
Mechanism of UV Disinfection

This chapter explores the process of how absorbed UV photons inactivate microorganisms so effectively. It is important to use the term *inactivate*, rather than *kill* because microorganisms inactivated by UV light are typically still metabolically *alive*.

The process starts with the absorption of UV photons in a given microorganism (First Law of Photochemistry, Chapter 2). Absorption of UV light by proteins in the outer cell membranes with high UV doses (fluence) ultimately leads to the disruption of these membranes and hence, the death of the cell arising from leakage of the protoplasm. However, at much lower UV dose (fluence) levels, absorption of UV photons by DNA (or RNA in some viruses) can disrupt the ability of the microorganism to replicate. *Cells that cannot replicate cannot cause infection.*

Most government regulations require that UV equipment inactivate microorganisms to a specific level. For example, Austria and Germany require four orders of magnitude, that is, 4 log (or 99.99 percent) inactivation for all microorganisms. This means that only one microorganism in 10,000 will remain viable. Chapter 4 provides an overview of UV disinfection regulations.

INACTIVATION MECHANISMS

Absorption of UV Light by Cell Components

As UV light (200–300 nm) passes through a microorganism, it is absorbed by various components in the cell, but only proteins and the nucleotides comprising DNA and RNA absorb a significant amount of UV light in this wavelength range. The relative absorbance of proteins versus that of nucleotides in a microorganism is shown in Figure 3-1. It is clear that below 230 nm, the proteins absorb most of the UV light; however, above 230 nm, absorption is dominated by the nucleotides. It should also be noted: the only amino acids that absorb significantly above 210 nm are the aromatic amino acids: tryptophan, phenylalanine, and tyrosine. These three amino acids have a small absorption peak at around 280 nm, with tryptophan being the strongest absorber at 280 nm. For UV disinfection of drinking water, the water itself strongly absorbs light below 230 nm, which makes these wavelengths unavailable to inactivate the microorganisms.

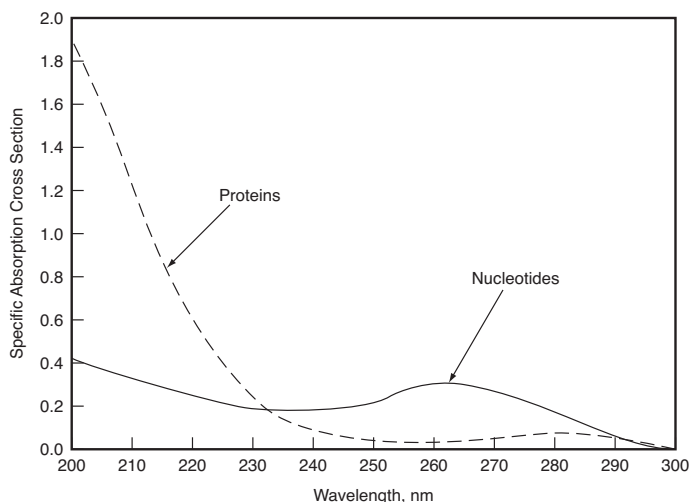


Figure 3-1 Specific absorption cross section versus wavelength for proteins and nucleotides within the cell of a microorganism. Here the dry weight percentages are protein (70%), nucleotides (20%), and other components (10%) (e.g., lipids).

Another factor to consider is that the overall absorbance of a microorganism is rarely more than 0.02 and usually much less because the optical path length in a cell is so small ($< 1 \mu\text{m}$). Thus, less than 5 percent (usually $< 1\text{--}2$ percent) of the UV incident on a microorganism is absorbed. Under these conditions, all absorbers behave independently. That is, it is not possible for the protein in the outer membrane of a cell to act as a *filter* to shield the UV light from the nucleus in the center of the cell.

Although UV light can be absorbed by proteins (mostly in the outer cell membranes), very high UV doses (fluences) are required, compared to those necessary to disrupt the DNA and RNA. Thus, the important fact is that almost all UV inactivation arises from UV absorption by nucleotides. Therefore, to understand the mechanism of UV inactivation of microorganisms, the structure and photochemistry of DNA and RNA need to be examined.

Structure and Photochemistry of DNA and RNA

DNA is the fundamental template for life. It is a polymer of nucleotides in a double-stranded helix linked by a sequence of four constituent bases (adenine, cytosine, guanine, and thymine), which constitute the genetic code. These form base pairs (adenine with thymine and cytosine with guanine) held together by hydrogen bonds. This is the glue that binds the two strands of DNA.

