Modeling UV-Damage to E. Coli Bacteria

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1 Introduction

Food poisoning is a very serious condition that may affect large populations and will often result in multiple deaths. Of the several microbes responsible for widespread food poisoning epidemics, the most common are strains of *E. Coli*, *Salmonella*, and *Botulinum*. Even in the industrialized world, where there exist high standards of hygiene, outbreaks of these bacteria occur frequently. The most recent example is the outbreak of *E. Coli* in Japan this August, which resulted in the death of a 10-year old schoolgirl and various ailments for over 6,000 other community members (Associate Press, 1996). The handling, storage, and distribution of food products in the modern world offers ample opportunities and time for the above mentioned microbes to grow and infest the foods. Consequently the sterilization of the food products prior to transportation and storage is imperative. The microbes can be effectively destroyed by heat, however, there are many fresh food products, e.g., fresh vegetables, fruit, and meat, that are heat-sensitive. These foods must therefore be treated by alternative methods.

Ultraviolet (UV) light is known to have bactericidal properties. Sunlight, due to its content of UV rays, has an important role in the sterilization that occurs under natural conditions ([1])]. Due to these properties, UV light radiation has found applications in control of bacteria in indoor air, water supplies, and many heat-sensitive food products, e.g., meat.

1.1 Statement of the Problem

A small company involved in meat packing uses UV radiation to sterilize the meat. The company has experimented with pulsed UV radiation versus continuous UV radiation, measuring the effectiveness of the treatment as the number of bacteria killed at varying amounts of radiation. An experimental procedure to validate the effectiveness of the UV light system involves irradiation of *E. Coli* culture in an aqueous solution on a petri dish by UV light. The preliminary results indicate that the bactericidal effect of pulsed UV radiation is superior to continuous, i.e., pulsed radiation achieved a higher kill ratio at lower power than continuous radiation, up to a point, after which increase in the amount of radiation resulted in a higher kill ratio using continuous radiation rather than pulsed. This phenomenon is illustrated in Fig. 1. Experimental results have indeed shown that the crossing of the graphs (continuous and pulsed) occurs ([10]).

The purpose of this study was to develop a mathematical model that
would explain the phenomenon described above and predict the bactericidal effect of UV radiation given the various physical characteristics of the radiation equipment and sterilization processes. The modeling efforts were restricted to experimental conditions, i.e., irradiation of E. Coli in an aqueous solution in a petri dish.
2 Effect of UV Radiation on Organisms

Radiation affects molecules of bacteria through absorption, which promotes photochemical reactions. UV light is absorbed by purines and pyrimidines, which are major components of the deoxyribonucleic acid (DNA). As they absorb photons from the light source, the molecules receive energy in discrete units called photons, one photon at a time. The energy of a photon is inversely proportional to its wavelength. The total energy a molecule absorbs is proportional to the product of the duration and intensity of radiation as well as the absorption coefficient of the material being irradiated ([1]). Radiation, i.e., absorption of photons, elevates the energy level of the molecule, which may use the energy for chemical changes, lose it as heat or fluorescence, or, if the photon has sufficient energy, the atoms of the molecule may lose an electron and become ionized. Although high-intensity UV light can ionize atoms ([9]; [6]), low-intensity UV light does not have sufficient energy to be ionizing ([1]). Since each photon has the same energy, we interpret this to mean that ionization requires the absorption of more than one photon. The likelihood of absorbing a second photon, before the energy the molecule has received by being “hit” for the first time dissipates and the molecule returns to a lower energy level, increases with radiation intensity.

The absorption rate of the nucleic acid and the bactericidal effect of UV light are functions of wavelength, which achieve their maxima at the same wavelength. The absorption rate of the nucleic acid is independent of the nature of the bacteria (Koller, 1965). The absorption maximum of DNA occurs at a wavelength of about 260 nm which subsequently causes maximum damage to the DNA structure.

While the absorption of UV light is the primary underlying mechanism of its bactericidal properties, it also poses some serious limitations to its use. Because UV light is readily absorbed, it does not penetrate into solids and penetrates into liquids only slightly ([1]). This fact is aggravated by the typically low energy of UV radiation. Another factor that limits the sterilizing effectiveness of UV radiation is the ability of the irradiated bacteria to repair the damage inflicted on their DNA strands.

