

# **UVGI for Cooling Coil Disinfection, Air Treatment, and Hospital Infection Control**

Report Prepared by

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for

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January 24, 2011

Revision 1.06

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## **1. Executive Summary**

This report reviews the history and current literature on ultraviolet germicidal irradiation (UVGI) systems used for air and surface disinfection applications. Three of the most promising applications are addressed in this report:

- 1) Cooling Coil Disinfection
- 2) Hospital Infection Control
- 3) Hotel & Residential Air Disinfection

Cooling coil disinfection has proved to be a most economical application and can produce a payback in terms of energy savings of about two years or less. These savings result from reduced coil cleaning maintenance costs and energy savings due to improved heat transfer and reduced pressure losses from airflow through the coils. The use of UVGI on cooling coils tends to restore them to original design conditions and will maintain them in a clean state for as long as the UVGI system is operated. This report summarizes the available information on laboratory and field testing of such installations. Information on the energy savings and payback period of cooling coil irradiation are provided along with an example of the computation of a typical payback period. Draft guidelines on cooling coil irradiation systems from the International Ultraviolet Association are reviewed.

Applications in hospital or other health care facilities include cooling coil cleaning, medical equipment disinfection, whole-room disinfection in unoccupied areas, Overhead surgical site disinfection, and air disinfection in operating rooms, procedure rooms, delivery rooms, isolation rooms, patient wards, and general areas. Hospital-acquired (nosocomial) infections have continued to present a challenge to health care facilities, and cause an undue number of fatal infections annually in the US, with concomitant economic costs. UVGI may be able to reduce many types of nosocomial infections, including surgical site infections (SSIs), and is especially effective against airborne pathogens. This paper explores some of the various applications for air and surface disinfection that may assist hospitals in reducing nosocomial infection rates.

Hotels may obtain significant economic benefits both from the use of UVGI to keep cooling coils clean and from improvements in air quality, which will provide a better environment for both guests and employees. The kinds of problems encountered in large hotels that are amenable to UVGI solutions are addressed in this report, and data is summarized from some previous applications. Residential applications are similar to hotels and these can include homes, apartments, and dormitories.

## **2. Introduction and Background**

The effects of ultraviolet light (UV) on microorganisms were first noted in the late 1800s and the first scientific study of UV is attributed to Downes and Blunt (1877). Baker (1948) indicates that the first mention of the use of ultraviolet light for water disinfection was in 1877. In 1909/1910 the first water disinfection system was operated at Marseilles, France, but it wasn't until 1916 that the first UV system for water disinfection was tested in the US, at Henderson, KY (AWWA 1971).

The first attempts to scientifically quantify the effects of UV irradiation of microorganisms were published in the 1920s. Bedford (1927) and Gates (1929) were among the first to study bacteria and establish UV dosages necessary for disinfection. The earliest study that demonstrated fungicidal action of UV was published by Fulton and Coblenz (1929). The first studies on irradiating viruses appear to have been those published by Sturm et al (1932). In the 1930s a flurry of research and applications demonstrated the feasibility of applying UV light systems in hospitals (Wells and Wells 1936, Wells 1938, Hart and Sanger 1939, Robertson et al 1939, Kraissl et al 1940, Overholt and Betts 1940). The first Upper Air UV systems appear to have been installed around 1938 (Wells 1938).

In the 1940s the first detailed studies of UV air disinfection were published along with basic guidelines for applying UV in ventilation systems (Rentschler and Nagy 1940, Sharp 1940, Wells 1940, Buchbinder and Phelps 1941, DelMundo and McKhann 1941, Henle et al 1942, Luckiesh and Holladay 1942, Sommer and Stokes 1942, Hollaender 1943). The first attempts to apply UV systems to schools and barracks to control respiratory infections occurred shortly thereafter (Wells et al 1942, Wells 1943, Schneiter et al 1944, Wheeler et al 1945, Higgons and Hyde 1947, Perkins et al 1947). Several early attempts were made to develop sizing methods and engineering guidelines for UV applications (Luckiesh and Holladay 1942a, Luckiesh 1945, Luckiesh 1946). Table 1 summarizes many of the seminal developments in UV history.

By 1950 it had been established that UV irradiation was effective at disinfecting both air and surfaces, and engineering applications were being developed. General Electric catalogs detailed many UV applications including various methods of installing UV lamps inside ducts and air conditioners (Buttolph and Haynes 1950, GE 1950). At this time it was not generally known that mold growth on cooling coils could cause respiratory problems. In 1954 it was demonstrated by Harstad et al (1954) that installation of UV lamps in air conditioners would reduce airborne contamination. It was further noted in this published study that microorganisms were impinging upon and collecting on internal AHU surfaces. Although it was understood that UVGI could be used to control microbial growth inside air handling equipment and on cooling coils, little or no attention would be focused on this issue for nearly twenty years.

In 1957, Riley and associates completed a demonstration of how a UV air disinfection could be used to control the spread of tuberculosis (TB) in hospital wards (Riley et al 1957). It wasn't until 1994 that the Centers for Disease Control (CDC) began to acknowledge that UV could be effective for controlling TB, and

this was not in response to the numerous published studies, but because the growing worldwide TB epidemic had resisted control by traditional methods (CDC 2005). Although UVGI systems had been in use in hospitals since 1936, it wasn't until 2003 that the CDC formally acknowledged that UV systems were effective and could be used in hospitals with their conditional blessing – UV Upper Room and in-duct systems could only be used to supplement other air cleaning systems (CDC 2003).

Bacterial growth on cooling coils had been recognized as a potential health problem as early as 1958 (Walter 1969). The first evidence that air cooling equipment could actually cause respiratory infections was presented by Anderson (1959) when an air cooling apparatus was found to be contaminated with microbial growth. This very same concern had been raised in hospital environments since about 1944 but the possibility of growth of bacteria on air-conditioning cooling coils wasn't conclusively demonstrated until 1964 (Cole et al 1964). The growth of microbes on other equipment like filters and dust inside air-conditioning ducts was first demonstrated by Whyte (1968). The fact that microbes growing in air handling equipment could be disseminated by ventilation systems and cause respiratory infections became widely recognized in the late 1960s and early 1970s in both the medical and engineering fields (Banaszak et al 1970, Schicht 1972, Zeterberg 1973). It was widely known at this time that microbial growth could occur anywhere that air came into contact with moisture (Gunderman 1980, Ager and Tickner 1983, Spendlove and Fannin 1983).

The first UVGI system designed specifically for disinfecting the surfaces of air handling equipment, including humidifier water and filters, was detailed by Grun and Pitz (1974). Luciano (1977) published a book detailing many applications of UVGI, including health care applications in which the UV lamps are specifically placed upstream of the cooling coils and downstream of the filters, which has become the primary location for both air disinfection and cooling coil irradiation systems.

In 1985 Phillips published a design guide in which the first definitive description of applications of UV lamps for the control of microbial growth on cooling coils were presented (Phillips 1985). This design guide, "Germicidal Lamps and Applications" provides details of how to locate lamps at specific distances from cooling coils or walls, and referred to installations that were already in operation at the time. Apparently, Europeans had been using such systems prior to 1985 but no publications exist to document such applications.

In January of 1996 the first UVGI system in the U.S. designed for controlling microbial growth on cooling coils was installed by Public Service of Omaha (PSO) in Tulsa. Tom McKain of PSO reports that the idea of irradiating their fouled cooling coils came both from Dr. Richard Shaughnessy of Tulsa University (TU), and from a European professor who suggested the idea at a recent conference (Kowalski 2005). PSO hired Steril-Aire to implement the system, which was found to be highly effective after studies by TU researchers. A patent for cooling coil irradiation was filed later by Steril-Aire. Table 1 summarizes the critical events described above and a number of other major events in the history of UVGI technology development and applications.