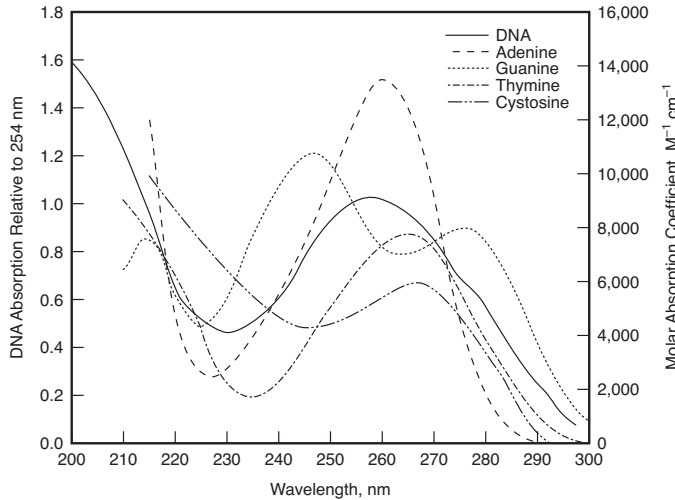


Figure 3-2 Absorption spectra of nucleotides and of DNA

RNA has several roles in a cell. Messenger RNA (mRNA) is transcribed from DNA and carries the genetic information from DNA to the ribosomes where proteins are synthesized. Transfer RNA (tRNA) binds with specific amino acids and carries them to the ribosomes for protein synthesis. RNA has four types of nucleotides: adenine, cytosine, and guanine, but uracil replaces thymine.

Most bacteria and protozoa contain DNA in the cell nucleus. Viruses are subcellular entities that can only reproduce by infecting specific host cells. They consist of a DNA or RNA nucleotide helix with a protein outer shell. The DNA or RNA in viruses can be single stranded or double stranded.

All of the nucleotides in DNA or RNA absorb UV light between 200–300 nm (see Figure 3-2). Of the four essential nucleotides in DNA, thymine undergoes a unique photochemical reaction (see Figure 3-3).¹ If two thymine bases are adjacent to each other, absorption of a UV photon by one of the thymines leads to a chemical bond between the two thymine molecules (called a *thymine dimer*).

The formation of thymine dimers (and other similar dimers) disrupts the structure of the DNA, so that if a critical number of dimers is formed,² the DNA cannot replicate. This prevents infection and is the fundamental mechanism of UV disinfection. Figure 3-4

- 1 Cytosine also forms dimers; however, the quantum yield is much lower than that of thymine.
- 2 Oguma et al. (2002) has shown that the generation of approximately 100 thymine dimers is sufficient to inactivate the entire DNA chain.

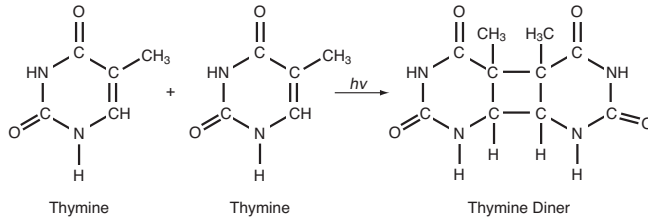


Figure 3-3 Photochemical dimerization of two thymine bases

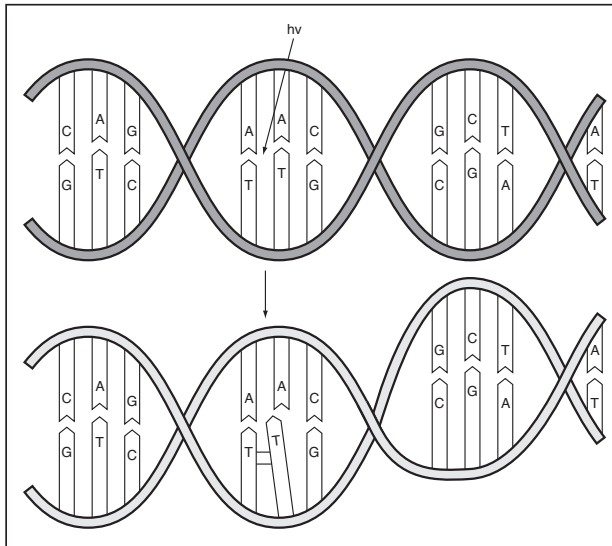


Figure 3-4 Double-stranded DNA chain showing how the formation of thymine dimers disrupts the structure of the chain (A = adenine; G = guanine; T = thymine; C = cytosine)

illustrates the disruption of a DNA chain. For viruses that contain only RNA, a similar photochemical dimerization reaction takes place between two uracil bases.