2.1 Heat

There is widespread consensus in the microbiological literature that the primary mechanism behind the bactericidal properties of UV light is damage
to the DNA of the bacteria. However, there was some anecdotal evidence that the very high heat (3000 °C, as claimed) resulting from absorption of photons from high-intensity, pulsed, UV radiation would contribute to the destruction and demise of the bacteria. To rule out this possibility and to allow models to be developed on a sound biological basis, the temperature rise of a single *E. Coli* cell, surrounded by an aqueous medium (i.e., water) when hit by a 200 ms pulse of UV laser was computed. The resulting temperature rise was only about 0.01 °C. If the bacterium is completely surrounded by air, however, the temperature rise under similar radiation is 154.54°C, which is sufficient to obliterate it. If the bacterium is assumed to be on the surface of a solid, e.g., meat, the rise in its temperature is about 60 °C. It is further shown that while a very powerful UV-laser light heat exposure may kill bacteria on the surface of a medium, it is ineffective at any depth. However, since the modeling effort was concentrated on explaining the data obtained from experimental studies, heat as a mechanism of devastation was given no further consideration, (see Appendix for computations).

### 2.2 Damage to the DNA

As organisms grow by division of individual cells, the genetic information is transmitted from a generation of cells to the next by splitting of the DNA ([5]). DNA consists of two very long chains of molecules twisted together. Molecules are grouped in four major building blocks, or bases, identified as T (Thymine), C (Cytosine), G (Guanine) and A (Adenine) to encode information. A’s on one strand must face T’s on the other strand, and Cs must face Gs. When a cell divides, the two strands are separated, both containing all the information needed to build a new cell. A second strand is then completed following the pairing rule above. This redundancy allows cells to correct defects in their DNA and is essential to the process of cell division.

If UV light hits a DNA chain, it will most likely be absorbed by purine and pyrimidine molecules present in the chain. This can interfere with the normal base pairing of DNA either as altered bases or single-strand breaks. Strand breaks require large amounts of energy, however, and double-strand breaks would require large amounts of ionizing energy ([7]). The most likely consequence of UV radiation is the formation of cyclobutane pyrimidine dimers ([9]) which will interfere with the reproduction of the cell.

In the simplified approach adopted here only two major damage types found in dead irradiated cells are considered: formation of dimers and strand
breaks. Pyrimidine dimer can be understood as a chemical change on a pair of the DNA base pairs that confuses the division process and thus stops the reproduction of the cell. When the cell loses its ability to reproduce, it is functionally dead. The amount of nutrients the cell needs to survive is proportional to its volume, but the amount of nutrients it can take in is proportional to its surface area. If the cell cannot reproduce, it continues to grow until it can no longer feed itself and dies. The formation of a Pyrimidine dimer requires the energy of one photon. If the damage is done when the cell has just finished a reproduction cycle, it most likely will repair itself before the next splitting occurs. However, if the damage is inflicted on a cell that has just started splitting or doubling the DNA, it will not be able to complete the cycle and will subsequently die.

A strand break represents a more severe damage to DNA. A chemical bond in one of the two strands is broken. A certain amount of energy is required to break this bond. The energy of one photon is not enough to do this, but the energy of two photons on the same site is sufficient. We include in this damage mechanism crosslinks between two adjacent pairs and denatured sites since those are also two photon reactions and are much more rare than single-strand breaks. After intercepting a photon, the strand is in a higher energy state. If the strand loses this extra energy before another photon hits the strand site again, the second photon does not destroy the DNA. Instead, it puts the strand back into the excited state, waiting for another photon to destroy it.