**Table 1: Chronology of Critical Events in UV History**

<b>Year</b>	<b>Event</b>	<b>Reference</b>
1870s	Bactericidal Effects of UV light discovered	Downes & Blunt 1877
1877	First demonstration of UV water disinfection	Baker 1948
1903	UV spectrum near 250 nm found to be germicidal	Lorch 1987
1904	First quartz lamp for UV developed	Lorch 1987
1906	UV first used to disinfect drinking water	von Recklinghausen 1914
1909	First European applications for UV water disinfection	AWWA 1971
1916	First USA applications of UV for water disinfection	AWWA 1971
1921	UV photoreactivity with TiO <sub>2</sub> first demonstrated	Renz 1921
1925	UV photodegradation of materials first demonstrated	Luckiesh & Taylor 1925
1927	Bactericidal action of UV first quantified	Bedford 1927, Gates 1929
1929	Fungicidal action of UV first quantified	Fulton & Coblentz 1929
1932	Virucidal action of UV first quantified	Sturm et al 1932
1936	First Overhead UV system in hospitals	Hart 1936, Wells&Wells 1936
1936	UV photoreactivation phenomena first identified	Prat 1936
1937	First Upper Air application in schools	Wells 1938
1938	First fluorescent gas discharge UV lamp	Whitby & Scheible 2004
1940	UV first applied to air conditioning systems	Rentschler & Nagy 1940
1942	First UV air disinfection sizing guidelines	Luckiesh & Holladay 1942
1942	Upper & Lower UV applied to Army/Navy barracks	Wells 1942
1949	UV doses for disinfecting various surface molds quantified	Luckiesh 1949
1950	First catalog sizing methods	Buttolph & Haynes 1950
1954	First air conditioner application	Harstad et al 1954
1954	Faulty British study concludes UV is ineffective	MRC 1954
1957	Riley proves effectiveness of UV for TB control	Riley et al 1957
1974	First microbial growth control systems	Grun & Pitz 1974
1977	Luciano specifies locating UV lamps between filters and cooling coils in air handling units	Luciano 1977
1985	Cooling coil UV systems in use in European breweries	Philips 1985
1994	CDC acknowledges UV effectiveness for TB control	CDC 2005
1996	First cooling coil irradiation system in US	Kowalski 2005
1997	First UV light emitting diodes at 265 nm (LEDs)	Guha & Bojarczuk 1998
1999	WHO recommends UVGI for TB control	WHO 1999
2000	US Army recommends UVGI for disease isolation	USACE 2000
2003	CDC formally sanctions UVGI use in hospitals	CDC 2003
2003	FEMA sanctions UVGI as a biodefense option for buildings	FEMA 2003
2003	In-duct UVGI system demonstrates lower illness symptoms	Menzies et al 2003
2003	ASHRAE forms Air Treatment Group	Martin et al 2008
2005	GSA specifies UV for cooling coil disinfection in buildings	GSA 2005
2007	Long-term hospital study demonstrates significant reductions in surgical site infections w/ overhead UV	Ritter et al 2007
2009	US Army addresses UVGI for medical facilities	USACE 2009
2009	ASHRAE issues Position Statement on airborne disease	ASHRAE 2009

The topic of UV air disinfection is currently being addressed in detail by the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) who have released a position paper on the subject of airborne infectious diseases as they relate to ventilation systems (ASHRAE 2009). ASHRAE's position as stated in the paper is as follows:

- Many infectious diseases are transmitted through inhalation of airborne infectious particles termed droplet nuclei
- Airborne infectious particles can be disseminated through buildings including ventilation systems
- Airborne infectious disease transmission can be reduced using dilution ventilation, specific in-room flow regimes, room pressure differentials, personalized and source capture ventilation, filtration, and UVGI.

ASHRAE further explains that some diseases are transmitted through the airborne route when the mean aerodynamic diameter of a droplet or particle is less than 20 microns. These particles may be generated by coughing or sneezing and to a lesser extent by talking. These particles may remain airborne for hours and be transported over great distances, and their distribution may be impacted by HVAC system operation. Increasing the amount of airflow to an indoor environment can dilute the concentrations of infectious particles and so lower the infection risk. Filtration and ultraviolet germicidal irradiation can also be used for engineering control. Three UVGI strategies are discussed by ASHRAE, in-duct air disinfection, upper room UVGI, and whole-room disinfection. Further research is needed, ASHRAE notes, to determine the efficacy of these air disinfection technologies and how these controls can be applied in buildings to reduce the risk of airborne disease transmission.

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### 3. Microbial Disinfection Model

A microbial population subject to UV exposure will tend to decay exponentially over time. The survival fraction at any time  $t$  after exposure can be defined by the following single stage exponential decay equation:

$$S = e^{-kt} \quad (1)$$

where  $k$  = UV rate constant,  $\text{cm}^2/\mu\text{J}$

Figure 1 illustrates the exponential decay curve on a logarithmic scale with various values of  $k$ . The slope of the logarithmic decay curve (the slope of the line in Figure 1) is called the rate constant. The rate constant will determine how fast the population decreases under exposure. The value of the rate constant depends on both the species and the UV irradiance.

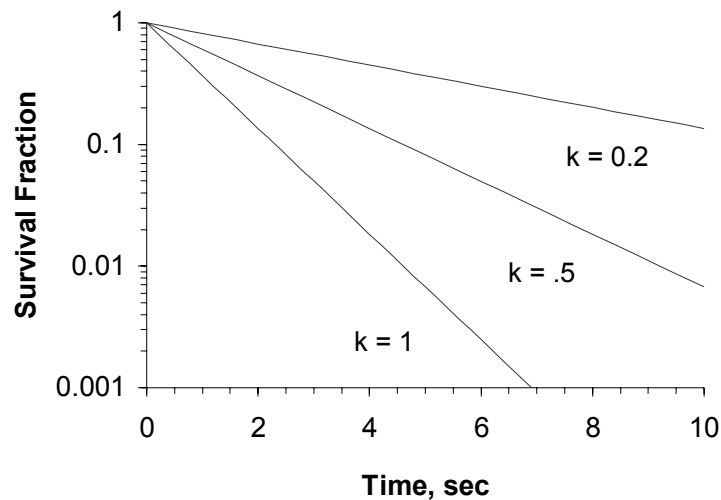


Figure 1: Survival curves for various rate constants.

The rate constant determines how fast the microbial population decays under the influence of UV. The UV irradiance may vary in intensity. The variation of irradiance is accounted for by a multiplier designated  $I$ . The classic exponential decay equation is then written as:

$$S = e^{-kIt} \quad (2)$$

In the form shown in equation (2), the rate constant  $k$  is known as the standard rate constant and it represents the susceptibility of the species for unit intensity only. In general,  $k$  is unique to each species. Often the quantity ' $It$ ' is combined into a single term called the dose. The dose can therefore be defined as:

$$D = It \quad (3)$$



When the dose is defined as in equation (3), the exponential decay equation is simply written as:

$$S = e^{-kD} \quad (4)$$

Sometimes a microbial population under UV exposure behaves as if it is two separate populations – one that succumbs rapidly and another that resists the factor. This effect has often been referred to as tailing or as nonlogarithmic survivor curves (Fujikawa and Itoh 1996, Moats et al 1971). Under these conditions the result is a two-stage decay curve. The two-stage curve is treated mathematically as if it were two distinct and separate populations that are simply added together. Each population has a unique rate constant, denoted by  $k_1$  and  $k_2$ . The fraction of the population that is resistant is denoted by  $f$ , while the complementary fraction is denoted by  $(1-f)$ , as follows:

$$S = (1-f)e^{-k_1D} + fe^{-k_2D} \quad (5)$$

Figure 2 shows a survival curve fitted to equation (7.12) based on UVGI data for *Streptococcus pyogenes*. The curve was fitted by splitting equation (5) into two halves and fitting them individually to the split data set. The intercept of the second stage provided the population fraction.