Viruses can be single- or double-stranded DNA or RNA. There does not appear to be any relation between the *strandedness* or the presence of DNA or RNA with the UV sensitivity of the virus.

Action Spectra and the Germicidal Factor

The relative inactivation response of a microorganism to UV light is determined by the ability of the UV light to be absorbed, causing inactivation of the organism through

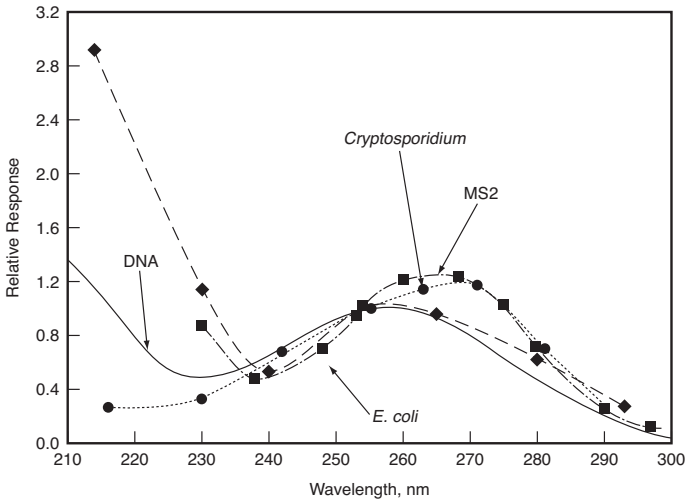


Figure 3-5 Action spectra (relative response versus wavelength) for DNA (—) (von Sonntag and Schuchmann 1992); MS2 coliphage (-----) (Mamane-Gravetz et al. 2005); *Cryptosporidium* (——) (Linden et al. 2001); and *E. coli* (-----) (Gates 1930)

photochemical reactions, such as the dimerization of adjacent thymine pairs in the DNA chains. This relative response is called the *inactivation action spectrum*. Figure 3-5 shows the action spectra of some microorganisms, along with the relative spectrum of DNA. It should be noted that the maximum germicidal effectiveness occurs around 260 nm. The differences in the action spectra probably arise from different compositions of nucleotides in their DNA and RNA.

In many models of the influence of UV light on microorganisms, it is necessary to include the relative germicidal effectiveness of the UV light at various wavelengths. This is particularly true for polychromatic light sources, such as medium-pressure UV lamps. For this purpose, the *germicidal factor* (GF) has been introduced, such that the GF = 1.000 at 254 nm, the principal wavelength emitted by a low-pressure UV lamp. If the action spectrum for a microorganism is available, it can be normalized to 1.000 at 254 nm. However, in most cases, the action spectra are not known; then the relative absorption spectrum of DNA is often used as the GF. The use of the GF is particularly important for UV equipment using UV lamps emitting polychromatic UV light (e.g., medium-pressure lamps).

REACTIVATION MECHANISMS

Some microorganisms (particularly bacteria) have mechanisms that can repair or bypass thymine dimer lesions in the DNA chain during the replication process. Some viruses are able to reactivate by using the reactivation enzymes of their host. *Giardia* and

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Cryptosporidium have not yet been shown to reactivate such that they regain infectivity (Linden et al. 2002, Shin et al. 2001, and Oguma et al. 2001). These mechanisms are divided into *dark* and *light* mechanisms. The latter are called *photoreactivation* and are activated by near UV and short-wavelength visible light. Harm (1980) has a good discussion of reactivation mechanisms, which is summarized in the following section.

Dark Reactivation Mechanisms

Most bacteria have some reactivation potential in the dark. Various mechanisms have been proposed by which a microorganism can repair DNA damage in the dark:³

- *Replacement of UV-damaged nucleotides.* In this case, the UV photoproduct (e.g., thymine dimer) and a sequence of adjacent nucleotides are removed from the DNA and a corresponding sequence is resynthesized.
- *Combination of undamaged regions in replicating DNA molecules.* Undamaged regions of DNA are replicated and reconstituted, so that an intact, double-stranded DNA, identical with the original undamaged DNA, is formed. This is possible because the complete information for a DNA molecule exists totally in each single strand. Thus, one strand can function even if the other strand is damaged.