2.3 Repair Mechanisms

The E. Coli bacteria has several strategies for repair of various types of damage. The repair mechanism for the most likely damage resulting from exposure to UV light, the formation of cyclobutane pyrimidine dimers, is a chemical reaction with DNA photolyase. Repair mechanisms can be as fast as picoseconds (1 picosecond = 10^{-12} s) after a photon hits a base or as slow as 2 seconds to repair a severe damage to a strand ([7]). These are complex processes that are not fully understood, so we were unable to incorporate fine details of the repair mechanism into the model. Instead, we assume that a return to a lower energy level after being hit by a photon allows the bacteria to repair the damage to the DNA. Repairs of strand breaks are assumed not to occur.
3 Model Development

The model simulates the effect of UV light on a solution of bacteria in a petri dish. The solution is assumed to be spread in a layer 2 mm thick. The irradiation is done in two ways, (1) by a continuous light or (2) by a pulsed light over the petri dish. The objective of the model is to explain the differences between the two techniques as well as the differences in bactericidal effectiveness.

The data to which the models were compared came from experiments made by [9]. The pulsed irradiation was done with a laser giving a 15 ns pulse light 5 times per second at 0.42 J/m² per pulse. The continuous irradiation was done with a UV lamp at 1 J/m² per second of exposure. Both were using a wavelength very near the maximal absorption spectrum of DNA of 260nm.

3.1 Definition of Variables

Intensity is effectively the energy density of the beam of light. When light is shined on a surface, the intensity is the rate at which energy hits a unit of area. Its unit is J/m²/s. In the continuous irradiation, the intensity is constant in time while in the pulsed irradiation the intensity is a step function: high intensity during the flash of the light and no intensity when the light is off. In order to compare the killing rates, the total number of photons received per second has to be the same in both irradiations. The type of damage inflicted on the DNA is dependent on the intensity.

A photon is a light particle. The energy of one photon is equal to $hc/\lambda$, where $h$ is Planck’s constant $(6.63 \times 10^{-34}$ J s), $c$ is the speed of light $(3.00 \times 10^8$ m/s), and $\lambda$ is the wavelength of the light. Considering a particle having a certain wavelength may be confusing. This is because particles and waves are both models for the behavior of light, but neither is sufficient to explain it entirely. The idea of light as an electro-magnetic wave is necessary to explain the interference phenomenon of light ([2]). The model of light as a particle is necessary to explain Planck’s radiation law ([3]) and the photo-electric effect ([2]).

If we use UV light with a wavelength of 260 nm, the energy of one photon is $7.65 \times 10^{-19}$ J/photon. Therefore, if the intensity of the light is $I$, the rate at which photons hit a unit area as $I/7.65 \times 10^{-19} = 1.31 \times 10^{18} \times I$. 
4 Two-Hit Model

In the simplest model, we consider only one kind of damage to the DNA, assuming it is a two-photon reaction. Moreover, it is assumed that the second photon could be absorbed anywhere on the DNA to be effective in killing the cell. This means that the probability of the first hit and the probability of the second hit are the same. We also allow a cell to repair damage done by a single photon at a given rate.

After a solution of bacteria has been irradiated, a number of healthy cells, i.e., cells with no damage, remain. Their concentration is denoted by $N(t)$. This quantity varies as a function of time of exposure. The time of exposure times the intensity is the total energy per area received by the solution of bacteria. The concentration of cells with one hit in the irradiated solution is denoted by $S(t)$. We call them sick cells. In order to kill sick cells, the cells have to receive a second photon. The concentration of dead cells, i.e., cells that received two photons during the irradiation, at time $T$ is computed as:

$$N(0) - N(T) - S(T). \quad (1)$$

The mathematical description of those concentrations in time is done by two ordinary differential equations. To derive them, consider the change of concentration for the two families.

The concentration of healthy cells changes because both healthy cells are hit by photons and become sick cells and sick cells repair themselves and become healthy cells. The concentration of sick cells changes in the following three ways: sick cells are hit by a second photon and die, sick cells repair themselves and become healthy cells, and healthy cells are hit by a photon and become sick cells. We then have the following system:

$$\begin{bmatrix}
\frac{dN}{dt} \\
\frac{dS}{dt}
\end{bmatrix} =
\begin{bmatrix}
-C_1 & C_2 \\
C_1 & -(C_1 + C_2)
\end{bmatrix}
\begin{bmatrix}
N \\
S
\end{bmatrix} \quad (2)$$

where $C_1$ is the rate at which cells received a photon and $C_2$ is the rate at which sick cells repair themselves.

