatments

A 100 W PTI xenon arc lamp in combination with a Jarrell-Ash 1/4 m monochromator fitted with two interchangeable ruled gratings blazed at 250 and 400 nm, provided wavelength selection for sample irradiation. Entrance and exit slits for the monochromator were opened to maximum values to provide high output intensity. Wavelengths selected were 250, 265, 275, 290, 300, 325, 350, 365, 375, and 400 nm. After leaving the monochromator, the light beams were refocused to the sample location. Intensities there were determined using a calibrated Laser Precision Corp. (Utica, NY) radiometer, consisting of a RL-3610 Power Meter and a RKP-360 pyroelectric detector. Radiation dosage of a treatment in ml/cm² was determined by the intensity and the exposure time of the treatment. Due to the spectral intensity profile of the xenon arc lamp and the optical through put of the monochromator, light intensities at the sample ranged from 0.2 mW/cm² at 250 nm to 4.0 mW/cm² at 400 nm, with an approximately linear intensity increase with wavelength. Given the measured intensities, lethal dose exposure times could be calculated and ranged from approximately 30 s at 250 nm to about an hour at 400 nm.

Once an E. coli cell suspension at $7.85 \times 10^7$ CFU/mL was prepared, 100 µL of the cell suspension was pipetted into a sterile ring device for UV treatment. This device consisted of a glass slide onto which an aluminum ring of 8-mm internal diameter and 3-mm depth was cemented. The cement used was a clear nail polish. The inner wall of the aluminum ring was tapered by about 8° resulting in a greater diameter at the top, to prevent shadowing of the samples during irradiation. The cell suspensions were exposed to specific UV wavelengths at various dosages. After treatment, the cell samples were serial diluted in a sterile phosphate buffer saline (PBS) and drop-plated on TSA plates for viable cell density analysis. Each graph representing a certain exposure wavelength is the result of 3–5 exposure runs. This procedure, while time consuming, provided multi-run validation of the repeatability of the results.

Visible Laser Radiation Treatments

Visible radiations at 458, 488, and 515 nm were obtained from a Coherent Innova 70–4 Argon Ion laser and the laser lines were separated using a glass prism. The power output of this laser was set to 700 mW at 488 nm and at 458 nm and 2.0 W at 515 nm. At 532 nm, a Spectra Physics Mellenia V frequency doubled Nd:YAG laser with a power output of 2.0 W was used. The actual radiation intensities received by the samples were determined by a Laser Precision radiometer or a Coherent 210 laser power meter, depending on the intensity level.

A 1-mL E. coli cell suspension was put into a sterile 4-mL glass cuvette with a small sterile magnet stir bar at the bottom. The cuvette was placed on a magnetic stir plate and stirred during a laser irradiation treatment lasting from minutes to about an hour, depending on the dose required. After treatment, a 100 µL portion of the sample was taken out of the treated E. coli suspension, serial diluted and drop plated on TSA plates for viable cell density analysis.

Absorbance Spectrum of E. coli

An E. coli cell suspension at $7.85 \times 10^7$ CFU/mL was prepared in PBS as described earlier. One-milliliter portion of the cell suspension was transferred to a quartz cuvette with a 1 cm optical path. The absorbance spectrum of the cell sample between 200 and 600 nm was determined using a Varian Cary 50 Probe UV–visible spectrophotometer (Fisher Scientific, Nepean, Canada).
Results

Effect of UV and Visible Radiation on E. coli

UV radiation at 265 nm was most effective in killing the E. coli cells and a lethal dose required to achieve 100% killing (LD100) was 1.17 log ml/cm² (Fig. 1). For 250 nm, the LD100 dose increased to 1.47 log ml/cm². Similarly, for wavelengths longer than 265 nm, a rapid increase in LD100 was observed with the increase in wavelength, reaching 3.5 log ml/cm² at 375 nm. The intensities of the UV light were ranged from 200 μW/cm² to a few tens of mW/cm². To avoid a heating effect on the cell samples, the visible laser radiation intensities were limited to 2 W/cm² or less. Despite such limitation, radiation at visible wavelengths of 458 and 488 nm were able to produce significant mortality to the E. coli cells. The radiation dosages required for a one-log reduction of the E. coli cell density at 458 and 488 nm were 5.5 and 6.9 log ml/cm², respectively. However, laser treatment at 515 and 532 nm did not produce a significant reduction in the viability of the E. coli cells with radiation doses up to 7 log ml/cm². A linear regression analysis of log dose (<7 log ml/cm²) versus log cell density on 515 and 532 nm produced a R² of 0.2718 and 0.0028, respectively (data not shown).