Photoreactivation Mechanisms

It is possible to have *light-induced reversal of the UV damage*. This is also called *photoenzymatic repair*. This mechanism is the most prevalent reactivation mechanism and was discovered independently in bacteria by Kelner (1949a,b) and in bacteriophages by Dulbecco (1949, 1950). Here, a special light-activated enzyme, *photolase*, splits the thymine dimers, restoring the original structure on the DNA.

This process is triggered by the absorption of UVA light and is thus called *photoreactivation*. This repair mechanism can be overcome, but this requires a higher UV dose (fluence). This is typically not of concern in water treatment systems that are totally covered; however, if water is stored in open ponds or reservoirs exposed to sunlight, photoreactivation must be considered. Photoreactivation may be particularly important for the UV treatment of wastewater, where the effluent is discharged into a river or lake. On the other hand, drinking water is rarely exposed to sunlight, and photoreactivation is unlikely.

If a residual disinfectant (e.g., chlorine or chloramines) is maintained in the distribution system, photoreactivation is not a concern because reactivated microorganisms will be inactivated by the residual disinfectant.

³ The meaning of the term *dark* in this context means mechanisms that do not require light activation.

UV SENSITIVITY OF MICROORGANISMS

Microorganisms exhibit a wide variation in their sensitivity to UV light. The measurement of the UV sensitivity of microorganisms at a specific wavelength, or narrow band of wavelengths, is performed through the determination of a UV dose (fluence)–response curve.

Collimated Beam Tests

The UV sensitivity of microorganisms is determined using a *collimated beam test*, where a sample suspension of the microorganisms⁴ is exposed to UV light (usually in an open petri dish) in a collimated beam apparatus where the sample is exposed to beams of UV light that are almost parallel.⁵ Figure 3-6 illustrates two types of collimated beam apparatus. Such an apparatus can be constructed from commonly available materials or purchased from a vendor.

In a properly conducted determination of the UV dose (fluence)–response curve for a microorganism,⁶ where the sample is well stirred and proper corrections are made, an accurate determination of the average irradiance (fluence rate) E_{avg} in the water suspension can be made from the incident irradiance as determined using a calibrated radiometer. The product of E_{avg} and the exposure time in seconds gives the delivered UV dose (fluence) F . For each exposure time, the sample suspension is sent to a microbiological laboratory where the concentration of viable organisms is determined. A detailed operating protocol for such measurements has been specified by Bolton and Linden (2003), and Excel spreadsheets are available on the IUVA web site (www.iuva.org; for members) or from Jim Bolton (jbolton@boltonuv.com).

UV Dose (Fluence)–Response Curves

Figure 3-7 shows an example of a UV dose (fluence)–response curve. The UV dose (fluence)–response curve can exhibit up to three phases. For many bacteria, there exists a *shoulder* phase, such as that seen for *B. subtilis* (see Figure 3-7), where $\log(N/N_0)$ changes only slowly with the UV dose (fluence) at the beginning. This is followed by a *semilog* phase, where $\log(N/N_0)$ decreases linearly with UV dose (fluence). Finally, there may exist a *saturation* phase, in which $\log(N/N_0)$ changes only slowly with UV dose (fluence).

4 Hereinafter, this will be referred to as the *suspension*.

5 The term *collimated beam apparatus* is widely used, but it is important to realize that the beam is not truly collimated, in the sense that all rays are parallel. The beam does diverge, and thus there is a need for a divergence factor. Often the term *quasi* collimated beam is used to reflect this reality.

6 Unfortunately, until recently most response curves were based on the incident irradiance, rather than the average irradiance in the microorganism suspension. Thus a significant part of the published literature should be considered as upper limits because the irradiance has been overestimated.