If $C_1$ is a constant in time (which is the case for continuous irradiation), this system is easily solved:
4 TWO-HIT MODEL

\[
\begin{bmatrix}
N(t) \\
S(t)
\end{bmatrix} = e^{At} \begin{bmatrix}
N(0) \\
S(0)
\end{bmatrix},
\]

(3)

where

\[
A = \begin{bmatrix}
-C_1 & C_2 \\
C_1 & -(C_1 + C_2)
\end{bmatrix}.
\]

(4)

We assumed we start with all healthy cells, the initial condition of the system. If \( C_1 \) is a step function in time, being \( C_1 \) for the pulse duration, \( \tau_p = 15 \times 10^{-9} \) s, and zero for the rest of the pulse, \( \tau_r = 0.2 \) s, we compute the solution to this system in two steps, one with the UV-laser on, and one with the UV-laser off. We obtain the following solution for the effect of \( K \) pulses:

\[
\begin{bmatrix}
N(K) \\
S(K)
\end{bmatrix} = (e^{A\tau_p} \cdot e^{B\tau_r})^K \begin{bmatrix}
N(0) \\
S(0)
\end{bmatrix},
\]

(5)

where \( B \) represents the matrix of the system when the light of the UV-laser is turned off, i.e.,

\[
B = \begin{bmatrix}
0 & C_2 \\
0 & -C_2
\end{bmatrix}.
\]

(6)

4.1 Analysis of the Model

The simplest model only incorporated one type of damage to the DNA. We assumed that two photons hitting at various locations on a DNA strand was sufficient to prevent the cell from reproducing. We observed from mathematical analysis that the continuous light model and the pulsed light model produced identical graphs corresponding to the fraction of bacteria killed as a function of the rate at which the bacteria was hit by photons. This conflicted with the experimental data graph which exhibits two separate, intersecting curves (Fig. 1). Since the graph resulting from the numerical analysis should exhibit the same intersection which is present in the graphs of the experimental data, we attempted to refine our mathematical model. We considered an N-hit model, i.e., a model for which \( N \) photons are required to prevent reproduction. For various values of \( N \), the graphs again
Figure 2: One-Hit Model: Continuous versus Pulse UV Light.

showed that the continuous and the pulse cases were identical. Upon further research, we discovered two errors with this model. First, we found that in order to effect damage on the DNA, the two photons must hit at the same location on a single strand. Second, it is not sufficient to consider only strand damage.
Figure 3: Two-Hit Model: Continuous versus Pulse UV Light.

Figure 4: Twenty-Hit Model: Continuous versus Pulse UV Light.
5 The Two-Mechanism Model

In the second model, we consider the two different mechanisms of damaging the DNA. We have four kinds of cells in a solution of irradiated bacteria: (1) Healthy cells, their concentration noted \( N(t) \), (2) cells with one hit on a strand, their concentration noted \( S_2(t) \), (3) cells that had one hit on a base site, their concentration noted \( S_1(t) \), and (4) cells with one hit on a strand and one hit on a base, their concentration noted \( S_{12}(t) \).

In order to kill cells having one hit on a base, the cells have to go under splitting process. In order to kill cells having one hit on a strand, the cells have to receive a second photon on the site already hit. The killed cell concentration at time \( T \) is computed as:

\[
N(0) - N(T) - S_1(T) - S_2(T) - S_{12}(T).
\]

Following the concentration of the different kinds of cells will gives us:

\[
\begin{bmatrix}
\frac{dN}{dt} \\
\frac{dS_1}{dt} \\
\frac{dS_2}{dt} \\
\frac{dS_{12}}{dt}
\end{bmatrix}
= \begin{bmatrix}
A
\end{bmatrix}
\begin{bmatrix}
N \\
S_1 \\
S_2 \\
S_{12}
\end{bmatrix},
\]

where

\[
A = \begin{bmatrix}
-(C_1 + C_2) & C_{hF} & C_{hS} & 0 \\
C_1 & -(C_c + C_2 + C_{hF}) & 0 & C_{hS} \\
C_2 & 0 & -(C_3 + C_1 + C_{hS}) & C_{hF} \\
0 & C_2 & +C_1 & -(C_{hF} + C_{hS} + C_c + C_3)
\end{bmatrix}.
\]