Bacterial Survival Versus Irradiation Dosage

The cell survival data presented as log–log plots of the viable E. coli cell density (S) in CFU/mL versus the irradiation dosage (D) in ml/cm² for 12 treatment wavelengths (12 panels) is shown in Figure 1. Each panel shows both the measured data set and a numerically determined best-fit curve. Each panel has y (where y = log S) ranging from zero to eight (over eight decades) and x-axis values (where x = log D) ranging from zero to as high as seven (up to seven decades of dose) depending on the excitation wavelength chosen. Since each panel appears to generally follow the form of a decaying exponential, this equation form was initially assumed for curve fitting purposes. Thus, the general starting curve for a curve fit to a particular wavelength λ was

\[ y = y_0 - P_2(\lambda)e^{(P_3(\lambda)x)} \quad \text{or} \]

\[ \log S = \log S_0 - P_2(\lambda)e^{(P_3(\lambda)\log D)} \]  

(1)

Figure 1. Curve fitting graphs of log dose (ml/cm²) versus Escherichia coli survival log (CFU/mL) treated to radiation of various wavelengths: (a) 250 nm, (b) 265 nm, (c) 275 nm, (d) 290 nm, (e) 300 nm, (f) 325 nm, (g) 350 nm, (h) 365 nm, (i) 375 nm, (j) 400 nm, (k) 458 nm, and (l) 488 nm.
Here $S_0$ is the normalized untreated initial cell density (CFU/mL), and log $S_0$ is the $y$-axis intercept. $S$ is the cell density after irradiation. $P_2(\lambda)$ is the wavelength dependent coefficient of the exponential, and $P_3(\lambda)$ is the coefficient of the horizontal (log $D$) coordinate. Numerically fitting the data expressed in log–log units has the advantage that each data point is weighted equally during the minimizing of the accumulated squared deviation $r^2$. The numerically calculated best-fit curve to each data set in Figure 1 was obtained using the “non-linear curve fitting tool” in the Origin 7.5 Program Tool Package (OriginLab Corporation, Northampton, MA).

For illustrative purposes, Figure 2 shows an expanded view of the 300 nm data set (also Fig. 1e) including the numerically generated best fit curve for the values of $P_2(\lambda)$ and $P_3(\lambda)$ stated in the caption. Thus, for this particular wavelength

$$\log S = 7.85 - 0.08354 e^{2.3 \log D}$$

After an initial attempt at fitting all treatment data sets, it was noted that $P_3(\lambda)$ values were always close to 2.3 ($\pm 5\%$). Setting $P_3(\lambda)$ to the fixed value of $e$ ($2.71826$) actually resulted in a reduced total deviation in the fit of $P_2(\lambda)$ versus wavelength $\lambda$ in Figure 3, to be discussed in the next section.

Since : 

$$2.3026 \log D = \ln D$$

yielding

$$\log S = \log S_0 - P_2(\lambda) e^{(\ln D)} \quad (2)$$

At 100% mortality, $S$ should be equal to 0. However, log 0 is equal to infinity. Therefore, we assume a cell concentration that falls between $\geq 0$ and $\leq 1$ CFU/mL is equivalent to 100% mortality.

Hence : 

$$\log S_0 = P_2(\lambda) D_{100\%\text{mortality}}$$

Therefore, the lethal radiation dose ($LD_{100}$) at a given wavelength is:

$$D_{100\%\text{mortality}} (or LD_{100}) = \frac{\log S_0}{P_2(\lambda)}$$

Using the value of 7.85 for $\log S_0$

$$LD_{100} = \frac{7.85}{P_2(\lambda)} \quad (3)$$

### Table 1. Best fit parameter $P_2(\lambda)$ obtained for photon wavelengths investigated.

<table>
<thead>
<tr>
<th>Wavelength, $\lambda$ (nm)</th>
<th>$P_2(\lambda)$ fit parameter</th>
<th>$\log P_2(\lambda)$</th>
<th>$\log P_2(\lambda)$ error</th>
</tr>
</thead>
<tbody>
<tr>
<td>250</td>
<td>0.27451 ± 0.02118</td>
<td>−0.56144</td>
<td>±0.03228</td>
</tr>
<tr>
<td>265</td>
<td>0.52329 ± 0.03434</td>
<td>−0.28125</td>
<td>±0.0276</td>
</tr>
<tr>
<td>275</td>
<td>0.44537 ± 0.02088</td>
<td>−0.351333</td>
<td>±0.0199</td>
</tr>
<tr>
<td>290</td>
<td>0.27691 ± 0.05898</td>
<td>−0.55766</td>
<td>±0.04556</td>
</tr>
<tr>
<td>300</td>
<td>0.08587 ± 0.00894</td>
<td>−1.06616</td>
<td>±0.02895</td>
</tr>
<tr>
<td>325</td>
<td>0.00509 ± 0.01989</td>
<td>−2.29328</td>
<td>±0.05521</td>
</tr>
<tr>
<td>350</td>
<td>0.00245 ± 0.00202</td>
<td>−2.6108</td>
<td>±0.01907</td>
</tr>
<tr>
<td>365</td>
<td>0.00265 ± 0.00077</td>
<td>−2.57675</td>
<td>±0.0819</td>
</tr>
<tr>
<td>375</td>
<td>0.00234 ± 0.00229</td>
<td>−2.63078</td>
<td>±0.01817</td>
</tr>
<tr>
<td>400</td>
<td>0.00006 ± 0.00001</td>
<td>−4.2218</td>
<td>±0.06695</td>
</tr>
<tr>
<td>458</td>
<td>$3.37 \times 10^{-6}$ ± $7.07 \times 10^{-7}$</td>
<td>−5.47237</td>
<td>±0.06182</td>
</tr>
<tr>
<td>488</td>
<td>$1.51 \times 10^{-7}$ ± $4.31 \times 10^{-8}$</td>
<td>−6.82086</td>
<td>±0.10913</td>
</tr>
</tbody>
</table>
Wavelength Dependence of $P_2(\lambda)$

Once $P_2$ was fixed to the value of $e \approx 2.3026$ in Equation (1), as already described, the simpler exponential form shown as Equation (2) remains.

$$\log S = \log S_0 - P_2(\lambda)D$$

In this equation, $P_2(\lambda)$ determines the rate of bending of the exponentially decreasing survival number (S) because it is a coefficient of $D$, and is the only remaining adjustable curve fitting parameter. Repeating the numerical fit process for all sub-graphs in Figure 1 using the Origin Software results in the data values contained in Table I. A plot of the log of $P_2(\lambda)$ values versus the corresponding radiation wavelengths $\lambda$, is shown in Figure 3. A strong linear relationship between values versus the corresponding radiation wavelengths $\lambda$ is employed. Combining log terms in Equation (2) for a decay equation provided a constant light treatment intensity $D$ and Equation (6), is consistent with the most general form of a time dependent population decay (Gray, 2004) namely

$$\frac{dS}{dt} = -\gamma S$$

and specifically

$$\frac{dS}{dt} = -2.3026 IP_2(\lambda)S$$

This work has further determined that the survivability factor ($\gamma$) contains a component $P_2(\lambda)$ that is exponentially dependent on wavelength $\lambda$.

**Bond Energies of Biomolecules**

The wavelength of a photon required to break a specific covalent bond in biological systems can be calculated from the bond energy of the covalent bond of interest. Photon energy can be determined by the equation:

$$E = h\nu = \frac{hc}{\lambda}$$

For $E$ in eV per photon, Planck’s constant $h$ is in eVs, $\nu$ is frequency of light in oscillations per second, $c$ is velocity of light in nm/s, and $\lambda$ is wavelength of radiation in nm.

Substituting $h = 4.13566743 \times 10^{-15}$ eVs and setting $c = 2.99792458 \times 10^{17}$ nm/s, for $\lambda$ in nm gives:

$$\lambda = \frac{1239.84}{E}$$

The energy required to break covalent bonds in biological systems is usually expressed in kilocalories per mole. These energy densities must be converted to eV per bond before comparison can be made to the photon energies ($E$) used in Equation (8).