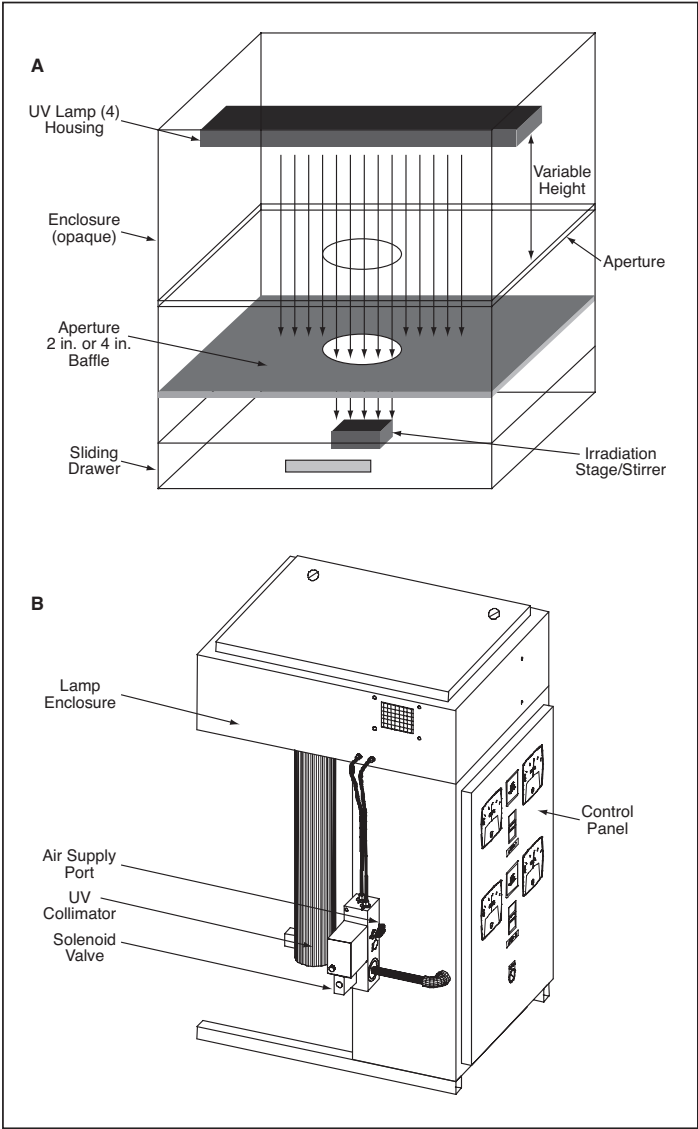


Figure 3-6 (A) Home-made collimated beam apparatus (courtesy of Prof. Linden, private communication); (B) Commercial collimated beam apparatus (courtesy Calgon Carbon Corporation, Pittsburgh, Pa.)

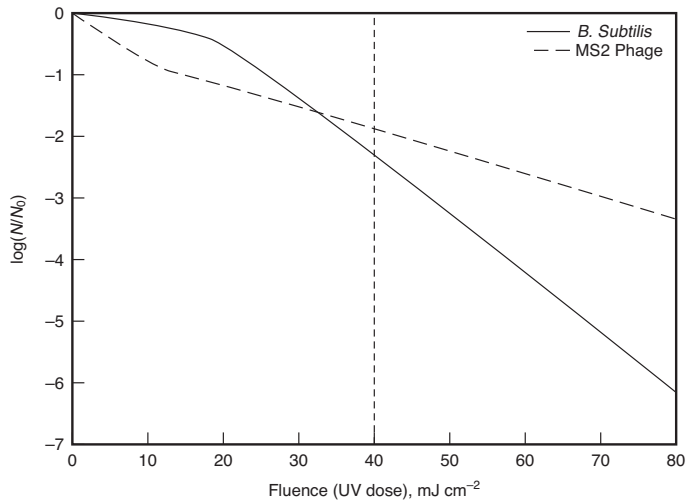


Figure 3-7 UV dose-response curves for *B. subtilis* and the virus MS2 colliphage

The *shoulder* phase is thought to be associated with DNA repair mechanisms, or it may arise from the requirement of several *hits* (DNA damage sites) before a microorganism becomes inactivated (Roessler and Severin 1996). The saturation phase may arise from several causes, such as microorganisms clumping or associating with particles (this is particularly prevalent in wastewater systems).

The *semilog* phase is characterized by the rate equation

$$\log\left(\frac{N}{N_0}\right) = -kF \tag{Eq. 3-1}$$

where *k* is a standard fluence-based rate constant [cm² mJ⁻¹ or m² J⁻¹] that is characteristic of the microorganism, and *F* is the UV dose (fluence). Other more sophisticated equations have been developed to model the other two phases (Roessler and Severin 1996).