We also have that \( C_1 \) is the rate at which healthy cells receive a photon on a base, \( C_2 \) is the rate at which healthy cells receive a photon on a strand, \( C_3 \) is the rate at which \( S_2 \) cells receive a second lethal photon (those rates are proportional to the intensity), \( C_c \) is the rate at which \( S_1 \) cells divide, \( C_{hF} \) is the rate at which \( S_1 \) cells repair dimers, and \( C_{hS} \) is the rate at which
5 **THE TWO-MECHANISM MODEL**

$S_2$ cells repair damaged strands. For this model we set $C_{hS} = 0$ since it is almost impossible for the cell to return to a healthy state where there is no excitation of the strands. Both the continuous and the pulsed solutions of this system have the same form of, respectively, the solutions (3, 5), where, in the present case, the matrix $B$ is given by

$$
B = \begin{bmatrix}
0 & C_{hF} & C_{hS} & 0 \\
0 & -(C_c + C_{hF}) & 0 & C_{hS} \\
0 & 0 & -C_{hS} & C_{hF} \\
0 & 0 & 0 & -(C_{hF} + C_{hS} + C_c)
\end{bmatrix}.
$$

(10)

### 5.1 Analysis of the Model

For the second model, we refined two of the assumptions made in the basic model. For the first refinement, we assumed that two photons must hit at the same location on a DNA strand to prevent the cell from reproducing. Second, we considered damage to the building blocks of the DNA. Unfortunately, our second model also failed to produce two separate, intersecting curves as observed in the experimental data graphs. Once again, the two models exhibited identical behavior.

Assuming that repair mechanisms cannot handle a very large number of damages at the same time, we included a maximum number of repairs that could take place simultaneously in our pulsed UV light, two photon model. This was implemented using the following assumptions. First, all damages were given equal probability for occurring. Second, we assume that there is no repair taking place while the light is on, a period of only 15 ns per pulse. A final modification was made concerning the healing rate of the bacterial cells. If the number of cells with one hit was less than a chosen fixed value, then the number of repairs was proportional to the number of sick cells, as in previous models. However, when the number of cells which have absorbed one photon exceeds the chosen fixed value, there is a constant maximal number of repairs which may take place. We did not find a significant difference with this modification and the experimental numerical behavior was not observed in this model.
Figure 5: Base Molecule and Strand-Break Model: Continuous versus Pulse UV Light.
6 A Nonlinear Model

Since the biochemical mechanisms affecting the repair of damaged DNA is limited, we assumed that the rate at which cells could heal themselves was limited. An examination of a similar chemical mechanism ([11]) shows that the repair rate could be modeled as \( C_2/(1 + kS_1)S_1 \), where \( k \) is a constant. This form shows a linear behavior (as assumed in the Two-Mechanism model) at low concentrations of \( S_1 \). We implemented this model, but further studies need to be done because the results were not significantly different from the results of the previous models.

7 Summary and Conclusions

To study the bactericidal properties of ultraviolet light, we considered the effect of heat from the ultraviolet laser on the bacteria and three models of DNA damage due to UV photons. From our models, we calculated that heat was not a factor in destroying the bacteria. The first of the DNA damage models was the two-hit model, where it takes two photon hits on the DNA to destroy it, and the DNA can repair damage inflicted by one photon. Our second model incorporated the effects of two types of DNA damage inflicted by the photons. The third model took into account the idea that the healing processes in the first model have a maximum rate. None of these were able to predict the intersection of the graphs for continuous and pulsed UV radiation seen in the experimental data.

The two-hit model involves a system of two first-order linear ODEs. We were able to solve this analytically and found that no matter how we varied the intensity or the frequency of the pulses, the graphs of the number of kills for pulsed and continuous radiation could never intersect. We changed this to an N-hit model, where it takes N photon hits to break the DNA strand and solved it numerically for \( N = 1, 2, \) and 20. This change still did not result in crossing of the curves.

The second model we used involves two mechanisms for DNA damage. Photons could break the DNA base pairs with one hit, or they could break a DNA strand with two hits. Each type of damage had its own repair mechanism. The base pair could be healed by a chemical reaction within the cell, while a site which previously absorbed a photon could drop out of the higher energy state before being hit a second time, effectively "healing" the damage caused by the first hit. In this model, the graphs still did not
intersect.