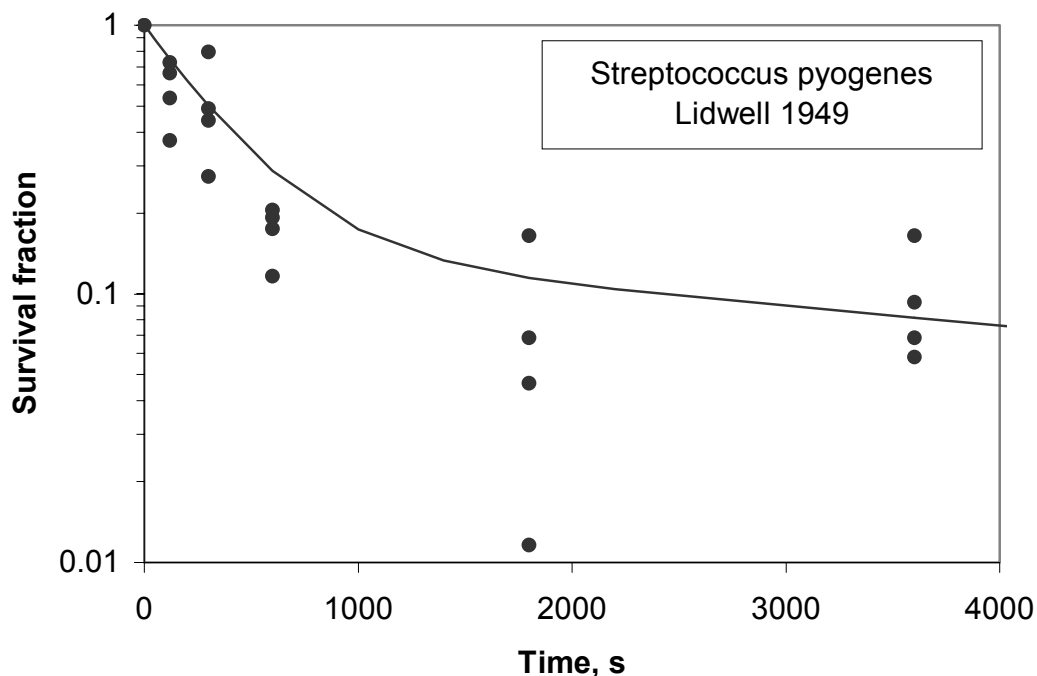


Figure 2: Survival of *Streptococcus pyogenes* under UVGI exposure. Two stage curve fitted to data from Lidwell (1949).

Data on two stage decay curves is limited and most of the available data for UVGI disinfection is for single stage curves only. The single stage rate constant is appropriate for use provided there is either no second stage or when the UV dose being applied does not result in the survival curve extending into the second stage region. This is certainly the case for most air and water disinfection systems. It may not be the case for many surface disinfection systems, but surface disinfection often involves extended exposure times (i.e. 1-24 hours or more) in which the applied dose often produces complete sterilization, as in medical equipment disinfection and cooling coil disinfection systems.

When a UV rate constant is used for design purposes, it should be applied only within the limits of the original test data. That is, the rate constant should not be extrapolated beyond the original test conditions. In general, a rate constant can be safely defined within the limits of a dose described by a  $D_{90}$  value. A  $D_{90}$  is the dose that produces 90% disinfection (or 10% survival). The  $D_{90}$  is defined as follows:

$$D_{90} = \frac{-\ln(1-0.90)}{k} = \frac{-\ln(0.1)}{k} \quad (6)$$

Related terms that are sometimes used include the  $D_{99}$  (99% inactivation or 1% survival),  $D_{99.9}$  (99.9% inactivation or 0.1% survival), and  $D_{99.99}$  (99.99% inactivation or 0.01% survival). A term that is less commonly used in the literature is the  $D_{37}$  value, which is often a misnomer since it is sometimes defined as the dose that produces 37% inactivation and sometimes defined as the dose that produces 37% survival. All D values other than  $D_{90}$  should be used with caution since the second stage may become manifest near the 1% survival level. In many cases  $D_{99.9}$  and  $D_{99.99}$  values are extrapolations from the first stage and may not be valid. The source document from which the UV rate constant is culled should be consulted to ensure that the UV dose or survival range is supported by the data. A summary of microbial rate constants is provided in Appendix A along with an associated  $D_{90}$  value, which should be considered an upper limit below which the rate constant is valid. Most of these rate constants will be valid up to a  $D_{99}$  value, but beyond that caution should be exercised.

Shoulders may also exist in the survival curve, but for viruses and bacteria these shoulders are often negligible. For fungal and bacterial spores, however, the shoulders may distort any predictions made with the rate constant. Even when shoulders are present, the  $D_{90}$  remains a reliable and absolute indicator of the UV susceptibility.

#### 4. Cooling Coil Disinfection Model

In typical cooling coil disinfection systems, a UV lamp, or array of UV lamps, is positioned so as to irradiate a coil surface. In the example shown in Figure 3, UV lamps are positioned so as to irradiate both the upstream and downstream sides of a cooling coil. Often, it is not possible to position lamps on both sides of a coil like this and only one side is irradiated. Lamps are often positioned in a crossflow arrangement in which the axis of the lamp runs perpendicular to the fins of the coil. The orientation of the lamp is not necessarily critical and lamps may be positioned horizontally, vertically, or at any angle relative to the coil surface. Lamp position will impact the irradiance levels at the coil surface but adjusting the total wattage, number of lamps, reflectivity, and other factors can compensate for less than optimum positioning of the lamp.

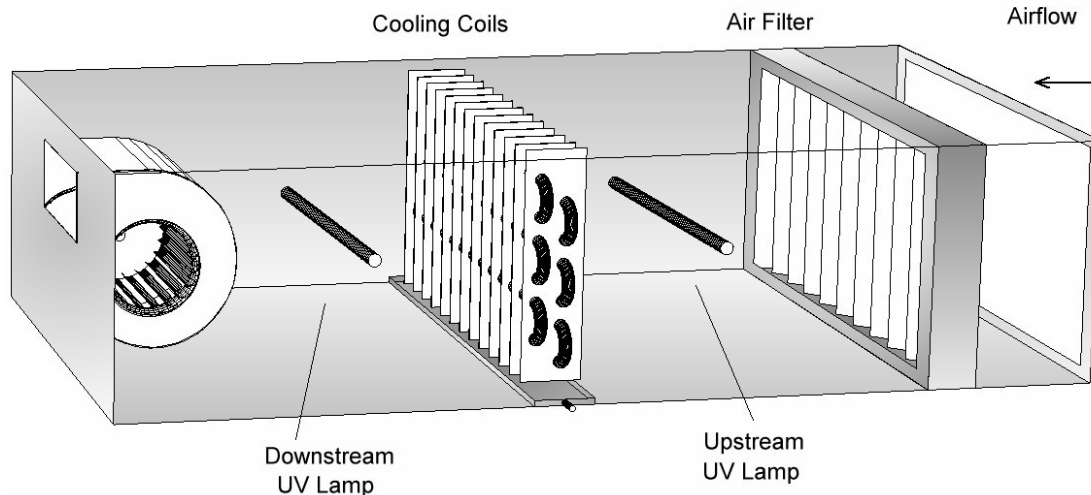


Figure 3: Air handling unit with UV lamps irradiating both upstream and downstream sides of the cooling coil.

When a single lamp is positioned with its axis parallel to the coil surface, the irradiance at any point on the coil surface can be determined using the view factor model of the lamp as a cylinder, as detailed by Kowalski et al (2000). Computer algorithms for this view factor model have been provided by Kowalski (2001 & 2003). The view factor model has been demonstrated to provide fairly accurate agreement with actual lamp irradiance measurements. Alternate lamp models have been proposed by others but there is either limited agreement with lamp data or a lack of quantitative data on the models (IESNA 2000, Krasnochub 2005). The view factor model can be used to generate irradiance profiles and contours such as those shown in Figure 4 and Figure 5, in which a single cylindrical lamp irradiates a rectangular cooling coil surface. The peak irradiance can be seen as a blunt outline of the cylindrical lamp.