Relative UV Sensitivity of Pathogenic Microorganisms

The UV sensitivity of pathogenic microorganisms varies considerably. The currently accepted order is

$$\begin{matrix} \text{bacteria} \approx \text{protozoa} > \text{most viruses} > \text{bacteria spores} > \text{adenovirus} > \text{algae} \\ \text{(most sensitive)} & \text{-----} & \text{(least sensitive)} \end{matrix} \tag{Eq. 3-2}$$

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Adenovirus is listed separately because it is unique among waterborne viruses because it is the least sensitive to LP UV light of any virus.⁷ At present, little is known about the reason for this insensitivity.

Tables 3-1a, 3-1b, and 3-1c present a compilation of data on the UV sensitivity of pathogenic microorganisms, as determined in a bench-scale collimated beam apparatus. The UV doses (fluence) listed are for 4 logs (99.99%) inactivation. Note that most bacteria are capable of photoreactivation and thus require a higher UV dose (fluence) for inactivation when near-UV and visible light are present.

Protozoa

Protozoa represent a class of pathogens (much larger in size than bacteria) that have become a threat to distributed water systems. The two most important pathogens are *Giardia lamblia* (causing giardiasis) and *Cryptosporidium parvum* (causing cryptosporidiosis). These pathogens are regulated by USEPA (as described in Chapter 4).

Giardia lamblia

Giardia lamblia can infect the intestinal tract of most fish, reptiles, birds, and mammals. *Giardia* cysts are quite large (about 10–15- μ m-long and oval in shape) compared to bacteria and are often found in rivers and streams exposed to wild life; for this reason the gastrointestinal disease giardiasis is commonly referred to as *beaver fever*.

Cryptosporidium parvum

Cryptosporidium parvum can infect the intestinal tract of most fish, reptiles, birds, and mammals. When ingested, *Cryptosporidium* oocysts (about 3–5 μ m in diameter) cause mild to severe diarrhea but can be fatal to immunocompromised individuals, such as those with HIV/AIDS. Large-scale outbreaks have been traced to water contaminated by agricultural runoff, particularly from large-scale livestock operations.

Since the major outbreak of cryptosporidiosis in Milwaukee, Wis. in 1993, where more than 400,000 persons were infected, *Cryptosporidium* has become the focal point of the new USEPA surface water regulations (LT2ESWTR) (USEPA 2006).

UV Sensitivity of Protozoa

Prior to 1998, protozoa would have been placed well to the right in Eq. 3-2 because it was widely perceived that protozoa (e.g., *Cryptosporidium* or *Giardia*) were very insensitive to UV light. This perception arose because the assays used to assess the viability of protozoa were based on UV damage to the cell membranes rather than on an infectivity

7 This apparently is only true for LP UV light (254 nm). Linden et al. (2007) have presented evidence that when an MP UV lamp is used, the UV dose (fluence) required for 4-log inactivation of adenovirus can be as low as 60 mJ cm⁻².

Table 3-1a Bench-scale UV dose (fluence) at 254 nm required for 4-log (99.99%) inactivation of various bacteria*.[†]

Bacterium	UV Dose (Fluence) (mJ cm ⁻²) for 4-log (99.99%) Inactivation	
	Without Photoreactivation	With Photoreactivation [‡]
<i>Aeromonas hydrophila</i>	5	NA
<i>Bacillus anthracis</i> spores [§]	>60	—
<i>Bacillus subtilis</i> spores	80	—
<i>Enterobacter cloacae</i>	10	33
<i>Enterocolitica faecium</i>	17	20
<i>Campylobacter jejuni</i>	4.6	NA
<i>Citrobacter diversus</i>	11.5	NA
<i>Clostridium perfringens</i>	23.5	NA
<i>Corynebacterium diphtheria</i>	6	NA
<i>Coxiella burnetii</i>	3	NA
<i>Escherichia coli</i> ATCC 11229	10	28
<i>Escherichia coli</i> O157:H7	6	25
<i>Escherichia coli</i> wild type	8.1	NA
<i>Klebsiella pneumoniae</i>	20	31
<i>Klebsiella terrigena</i>	11	NA
<i>Legionella pneumophila</i>	9.4	NA
<i>Mycobacterium smegmatis</i>	20	27
<i>Mycobacterium tuberculosis</i>	20	NA
<i>Pseudomonas aeruginosa</i>	11	19
<i>Pseudomonas mallei</i>	14.5	NA
<i>Salmonella anatum</i> (from human feces)	15**	NA
<i>Salmonella enteritidis</i> (from human feces)	10	NA
<i>Salmonella typhi</i> ATCC 19430	8.2	NA
<i>Salmonella typhimurium</i> (from human feces)	9	NA
<i>Salmonella marcescens</i>	13	30
<i>Shigella dysenteriae</i> ATTC 29027	3	NA
<i>Shigella sonnei</i> ATTC 9290	8.2	NA
<i>Staphylococcus aureus</i>	10.4	NA