The third model was a variation on the first. In the previous models, the rate at which the DNA healed itself was fixed, but it is possible that the healing rate slows down when many base pairs are damaged due to limitations in the healing mechanisms. After incorporating this possible effect into the model, we plotted the solutions for various values of the UV intensity and pulse time, but the results were no different than those for the first model.

There are other effects that one might consider in further modeling how ultraviolet light kills bacteria. For example, we didn’t consider “shadowing”, which is when bacteria on the top of the petri dish block the rays from hitting bacteria at the bottom. There is a significant drop in the intensity of the UV light even in a 1 mm thick layer where the \( E. \ colib \) occupies 1of \( 4.544 \times 10^{-5} \text{ cm} \) for the radius of an \( E. \ coli \) bacterium, we found that the intensity at the bottom is about \( 10^{-7} \) of the original intensity. Thus, the high intensity pulses should be much more effective in killing the bacteria at the bottom than the continuous beam. It may be that this could account for the intersection of the two graphs, but we were unable to incorporate this into our model.

Another consideration is the distribution of DNA strand sites in the higher energy states after being hit by one photon. If the DNA strand intercepts one photon, that part of the molecule has a higher energy. The strand breaks when a second photon hits a part of the strand in the high energy state, providing enough energy to break the molecule. The probability of a cell’s DNA being broken by the second photon depends on how many pieces of the strand are excited, i.e., if only a few strand sites have high energy, the probability that a second photon hits one of those sites is very low. If most of the strand sites are excited, the probability of being broken is very high. In our second model, the probability of getting the second hit was constant. We tried to get some idea of the distribution of excited DNA strand sites, but failed to get any answers analytically or numerically. A model which takes this distribution into account would certainly be more accurate.
References


Appendix

We study the heating effects of irradiating food with a ultraviolet light laser in order to see if it is a meaningful sterilization method. We make the following assumptions:

1. We consider a 200 ms pulse of an ultraviolet laser which emits an energy density of 3.8 J/cm$^2$.
2. We shall assume that the bacterium and the medium it is in closely resemble water in regard to their physical characteristics.

Physical Parameters and variables of the model.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
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<tbody>
<tr>
<td>$\rho$</td>
<td>0.99821 g/(cm$^3$)</td>
</tr>
<tr>
<td>$D_1$</td>
<td>598.4 mW/K m</td>
</tr>
<tr>
<td></td>
<td>59840 cm g s$^{-3}$/K</td>
</tr>
<tr>
<td>$D_2$</td>
<td>2.7882 x 10$^{-5}$ J/m s K</td>
</tr>
<tr>
<td></td>
<td>2.7882 cm g s$^{-3}$/K</td>
</tr>
<tr>
<td>$c$</td>
<td>4.1818 J/g K</td>
</tr>
<tr>
<td></td>
<td>4.1818 (cm$^2$) s$^{-3}$/K</td>
</tr>
<tr>
<td>Vol. of</td>
<td>3.93 x 10$^{-13}$ (cm$^3$)</td>
</tr>
<tr>
<td>a bacterium</td>
<td></td>
</tr>
<tr>
<td>$r_b$</td>
<td>4.544 x 10$^{-5}$ cm</td>
</tr>
<tr>
<td>$q$</td>
<td>J/cm$^3$ s K</td>
</tr>
<tr>
<td>$T$</td>
<td>temperature [K]</td>
</tr>
</tbody>
</table>

MODEL 1

We first model how hot a bacterium in a vacuum gets when exposed to a celcius in vacuum when exposed to ultraviolet laser. Let $T(t)$ denote the temperature of the bacterium at time $t$-seconds. We first need to convert the energy density of the laser to energy density per unit volume. Now a laser emitting an energy density of 3.8 J/cm$^2$ is equal to a laser emitting 15000$\rho c$ J/cm$^3$ s K. Since the heat conductivity in a vacuum is zero, $T(t)$ satisfies the differential equation

$$\rho c \frac{dT}{dt} = 15000\rho c,$$
$T(0) = 0,$

where the temperature $T$ is measured in Celcius and time $t$ is measured in seconds. Solving the above differential equation we obtain

$$T = 15000t.$$  

Since the duration of the pulse is 200 milliseconds, it follows that the temperature of the bacterium reaches $T(.2) = 3000^\circ C$.