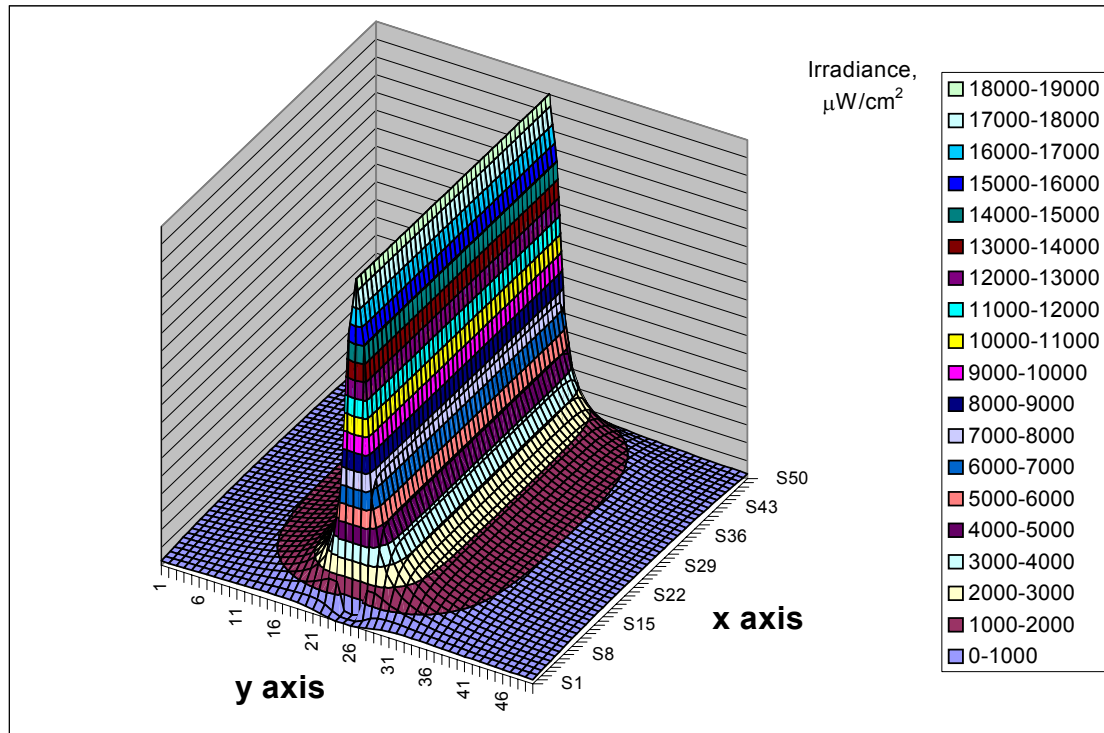


Figure 4: Example of irradiance profile on a cooling coil surface (x-y axes) from a single UV lamp located a short distance away.

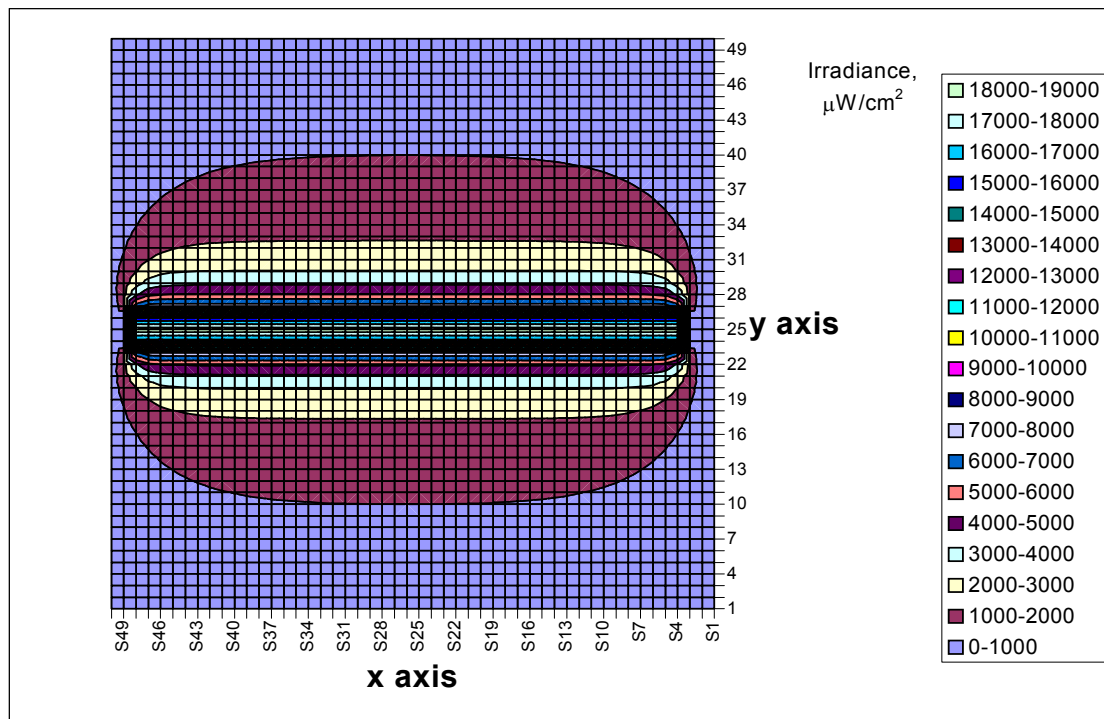


Figure 5: Irradiance contour on the cooling coil face (x-y axes) from the example in Figure 4.

Placing a UV lamp in front of a cooling coil entrance or exit plane will produce an irradiance contour on the leading edges or exit edges of the coil fins similar to that of Figure 5. Only the front surface (or back surface) irradiance levels can be predicted with certainty because determination of the irradiance within the cooling coil fins is an exceedingly complex problem that involves a limited field view factor and the reflective characteristics of the fins and coil tubes. At present, predictions of the surface irradiance must suffice as an indicator of the adequacy of UV exposure levels, but photometer measurements can also be used to confirm irradiance levels upstream and downstream. The ultimate confirmation of the adequacy of UV irradiance levels can only be obtained via surface sampling for spores. An alternative indicator of the effectiveness of UVGI may be coil performance, since the elimination of surface contamination should theoretically restore cooling coil performance to original design values.

Under UV exposure, the disinfection of cooling coil surfaces follows the basic mathematical decay models detailed in the previous section. Because the exposure times are extended in these types of surface disinfection systems, it is appropriate to use the two-stage decay equation to define the disinfection rates. The reason is that if a second stage does exist (i.e. for any mold or bacterial spore) it will likely become the only remaining stage after relatively brief initial exposure period. That is, the first stage will show rapid decay, after which only the second stage remains. Since the second stage becomes dominant in the long run, it is a better predictor than the single stage rate constant. However, few second stage rate constants are known with any certainty and predictive methods generally rely on theoretical values.

Figure 6 shows an example of a two stage decay curve for *Aspergillus niger* spores compared with predictions from a single stage model. The single stage model (in red) shows a log-linear decrease in microbial population over time, while the two-stage model (in blue) shows a second stage (a tail) becoming dominant after about 1000 seconds. It is clear that after extended exposure the single stage model will grossly overpredict the survival rate of the spores. This two stage behavior is typical for most spores under prolonged exposure and indicates the need to use a two stage model when evaluating cooling coil surface disinfection. Data for the single stage is based on IESNA (2000) while the two stage curve is based on laboratory data from UVDI (2000).