Since:

1 kilocalorie = $2.6127 \times 10^{22}$ eV
1 mole = $6.023 \times 10^{23}$ items i.e., single covalent bonds in this case
1 kilocalorie / mole = 0.04337871 eV per bond

Using these relations, the P-O bond energy density of 100 kcal/mole becomes 4.3379 eV per bond. Based on Equation (8), the wavelength for a photon of this single bond is:

$$\lambda = \frac{1239.84}{4.3379} = 286.6 nm$$
The bond energy is:

$$\lambda = \frac{1239.84}{4.3379} = 285.82 \text{ nm}$$

Table II lists the characteristic energy of the major single covalent bonds in biomolecules and the equivalent photon wavelength representing each bond energy. These conversions indicate that UV and visible photons carry enough energy to break strong bonds in biological systems or otherwise induce reactions or free radicals that are deleterious to bacteria.

**UV and Visible Light Absorbance Spectrum of E. coli**

The absorbance spectrum of the E. coli cell suspension (Fig. 4) shows a gradual increase with decreasing wavelength, accompanied by a peak near 265 nm and then a rapid increase below about 240 nm. Plotted on the same graph are the single covalent bond energies for organic molecules as listed in Table II. Most of the longer wavelength absorption is likely to be single bond absorption events.

Wavelengths near that of the O–H and P–O bond energies show a strong absorption peak. At wavelengths ($\lambda$) less than 230 nm, bond absorption processes, absorption resulting in photo-emission from conjugated bonds, increasing Rayleigh scattering ($1/\lambda^4$), and even atomic ionization processes can contribute to the absorbance spectrum.

### Discussion

Despite the fact that UV radiation has been used as a disinfectant for decades, to our knowledge, there has not as yet been a predictive equation reported to quantify the killing capacity of radiation at various wavelengths. Using our survival data as a function of dose and wavelength, Equation (6), with a single fit parameter, fits all the data for E. coli for treatment wavelengths from 250 to 488 nm and a range of required dose of almost seven orders of magnitude. At a particular wavelength, the kill rate increases exponentially with dose, and as treatment wavelength increases, the coefficient of the required dose changes by an additional exponential.

The conditions chosen for illuminating the sample suspensions used in this study permitted uniform illumination of all bacterial cells in the treatment chamber. In order that the lethal dose predictions of Equation (3) and the fractional survival ratio of Equation (6) apply to larger scale applications, a similar degree of uniformity in the treatment container should be ensured. This might require a combination of large particle filtering, dilution of the sample volume and/or vigorous mixing of the treatment medium.

The peak in the killing curve in Figure 3 coincides with the 265 nm peak in the UV–visible absorbance spectrum of the E. coli cells (Fig. 4). At this wavelength, the photons can disrupt essentially all of the chemical bonds of the biomolecules in the cell samples. The short wavelength UV that can break the strong O–H, P–O, and N–H, bonds (Table I) or that can produce energetic free radicals; clearly have the most destructive effect on the cells. P–O bonds are a part of the backbone structure of nucleic acids (Becker et al., 2003) and the O–H and N–H bonds are essential to the hydrogen bonds that maintain tertiary structure of proteins and DNA (Lodish et al., 1995). One explanation for a decrease in the killing for wavelengths shorter than 265 nm is the loss of resonance in the transfer of photon energy to the system. Another factor could be due to the exponential increase in UV absorption by water at wavelengths less than 265 nm resulting in a less uniform killing dose within the sample.

One hundred percent mortality was not obtained in the visible light spectrum due to the lengthy exposure times required at the relatively low (non-heating) radiation intensities used. However, there was still significant killing