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Table 3-1a Bench-scale UV dose (fluence) at 254 nm required for 4-log (99.99%) inactivation of various bacteria*† (continued)

Bacterium	UV Dose (Fluence) (mJ cm ⁻²) for 4-log (99.99%) Inactivation	
	Without Photoreactivation	With Photoreactivation‡
<i>Streptococcus faecalis</i> ATTC 29212	11.2	NA
<i>Vibrio cholerae</i> ATTC 25872	2.9	21
<i>Yersinia enterocolitica</i> ATTC 27729	4.6	NA

* Data extracted from Hoyer (1998) and Chevrefils et al. (2006).
† The UV doses (fluences) listed in this table were obtained at the bench scale and are approximate for the bacterium listed. The UV doses (fluences) do vary somewhat among various strains of a given bacterium.
‡ “With photoreactivation” means that the cell suspension as has been exposed to visible and/or near-UV light after the UV exposure.
§ Nicholson and Galeano (2003).
** For 3 log inactivation.

Table 3-1b Bench-scale UV dose (fluence) at 254 nm required for 4-log (99.99%) inactivation of various viruses*†

Virus	Classification	UV Dose (Fluence) (mJ cm ⁻²) for 4-log (99.99%) Inactivation	
		Without Photoreactivation	With Photoreactivation‡
Adenovirus Type 40	dsDNA	124§	—
Adenovirus Type 41	dsDNA	112§	—
B40-8 phage		28	—
Coxsackievirus B5	ssRNA	36	—
Hepatitis A HM175	ssRNA	16	—
Hepatitis A	ssRNA	21	—
MS2 Bacteriophage	ssRNA	62	—
Poliovirus Type 1	ssRNA	27	—
PRD-1 phage		30	—
ΦX174 phage	ssDNA	10	—
Rotavirus SA11	dsRNA	36	—
S. aureus phage A994		38	—

* Data extracted from Hoyer (1998) and Chevrefils (2006).
† The UV doses (fluences) listed in this table were obtained at the bench scale and are approximate for the virus listed. The UV doses (fluences) do vary somewhat among various strains of a given virus.
‡ It has been found that generally viruses do not undergo photoreactivation.
§ The USEPA has set a 4-log inactivation UV dose of 186 mJ/cm² as the virus limit, based on the inactivation of adenovirus but taking into account an 80-percent credible interval.

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Table 3-1c Bench-scale UV dose (fluence) at 254 nm required for 4-log (99.99%) inactivation of various protozoa*†

Protozoa	UV Dose (Fluence) (mJ cm ⁻²) for 4-log (99.99%) Inactivation	
	Without Photoreactivation	With Photoreactivation‡
<i>Giardia lamblia</i>	<10	—
<i>Giardia muris</i> §	<10	—
<i>Cryptosporidium parvum</i>	<10	—

* Data extracted from Clancy et al. (2000), Craik et al. (2000, 2001), and Chevretils et al. (2006).

† The UV doses (fluences) listed in this table were obtained at the bench scale and are approximate for the protozoan listed.

‡ It is generally not known whether these specific protozoa undergo photoreactivation.

§ Not considered to be a human pathogen.

assay. The following quote from the USEPA illustrates the widely held perception (USEPA 1996):

Ultraviolet light is an effective disinfection agent for drinking water at sufficient intensity and appropriate wavelength and exposure time. It is suitable for use on clean water sources more than it is for suspended matter-containing water. The order of disinfection resistance with few exceptions is bacteria (least resistant) < viruses < bacterial spores < protozoan oocysts (most resistant).

In 1997 and 1998, a unique mix of photochemists, photobiologists, and parasitologists worked together to conduct a series of experiments on *Cryptosporidium*, involving an in vivo assay, in which oocysts exposed to UV light were fed to neonatal mice. The advantage of this assay was that it provided a test that was focused on the ability of UV-treated oocysts to cause an infection in the mice. The results were remarkable in that almost no mice were infected by UV-treated oocysts even at very low UV doses (fluences). A paper describing this work was presented in June, 1998 (Bolton et al. 1998) announcing the new results (see also Bukhari et al. 1999). Research from many laboratories (Clancy et al. 2000; Craik et al. 2000, 2001; Linden et al. 2001; Mofidi et al. 2001; Shin et al. 2001; Rochelle et al. 2002; Morita et al. 2002) has confirmed that *Cryptosporidium* and *Giardia* can be inactivated by more than 4 logs at UV doses (fluences) as low as 10 mJ cm⁻² or less.