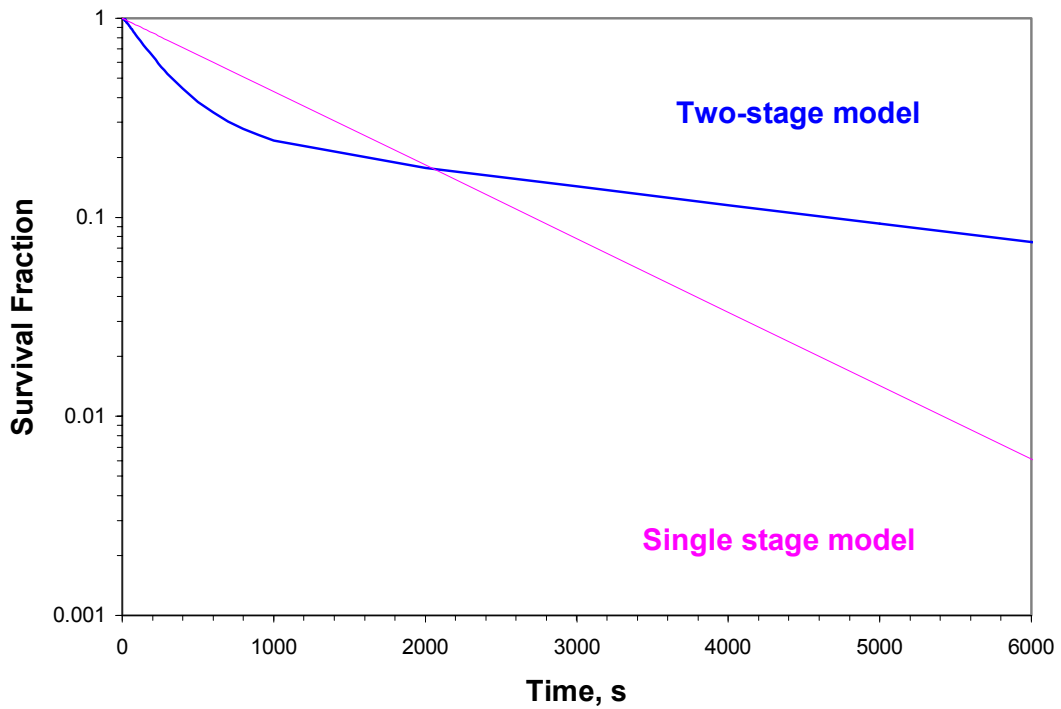


Figure 6: Comparison of a single stage model vs. a two stage model of the inactivation of *Aspergillus* spores under UV exposure of  $50 \mu\text{W}/\text{cm}^2$ . The single stage model will underestimate the required dose for sterilization.

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### **5. Performance of Cooling Coil Disinfection Systems**

Although studies on the inactivation of mold spores and inhibition of mold growth by UV abound in the literature, information on the actual disinfection of cooling coils remains limited and reports of successful disinfection are primarily anecdotal, although some formal studies are underway (EPRI 2004, Shaughnessy et al 1999). There is, however, no reason to believe that the anecdotal reports are not accurate, and the indications are that disinfection of cooling coils with UV is so effective that payback periods of about 2-4 years are possible. That is, the cleaning of the coils under UV exposure proceeds so rapidly that fouled coils are restored to pristine condition and save energy and maintenance costs so effectively that the retrofit of a UV coil cleaning system pays for itself in about 2-4 years.

Theoretically, continuous exposure of cooling coil surfaces to UV should result in eradication of virtually all surface contamination within a few hours or days, depending on the irradiance levels. That is, any contamination on the exposed surface of the coils (entrance or exit respectively) should be sterilized rapidly. Figure 7 shows a system for which surface samples taken by the author indicated virtual sterilization of the leading edges after two weeks of operation.



Figure 7: A UVGI system installed in front (upstream) of a cooling coil that sterilized the front face of the coil after two weeks of operation.

Contamination on the internal surfaces of the cooling coil fins should also be sterilized over time, but it is difficult to predict how much time this might require. It does appear that, based on anecdotal field reports, that a few weeks or months is all that is required to restore coils to original design operating conditions, suggesting that internal coil contamination is sterilized in these time periods.

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## **6. Economics of Cooling Coil Disinfection Systems**

The economic savings that can result from the installation of a UV cooling coil disinfection system can be estimated by comparing the operating costs after installation with the operating costs before installation, minus the cost of installing and operating the UV system. Ideally, operating data would be drawn from field test results, but this necessitates installing such a system first. Little published data is available for installed systems but anecdotal evidence suggests that UV disinfection systems are fully capable of restoring a fouled cooling coil to approximately the original design operating conditions. The cost savings will then depend on how much coil fouling has occurred and how far the system capacity has been diminished in comparison with the original design conditions. Table 2 shows the basic costs and the basic savings of UV cooling coil disinfection systems.

**Table 2: Costs vs. Savings of Cooling Coil Disinfection**

Costs	Savings
First Cost of installation	Fan energy savings
Operating Cost of UVGI	Cooling energy savings
Maintenance costs of UVGI	Maintenance savings

The first cost of the UVGI system will always be known, as will the operating and maintenance costs, which consist of electrical energy consumption and lamp replacement. The heat added to the system by the lamps is generally negligible and can be ignored, and furthermore, in cold climates the heat becomes a credit but this will also be ignored. The energy savings will result from two effects, the first being the reduced pressure drop through the coils once the fouling is removed, and the second being the increased rate of heat transfer from the coils when the fouling film is gone. Both of these can be significant, as can the reduction or elimination of maintenance on the cooling coils. Since the coils will be maintained in a clean condition, there is likely to be no requirement for periodic cleaning of the coils. In fact, since the UV system will maintain the coils in pristine condition, the lifetime of the coil will likely be extended well beyond the normal lifespan of unirradiated cooling coils, but this aspect of the savings will be difficult to quantify until field data is accumulated from installations.

In order to estimate cost savings, it is necessary to assume that 1) the cooling coil is fouled, which is usually true if a system is being considered, and 2) the fouling will be completely eliminated and the coils restored to design condition, which is reportedly the usual case. Alternatively, a UV system may be installed on a brand new cooling coil, in which case the savings would have to be estimated based on the projected rate of fouling.

The cost savings in dollars of a UV cooling coil disinfection system can be written as:

$$Savings = (FE_F - FE_C) + (CE_F - CE_C) + (M_F - M_C) - FC_{uv} - OC_{uv} - MC_{uv} \quad (1)$$

where  $FE_F$  = Fan Energy cost, Fouled (\$)
   
 $FE_C$  = Fan Energy cost, Clean (\$)
   
 $CE_F$  = Cooling Energy cost, Fouled (\$)
   
 $CE_C$  = Cooling Energy cost, Clean (\$)
   
 $M_F$  = Maintenance cost, Fouled (\$)
   
 $M_C$  = Maintenance cost, Clean (\$)
   
 $FC_{uv}$  = First Cost of UV (\$)
   
 $OC_{uv}$  = Operating Cost of UV (\$)
   
 $MC_{uv}$  = Maintenance Cost of UV (\$)

The fan energy in kW is computed as follows:

$$FE = \frac{dP \cdot CFM}{6350(0.75 \cdot 0.75)} \cdot 0.7355 \quad (2)$$

where  $dP$  = pressure drop, in.w.g.
   
 $CFM$  = airflow, cfm
   
0.7355 = conversion factor from BHP to kW
   
0.75 = typical motor efficiency
   
0.75 = typical fan efficiency
   
6350 = conversion factor (in.w.g-cfm) to BHP

The fan energy savings is then the fan energy in the operating condition (fouled coils) minus the fan energy under design conditions.

The cooling energy savings in kW is computed as follows:

$$CE = \left( \frac{CL}{3412 \cdot COP} \right) \quad (3)$$

where  $CL$  = the capacity loss due to fouling, Btuh
   
 $COP$  = Coefficient of Performance
   
3412 = conversion from Btuh to kW

The  $COP$  can be computed as the seasonal energy efficiency ratio (SEER) divided by 3.412. The typical value for the SEER is about 9 or 10, and the respective  $COP$  would be about 2.64 – 2.93.