### Table II. Covalent bond energy expressed in equivalent photon wavelength units.

<table>
<thead>
<tr>
<th>Bond type</th>
<th>Bond energy$^a$ (kcal/mole)</th>
<th>Bond energy (eV/bond)</th>
<th>Photon wavelength (nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>O–H</td>
<td>110</td>
<td>4.7722</td>
<td>259.80</td>
</tr>
<tr>
<td>P–O</td>
<td>100</td>
<td>4.3384</td>
<td>285.82</td>
</tr>
<tr>
<td>C–H</td>
<td>99</td>
<td>4.2520</td>
<td>291.58</td>
</tr>
<tr>
<td>N–H</td>
<td>93</td>
<td>4.0347</td>
<td>307.29</td>
</tr>
<tr>
<td>C–O</td>
<td>84</td>
<td>3.6442</td>
<td>340.22</td>
</tr>
<tr>
<td>C–C</td>
<td>83</td>
<td>3.6009</td>
<td>344.32</td>
</tr>
<tr>
<td>S–H</td>
<td>81</td>
<td>3.5141</td>
<td>352.82</td>
</tr>
<tr>
<td>C–N</td>
<td>70</td>
<td>3.0369</td>
<td>408.26</td>
</tr>
<tr>
<td>C–S</td>
<td>62</td>
<td>2.6898</td>
<td>460.94</td>
</tr>
<tr>
<td>N–O</td>
<td>53</td>
<td>2.2994</td>
<td>539.21</td>
</tr>
<tr>
<td>S–S</td>
<td>51</td>
<td>2.2126</td>
<td>560.36</td>
</tr>
</tbody>
</table>

$^a$Bond energy values are obtained from Lodish et al. (1995).

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**Figure 4.** Absorbance spectrum of E. coli cells plotted against wavelength. The corresponding bond breaking energies at specific wavelengths are shown on the x-axis.
observed at the 458 and 488 nm laser lines (Fig. 1) after sufficient photon doses were delivered. The high doses were required at these longer wavelengths, presumably because only the low energy C–S, N–O, and S–S bonds (Table I) could be broken and these are apparently less damaging to the bacterial cells. No significant kill was observed for the longer visible light wavelengths of 515 and 532 nm for doses similar to those used at 458 and 488 nm. Using the Equations (5) and (3), one can obtain an estimate of the required lethal dose for \( \lambda \) at 520 nm of about 1.4 \( \times 10^8 \) mJ/cm\(^2\). At high intensity, a visible laser could easily deliver such a dose in a relatively short time, however, in order to prevent possible heat related damaging effects to the small samples used, such high intensities were not employed. Temperature increases in all sample runs were kept below 1°C.

The observation that the bacterial survival curves versus dose all follow an exponential relation is quite surprising, especially for the more visible region of the treatment range where only a few relatively low strength bond types can be directly altered. The exponential form requires that the reduction in survival with increase in dose be a constant fraction of the remaining survivor number, even after several decades of kill has already taken place. This suggests that to within the precision of the measurements, there is little variation in the characteristics of any of the bacteria in a treatment sample. In addition, the relatively smooth exponential increase in mortality as photon energy is increased, is also surprising. One might have expected that certain photon energies in the treatment range would have a stronger resonance killing effect due perhaps to more biologically important nearby bond energies. However, except for a slight flattening of the response in the data set between 325 and 375 nm, an exponential dependence on wavelength is the best general fit form.

While other bacterial strains and environmental conditions must be tested to explore the dependence of the survival equation on these factors, the experimental data from this study fit very well and add novel parameters (i.e., radiation wavelength and dosage) to the general time dependent population decay equation of bacteria (Eq. 7). Therefore, it is reasonable to expect that other bacterial species would conform to our predictive equation (Eq. 2) and the general population decay equation after determining new fit constants \( P_2(\lambda) \).

**Conclusion**

The survival of the *E. coli* ATCC 25922 strain subjected to various dosages of irradiation ranging from 250 to 532 nm was examined. The data showed that for treatment wavelengths from 250 to 488 nm, the viable cell density of the bacteria decreased exponentially by 7.85 orders of magnitude in most cases, with dose varying by as much as six orders of magnitude (from 1 to 6.5 log mJ/cm\(^2\)), and that a lethal dose could be deduced for any wavelength chosen (Eqs. 3 and 5). A predictive equation estimating the survival number of the *E. coli*, based on the wavelength and dose of the applied radiation was also generated (Eq. 6). The exponential dependence on dose determined after 16 h of drop plating (many life cycles of the bacteria), implies that to within the precision of the data collection and analysis, bacteria in the sample are essentially identical. The lethal photon dose required grows very close to exponentially as the wavelength increases (photon energy decreases). This appears to be related to the fact that the major covalent bonds present in this organic sample are spread throughout the UV–visible range and that even visible photons can provide adequate structural damage to compromise the metabolic viability of the cells.

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