As a result of this research, the attitude of the USEPA changed drastically such that in the new LT2ESWTR regulations (USEPA 2006), UV disinfection is treated as the best available technology.

UV DOSE (FLUENCE) IN A UV REACTOR

It is important to realize that at the bench scale in a collimated beam apparatus, all the microorganisms receive essentially the same UV dose (fluence) because the suspension is well stirred. This is not the case in a UV reactor where microorganisms can traverse a

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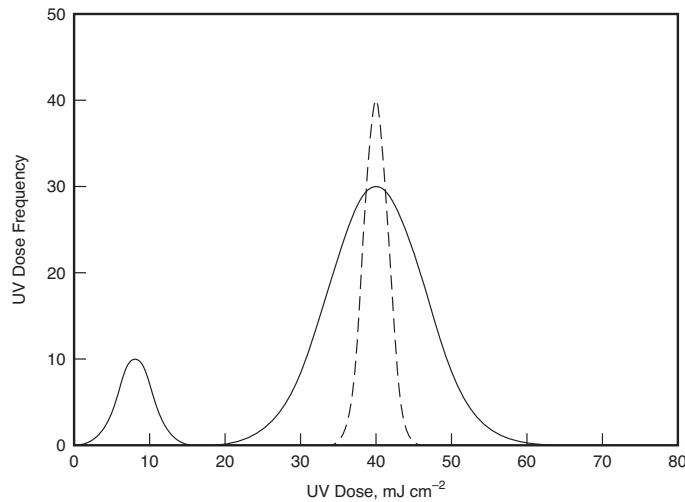


Figure 3-8 Two possible UV dose distributions

variety of paths through the reactor. The total UV dose (fluence) experienced by each individual microorganism is the product of the irradiance (fluence rate) in each volume element traversed times the transit time over that volume element in seconds integrated over the actual path taken by the microorganism. Because each path is different, the UV doses (fluences) received by the various microorganisms are different. This results in a UV dose (fluence) distribution (see Figure 3-8).

Using a mathematical model for the irradiance (fluence rate) distribution in the UV reactor, one can calculate the volume averaged irradiance (fluence rate). Multiplication of this value by the hydraulic residence time in seconds yields the *maximum theoretical UV dose (fluence)* $F_{ideal}(D_{ideal})$ for that reactor under specified conditions of flow rate, UVT and lamp power. An *ideal* UV reactor, with perfect radial mixing, would have a UV dose (fluence) distribution with an extremely narrow UV dose (fluence) distribution, approximately the value $F_{ideal}(D_{ideal})$. However, real UV reactors have UV dose (fluence) distributions that are broader (the breadth is a qualitative measure of the mixing efficiency). Figure 3-8 illustrates two distributions, one of which is much broader than the other. Even though each UV reactor may have the same value of F_{ideal} , the real delivered UV dose (fluence) will be slightly lower than $F_{ideal}(D_{ideal})$ for the UV reactor with the narrower UV dose (fluence) distribution (dashed curve in Figure 3-8) and much lower than $F_{ideal}(D_{ideal})$ for the UV reactor with the broader and more complex UV dose (fluence) distribution (solid curve in Figure 3-8). In the case of the latter distribution, the situation is even worse because of the separate distribution at low UV doses. This will severely limit the ability of this UV reactor to achieve high UV doses.

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It is important to understand that in any real UV reactor, microorganisms flowing through the reactor will receive a variety of UV doses (fluence), some higher and some lower than the mean UV dose (fluence). The microorganisms that receive a low UV dose (fluence) are of particular concern (e.g., the lower peak in Figure 3-8) and can limit the ability of the UV reactor to deliver the required log inactivation.

The method currently used to assess the average UV dose (fluence) is to carry out a biosimetry test (see Chapter 6), which yields the reduction equivalent dose (RED). If the UV dose (fluence) distribution can be obtained (e.g., from computational fluid dynamics models, see Chapter 6), the RED can be estimated by convoluting the UV dose (fluence) distribution with the UV sensitivity kinetics of the test microorganism. Chapter 6 provides a more detailed description of the RED concept and calculations.

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