The maintenance cost Before,  $M_B$ , can vary and depends on local facility procedures. Cooling coil maintenance is typically a few hours of labor a year, and may vary from a few hundred to a few thousand dollars. A reasonable estimate for small cooling coil units might be about  $M_B = \$500$  a year. Presumably, there will be no maintenance cost After, or  $M_A = 0$ .

The first cost of the UV system,  $FC_{uv}$ , will be established at the beginning of any project and no estimates can be provided. The operating cost,  $OC_{uv}$ , of the UV system is simply the electrical energy consumed by the lamp and ballast. The energy cost can be written as follows:

$$OC = \frac{W \cdot 8760}{1000} P_c \quad (4)$$

where  $W$  = total watts of power consumed by lamp fixture  
 $P_c$  = power charge (typically 0.08 – 0.1 \$/kWh)  
 8760 = hours of operation per year (continuous assumed)  
 1000 = conversion from kW to W

The maintenance cost of the UV system consists of the annual replacement of the UV lamps, which is simply the cost per lamp times the number of lamps. This cannot be estimated in advance and will depend on the particular project.

Application of the above equations can be demonstrated through an example of a typical cooling coil disinfection system. Consider a system with the following parameters:

- Airflow, cfm - 48,500 CFM
- Cooling Coil leaving air temperature: 52 degrees F.
- Cooling Coil pressure drop, 0.75 in.w.g.
- UV wattage – 552W UVC output.
- UV lamp fixture first cost - \$3,528 per total number of fixtures per coil.
- UV lamp installation labor cost, \$1000.
- UV lamp replacement bulb cost - \$1,800 annually.
- Annual hours of cooling – approximately 4,500 hours per year.
- Cost per kWh, \$0.09.
- COP = 4.1 (typical for chilled water system)
- Cooling Load (design), 1,500,000 Btuh (assumed)

Keikavousi (2004) reports that a 27 year old system retrofitted with UV had a reduction in fan static pressure from 1.8 iw.g to 0.7 iw.g. The fan energy in our example above assumes only a fouled condition of 0.9 iw.g, reducing to 0.75 after UV installation. The fan energy under design conditions is:

$$FE = \frac{0.75 \cdot 48,500}{6350(0.75 \cdot 0.75)} \cdot 0.7355 = 7.49 \text{ kW}$$

The fan energy under fouled conditions (assumed 0.9 in.w.g.) is:

$$FE = \frac{0.9 \cdot 48,500}{6350(0.75 \cdot 0.75)} \cdot 0.7355 = 8.988 \text{ kW}$$

The fan energy savings (Fouled-Clean) is:

$$FE_{F-C} = (8.988 - 7.49)4500 \cdot 0.09 = 607 \$$$

The cooling energy (design operating conditions) is:

$$CE = \left( \frac{1,500,000}{3412 \cdot 4.1} \right) = 107.2 \text{ kW}$$

Assuming a 20% loss due to fouling, the energy savings would be:

$$CE_{F-C} = (0.20 \cdot 107.2) \cdot 4500 \cdot 0.09 = 8685 \$$$

The operating costs are:

$$OC = \frac{552 \cdot 8760}{1000} 0.09 = 435 \$$$

The total savings can then be summed up as follows, assuming \$1000 in maintenance savings:

$$Savings = (607) + (8685) + (1000) - 3528 - 435 - 1800 = 4529 \$$$

The payback (PB) period can be approximated by dividing the initial cost by the annual savings as follows:

$$PB = \frac{3528}{4529} = 0.8 \text{ years}$$

Some examples of estimates of the savings that might be accrued from the use of cooling coil disinfection systems in health care facilities are provided in Appendix D and Appendix E. In Appendix D summaries for six facilities are provided showing inpatient and outpatient occupancies, number of clinical procedure rooms and number of procedures performed. Appendix E provides estimated costs for cooling coil disinfection systems, in-duct UV systems, and operating room UV systems, along with estimated savings based on assumptions regarding nosocomial infection rates and operating costs. Although the available data on nosocomial infection rates due to airborne infections is not specific enough to isolate the true savings that might be anticipated, the ball-park figures provided in Appendix E clearly show the potential savings are great and that payback periods computed from these estimates would be in the range of 1-2 years or less, similar to the payback periods demonstrated previously.

## **7. Guidelines for Cooling Coil Disinfection Systems**

Several guidelines have been recently introduced, or are in preparation that address the use of UV for either cooling coil disinfection or air disinfection (GSA 2003, NIOSH 2005, IUVA 2005). Based on the literature, including draft guidelines from IUVA (2005), and the analysis previously presented, certain basic design guidelines can be summarized. These are as follows:

- **Guidelines for Cooling Coil Disinfection**
- Minimum Filtration: MERV 6
- Recommended Filtration: MERV 8-11
- Maximum air velocity of between 400-500 fpm
- Maximum air temperature between 40°F-110°F
- Maximum ballast operating temperature of 40°C or 50°C (104°F or 122°F) depending on ballast
- Lamp placement: upstream, downstream, or both sides of coils
- Lamp distance from coil face: 1-4 feet (30-120 cm)
- **Exposed Coil Surface:**
  - Recommended coil average irradiance: 50-500  $\mu\text{W}/\text{cm}^2$
  - Minimum coil irradiance: 50  $\mu\text{W}/\text{cm}^2$
  - Minimum coil irradiance in any corner or side: 10  $\mu\text{W}/\text{cm}^2$
- **Opposite Coil Surface (if unexposed)**
  - Recommended coil average irradiance: 50-100  $\mu\text{W}/\text{cm}^2$
  - Minimum coil average irradiance: 10  $\mu\text{W}/\text{cm}^2$
  - Minimum coil irradiance in any corner or side: 1  $\mu\text{W}/\text{cm}^2$

The above recommendations are preliminary (per IUVA 2005) and should not be considered to be strict requirements as these matters are still under study. In addition to the above guidelines, it is recommended that UV lamp ballasts be placed externally if possible, or, if placed internally, be shielded from any heat sources. All electrical wiring should be in accordance with UL/ETL requirements. Alarms or disconnect switches should be included to disengage the UV lamps if an access door is opened. Warning signs should be placed in the vicinity and proper training given to maintenance personnel. UV lamps should be handled with care and used lamps disposed of in accordance with regulations regarding mercury content.

As verification of coil disinfection, surface sampling for fungi and/or bacteria could be performed before UV lamp installation, and then follow-up testing could be performed about 2 weeks or any time later. Major reductions in coil contaminants would suggest effective disinfection while the absence of all fungal contamination would indicate complete sterilization. As an alternative to microbiological testing, coil performance could be monitored over time to verify that the cooling coil heat transfer and pressure drop characteristics are being returned to design conditions, a process that requires an unknown amount of time, but for highly fouled coils it may require weeks or months.

## **8. Hospital Air & Surface Disinfection Systems**

There are four primary applications for UVGI in hospitals – 1) Overhead UV systems for control of surgical site infections (SSIs), 2) UV Air Disinfection systems for control of airborne nosocomial (hospital-acquired) infections (i.e. TB isolation rooms), 3) Medical Equipment Disinfection, and 4) Cooling Coil Disinfection. The latter subject has been addressed in the previous section and is not re-addressed here. Equipment disinfection systems such as UV cabinets are in wide use in health care settings but this subject is not addressed here in favor of the first two applications, which are topical subjects of much current interest.

Although UVGI systems have been in use in hospitals since the very beginnings of UV technology, they have never been formally recognized and accepted by governing agencies until very recently. There continues to be a lack of formal acknowledgement on the part of the responsible health authorities and a shortage of guidelines, information, and training for health professionals in the application of UVGI to hospital infection control. Some recent guidelines have acknowledged the potential effectiveness of UVGI for the control of nosocomial infections (CDC 2003, ASHRAE 2003). The most dramatic success of UVGI in the health care industry has been in the area of surgical site infection control, although cooling coil systems are poised to make major inroads because of their favorable economics.