**MODEL 2**

We shall denote, again, by $T$ the temperature at a point in the medium in the following.

Next, we model the heat effect of exposing a bacterium in an infinite medium of water by a 200 millisecond pulse of our laser by the radially-symmetric steady state heat-conduction equation as follows:

$$D_1 \left( \frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} \right) + q = 0, \; r < r_b,$$

$$\frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} = 0, \; r > r_b,$$

$$\frac{dT}{dr}(0) = 0,$$

$$T(\infty) = 0,$$

$$T(r_b-) = T(r_b+),$$

$$(D_1 \frac{dT}{dr})(r_b-) = (D_1 \frac{dT}{dr})(r_b+)$$

Now, for $r > r_b$ we have

$$\frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} = 0,$$

$$\frac{d}{dr} \left( r^2 \frac{dT}{dr} \right) = 0,$$

$$r^2 \frac{dT}{dr} = A \text{ (a constant)},$$

$$T = \frac{A}{r} + A_1,$$

$$T(\infty) = 0 \implies A_1 = 0,$$

$$T = \frac{A}{r}.$$
Next, for \( r < r_b \) we have
\[
D_1 \left( \frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} \right) + q = 0,
\]
\[
D_1 \frac{d}{dr} \left( r^2 \frac{dT}{dr} \right) + qr^2 = 0,
\]
\[
D_1 r^2 \frac{dT}{dr} + \frac{1}{3} qr^3 = B \text{ (a constant)},
\]
\[
\frac{dT}{dr}(0) = 0 \implies B = 0,
\]
\[
D_1 \frac{dT}{dr} + \frac{1}{3} qr = 0,
\]
\[
D_1 T + \frac{1}{6} qr^2 = B \text{ (a constant)}.
\]

We now apply the continuity conditions when \( r = r_b \) to obtain
\[
\frac{1}{D_1} (B - \frac{1}{6} qr_b^2) = -\frac{A}{r_b},
\]
\[
D_1 \frac{A}{r_b^2} = -\frac{q}{3} r_b,
\]
\[
\frac{1}{D_1} (B - \frac{1}{6} qr_b^2) = \frac{q}{3D_1} r_b^2,
\]
\[
B = \frac{q}{2} r_b^2.
\]

We thus obtain the temperature at the center of the bacterium to be
\[
T(0) = 0.0108^\circ C.
\]

**Conclusion:** The temperature of the bacterium barely rises. The explanation is that the heat is conducted away by the surrounding medium. Note that we have assumed that the bacterium and the surrounding medium have the same thermal characteristics.

If we assume that the bacterium is surrounded by air then we need to solve the analogous model given by
\[
D_1 \left( \frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} \right) + q = 0, \quad r < r_b,
\]
\[
\frac{d^2T}{dr^2} + \frac{2}{r} \frac{dT}{dr} = 0, \quad r > r_b,
\]
\[
\frac{dT}{dr}(0) = 0,
\]
\[
T(\infty) = 0, \\
T(r_s-) = T(r_s+), \\
(D_1 \frac{dT}{dr})(r_s-) = (D_2 \frac{dT}{dr})(r_s+). 
\]

Solving this model, as done above, we obtain the temperature at the center of the bacterium to be
\[
T(0) = 154.54^\circ C.
\]

**MODEL 3**

It is known among food scientists that UV-laser light radiation as a method of sterilization for food is effective only when the bacterium is present on the surface of the food and the light source is very near to the food, something on the order of 6 inches.

We shall develop a mathematical model which confirms this.

We model this problem as a one dimensional heat diffusion problem, with the depth into the medium as the only meaningful space variable. All points in a plane parallel to a horizontal plane being assumed to be at the same temperature.