The use of UVGI to clean cooling coils in hospital ventilation systems has the same economic benefits as those addressed in the previous sections of this report. However, the removal of mold and bacterial slime from cooling coils is much more important in the hospital environment which should be kept cleaner than the average commercial office building. That is, environmental bacteria and mold spores may pose hazards to patients in hospitals, especially those with impaired immune systems or burn victims. Microbial levels on surfaces and in the air need to be lower in hospitals due to the higher risks of nosocomial infection. The disinfection of cooling coils is one way to reduce microbial loading in hospitals. In theory, the requisite levels of air filtration should keep microbial contamination from accumulating on cooling coils, which are typically downstream of the filters, but casual inspections of hospital cooling coils and ductwork typically show levels of biocontamination above and beyond what would be expected with such high efficiency filters installed. The reasons for the apparent penetration of the filters are unclear but may be due to poorly fitting filters, leaking filters “bypass factor”, or the performance of filter change-outs while the ventilation system continues to operate. The installation of a UVGI cooling coil disinfection system should resolve such problems and contribute to net reductions of biocontamination in hospitals. The use of dual purpose UVGI systems that both clean coils and disinfect air, should also enhance the reductions of microbial loading.

## 8.1 Hospital-Acquired (Nosocomial) Infections

Nosocomial infections include many diverse diseases, the sources and etiology of which are uncertain at present. Table 3 summarizes nosocomial agents that have the potential to transmit by the airborne route. The majority of nosocomial agents are potentially airborne, although most of the actual transmission is probably through direct contact (Kowalski 2005). The degree to which a cooling coil disinfection system will decrease nosocomial infections is probably quite limited, although the cost savings alone should justify such systems in any health care facility. The degree to which air and surface disinfection systems can reduce nosocomial infections is much more quantifiable, and a number of studies have been performed in this regard.

There are some aspects of the savings that could be expected in health care facilities that cannot be generalized or quantified exactly, such as the reduction in worker illness (nosocomial worker illnesses are an ongoing problem in health care facilities for which rates and costs are unknown at present), and possible reductions in insurance costs once such air cleaning systems are installed. The estimated costs in these examples, including the labor costs and energy costs, are based on assumptions and should not be construed to represent the actual costs for any specific installation, which need to be independently determined. The geographic area can also impact the energy costs through climate. One related report, that addresses air cleaning and bioterrorism, is a guideline from FEMA on the subject of insurance costs that may also be used to address reductions of naturally occurring diseases and possible savings from UVGI (FEMA 2003). UVGI may also allow for the use of filters with lower pressure drops (ACEEE 2005).

**Table 3: Nosocomial Agents with Airborne Transmission Potential**

AIRBORNE PATHOGEN	TYPE	PRIMARY INFECTION CAUSED	Annual Cases	Annual Fatalities
Varicella-zoster virus	C	chickenpox	common	250
Streptococcus pyogenes	C	scarlet fever, pharyngitis	213,962	-
Streptococcus pneumoniae	C	lobar pneumonia, sinusitis, meningitis	500,000	50000
Staphylococcus aureus	E	staphylococcal pneumonia, opportunistic	2,750	-
Serratia marcescens	E	bacteremia, endocarditis, pneumonia.	479	-
SARS virus	C	Severe Acute Respiratory Syndrome	(10)	(?)
Rubella virus	C	rubella (German measles)	3,000	none
Rhizopus stolonifer	NC	zygomycosis, allergic reactions	rare	-
Respiratory Syncytial Virus	C	pneumonia, bronchiolitis	common	rare
Pseudomonas aeruginosa	NC	pneumonia	2,626	-
Pneumocystis carinii	NC	pneumocystosis	rare	rare
Parainfluenza virus	C	flu, colds, croup, pneumonia	common	-
Nocardia brasiliensis	NC	nocardiosis	uncommon	-
Nocardia asteroides	NC	nocardiosis	uncommon	rare
Mycobacterium tuberculosis	C	tuberculosis, TB	20,000	-
Mucor plumbeus	NC	mucormycosis, rhinitis	rare	rare
Moraxella	E	otitis media, opportunistic	rare	0
Measles virus	C	measles (rubeola)	500,000	rare
Legionella pneumophila	NC	Legionnaire's Disease, opportunistic	1,163	10
Klebsiella pneumoniae	E	opportunistic, pneumonia	1,488	-
Influenza A virus	C	flu, secondary pneumonia	2,000,000	20000
Histoplasma capsulatum	NC	histoplasmosis, fever, malaise	common	-
Haemophilus parainfluenzae	E	conjunctivitis, pneumonia, meningitis	common	-
Haemophilus influenzae	C	meningitis, pneumonia, endocarditis	1,162	-
Cryptococcus neoformans	NC	cryptococcosis, cryptococcal meningitis	high	rare
Corynebacterium diphtheriae	C	diphtheria, toxin produced.	10	-
Coccidioides immitis	NC	coccidioidomycosis, valley fever	uncommon	-
Chlamydia pneumoniae	C	pneumonia, bronchitis, pharyngitis	uncommon	-
Cardiobacterium	E	opportunistic infections, endocarditis	rare	-
Burkholderia pseudomallei	NC	meliodosis, opportunistic	rare	rare
Burkholderia mallei	NC	Glanders, fever, opportunistic	-	none
Bordetella pertussis	C	whooping cough	6,564	15
Blastomyces dermatitidis	NC	blastomycosis, Gilchrist's Disease	rare	-
Aspergillus	NC	aspergillosis, alveolitis, asthma	uncommon	-
Alcaligenes	E	opportunistic	rare	rare
Acinetobacter	E	opportunistic/septic, meningitis	147	-

Abbreviations: C = Communicable, NC = Noncommunicable, E=Endogenous.



## 8.2 Surgical Site Infections (SSIs) and Overhead UV Systems

The aerobiology of operating rooms (ORs) is primarily a function the microbial flora of the occupants, with common skin microbes like *Staphylococcus* and *Streptococcus*, and some intestinal flora like *Escherichia coli* and *Enterobacter*, being major contributors. Environmental contaminants like *Pseudomonas aeruginosa* and *Bacillus subtilis* also show up in ORs. Direct contact is generally regarded as being the primary mode of contamination of surgical sites, but airborne transmission may be a major factor. Airborne transport can result in contamination of equipment and surfaces in the OR, which may result in direct contact infections. Figure 8 shows the major microbial causes of SSIs ranked by their frequency of occurrence and proportioned by their relative size.