We take \( q_1 \), the energy of the laser to be 3.8 \( J/cm^2 \). Let \( T(t, y) \) denote the temperature of the food at time \( t \) and at a depth of \( y \). We measure time in seconds and depth in centimeters. The differential equations of our model are:

\[
\rho c \frac{\partial T}{\partial t} = D_1 \frac{\partial^2 T}{\partial y^2}, \\
T(0, y) = 0, \ T(t, \infty) = 0, \ y \geq 0, \ t \geq 0, \\
-D_1 \frac{\partial T}{\partial y}(t, 0) = q_1, \ t \geq 0.
\]

We note that the units of \( q_1 \) are \( J/cm^2 \) s K. We set
\[
D = \frac{D_1}{\rho c}, \\
\tilde{q} = \frac{q_1}{\rho c}.
\]
We see that the values of $D$, $\tilde{q}$ are given by

\[
D = \frac{D_1}{\rho c} = \frac{59840}{4.1818 \times .9981 \times 10^7} = .001433686748,
\]
\[
\tilde{q} = \frac{q_1}{\rho c} = \frac{3.8}{\rho c} = .910429418938.
\]

In terms of $D$ and $\tilde{q}$ the differential equations of the model become

\[
\frac{\partial T}{\partial t} = D \frac{\partial^2 T}{\partial y^2}, \quad T(0, y) = 0, \quad T(t, \infty) = 0, \quad y \geq 0, \quad t \geq 0,
\]
\[
-D \frac{\partial T}{\partial y}(t, 0) = \tilde{q}, \quad t \geq 0.
\]

Now, $T(t, y) = \sqrt{t} f\left(\frac{y}{\sqrt{D}}\right)$ is a solution of the above equations, where $f(x)$ is a function of one variable $x$ and satisfies the following differential equation

\[
f''(x) + \frac{1}{2} f'(x) - \frac{1}{2} f(x) = 0,
\]
\[
f(\infty) = 0,
\]
\[
f'(0) = -\frac{\tilde{q}}{D}.
\]

It is easy to see (using Maple) that

\[
f(x) = C_1 x + C_2 e^{-\frac{x^2}{2}} + \frac{1}{2} \sqrt{\pi} \text{erf}\left(\frac{1}{2} x\right)
\]

is the general solution of the above differential equations. Using the boundary conditions, we obtain that

\[
C_1 = -\frac{\tilde{q}}{\sqrt{D}},
\]
\[
C_2 = \frac{2}{\sqrt{\pi} \sqrt{D}} 032.
\]

Thus,

\[
T(t, y) = \sqrt{t} f\left(\frac{y}{\sqrt{D} t}\right),
\]
\[
= \frac{\tilde{q}}{\sqrt{D}} \sqrt{t} \left[-\frac{y}{\sqrt{D} t} + \frac{2}{\sqrt{\pi}} (e^{-\frac{y^2}{2\sqrt{D} t}} + \frac{1}{2} \sqrt{\pi} \frac{y}{\sqrt{D} t} \text{erf}\left(\frac{y}{2\sqrt{D} t}\right)]\right].
\]
It follows that the temperature at the center of the bacterium which is at a depth of $r_b$ after 200 millisecond of UV-laser exposure is given by

$$T(0, r_b) = \frac{\bar{q}}{\sqrt{D}} \left[ -\frac{r_b}{\sqrt{D}} + \frac{2\sqrt{2}}{\sqrt{\pi}} \left( e^{-\frac{r_b^2}{4D}} + \frac{1}{2} \frac{r_b}{\sqrt{2D}} \text{erf}\left( \frac{r_b}{2\sqrt{2D}} \right) \right) \right],$$

$$= \frac{\bar{q}}{\sqrt{D}} \left[ -\frac{r_b}{\sqrt{D}} + \frac{2\sqrt{2}}{\sqrt{\pi}} e^{-\frac{r_b^2}{4D}} + \frac{r_b}{\sqrt{D}} \text{erf}\left( \frac{r_b}{2\sqrt{2D}} \right) \right],$$

$$= 12^\circ C$$ approximately.

**Conclusion:** Since we assume that the food is at room temperature of $20^\circ C$ the UV-laser exposure of 200 ms will cause the food temperature to rise about $32^\circ C$. It can be seen from our work that if we use a laser which emits an energy density of $19 J/cm^2$, a laser five times more powerful, then the temperature of the bacterium will rise to approximately $160^\circ F$, an FDA standard.
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