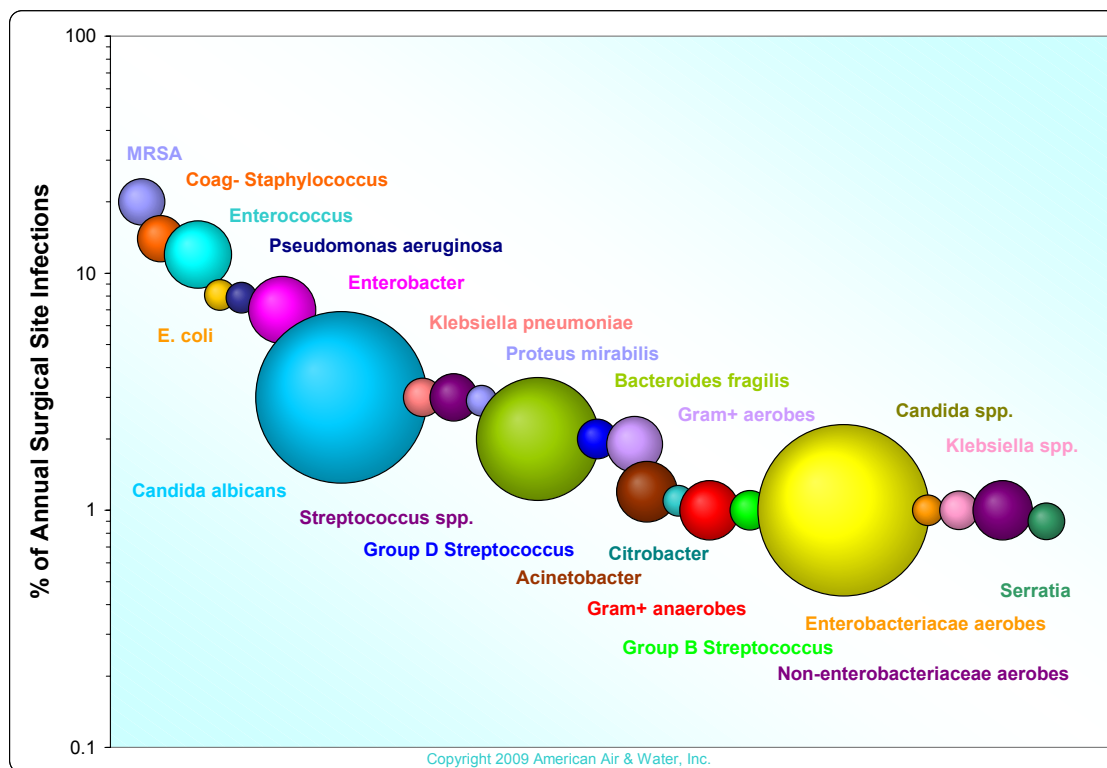


Figure 8: Major SSI infectious agents ranked by occurrence and proportioned by relative size.

In one study of airborne microbial contamination by Holcatova et al (1993) in the OR and ICUs of a surgery clinic, airborne bacterial concentrations were measured to be 150-250 cfu/m<sup>3</sup>. The most frequently isolated microbes included *Staphylococcus epidermis*, *S. haemolyticus*, *Pseudomonas* spp., *Enterococcus* spp., *Enterobacter*, *Micrococcus*, *Corynebacteria*, and *Streptococcus faecalis*. The airborne microorganisms most frequently found in the OR include *Staphylococcus epidermis* and *S. aureus* (MRSA) (Nelson et al 1973). *Streptococcus pyogenes* has been found in some 15% of preoperative throat swabs from patients (Dubuc et al 1973). The total number of personnel in an OR

directly influences the total concentration of airborne bacteria (Hart 1938, Hambreaus et al 1977, Mangram et al 1999, Duvlis and Drescher 1980). OR personnel may shed from 3,000-50,000 microorganisms per minute depending on activity and the effectiveness of protective clothing. Conversation among personnel can increase the bacterial load of the air, and contaminated face masks (measured postoperatively) occur in about 9-10% of surgeons and nurses (Ritter 1984). Aerosolized blood pathogens can present hazards to surgeons that face masks may not protect them against. Aerosols in the respirable size range (less than 5 microns) containing blood can be generated in an operating room during surgery with common surgical power tools (Kowalski 2006).

Previous studies have shown UV installations in operating rooms to be effective in reducing surgical site infections. The first UVGI systems developed and installed were overhead UV systems for ORs and were intended to reduce the rate of surgical site infections (SSIs). SSIs cause undue costs through repeat procedures and unnecessary fatalities. From the first applications, these systems proved highly effective. Overhead UV systems have been in continuous use in some operating rooms since at least 1937 and some of these are still operating today (Hart and Sanger 1939). Reductions in post-operative infection rates of about 24-44% have been reported (Goldner and Allen 1973). Duke University reduced the SSI rate by over 80% and has continued to use overhead UVGI systems to maintain a low level of orthopedic infections (Lowell et al 1980). Table 4 summarizes the SSI reductions that have been demonstrated by previous Overhead UV installations.

**Table 3: Results of Hospital Field Trials of Overhead UV Systems**

Type	System	Location	Infection	Infection Cases		Decrease		Reference
				Before	After	Net	%	
Surgical Site Infections	Overhead Surgical Site UVGI	Duke University Hospital	SSI	5%	1%	4%	80%	Kraissl et al 1940
		Duke University Hospital	Hip arthroplasty infection	5%	0.5%	5%	90%	Lowell et al 1980
		NE Deaconess Hospital	SSI	15%	6.53%	8.5%	56%	Overholt and Betts 1940
		Infant & Children's Hospital, Boston	SSI	12.5%	2.7%	9.8%	78%	Del Mundo & McKhann 1941
		Watson Clinic, FL	Mediastinitis	1.4%	0.23%	1.2%	84%	Brown et al 1996
		St. Francis Hospital	SSI	1.77%	0.57%	1.2%	68%	Ritter et al 2007
		<b>AVERAGE REDUCTION</b>						<b>76%</b>

### 8.3 UVGI Air Disinfection for Controlling Nosocomial Infections

Airborne microbes exist in all indoor environments, but hospitals require air that is cleaner than found in normal indoor environments because of the potential for transmission of respiratory and other infections, and because of the presence of patients who may be more vulnerable to infections. In spite of this, hospitals generally do not attempt to control or monitor their airborne microbial levels beyond a basic adherence to existing guidelines for ventilation systems. Standards set by ASHRAE (1999 & 2003) and AIA (2001) for ventilation rates and filter use are assumed to provide clean, disinfected air to all areas of the hospital, but this is not the case. Indoor airborne levels in hospitals have been measured and although they tend to be lower than those in commercial office buildings and residential environments, they tend to be higher than what would be expected for health care facilities.

The aerobiology of open areas in hospital facilities does not differ greatly different from levels we might find in ordinary occupied buildings. In a study of mold spores in the air of a hospital ward, Tormo et al (2002) found twenty-two different types of spores, with total concentrations of 175-1396 spores/m<sup>3</sup>. The most frequently isolated were *Cladosporium*, *Ustilago* and various basidiospores. Considering the filtration requirements specified by ASHRAE (1999), virtually no spores should get through the filters at all. Tighe and Warden (1995) found airborne levels of bacteria to be about 104 cfu/m<sup>3</sup> in patient rooms and about 207 cfu/m<sup>3</sup> in general areas. Andrade and Brown (2003) found levels of bacteria as high as 160 cfu/m<sup>3</sup> in general areas.

A growing list of reports and clinical studies have addressed the effectiveness of UVGI systems for air disinfection in health care facilities. Upper room UVGI systems have been used at The New England Deaconess Hospital, The Infant and Children's Hospital in Boston, The Cradle in Evanston, and St. Luke's Hospital in New York for the control of respiratory infections, which decreased by a net average of 50% (Overholt and Betts 1940, Del Mundo and McKhann 1941, Sauer et al 1942, Higgons and Hyde 1947). The Home for Hebrew Infants in New York successfully brought a halt to a Varicella epidemic using UVGI (Wells 1955). Table 4 summarizes the reductions in nosocomial infections that have resulted from installation of UV systems for air disinfection, which have primarily been Upper Room UVGI systems.

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**Table 5: Results of Hospital Field Trials of UV Air Disinfection Systems**

Type	System	Location	Infection	Infection Cases		Decrease		Reference
				Before	After	Net	%	
Airborne Infections	Upper Room UVGI	The Cradle, Evanston	Respiratory infection	14.5%	4.6%	9.9%	68%	Sauer et al 1942
		St. Luke's Hospital, NY	Respiratory infection	10.0%	6.6%	3.4%	33%	Higgons & Hyde 1947
		Livermore, CA Veteran's Hospital	Influenza epidemic	19.0%	2.0%	17.0%	89%	McLean 1961
		North Central Bronx Hospital	TB conversions among staff	2.5%	1%	2%	60%	EPRI 1997
		Home for Hebrew Infants, NY	Varicella epidemic	97%	0%	97%	100%	Wells 1955
		<b>AVERAGE REDUCTION</b>						<b>70%</b>

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## **9. Hotel & Residential Air Disinfection Systems**

Hotels, apartments, private residential homes, and dormitories are all types of living facilities where individuals or families spend much of their time. In single family residences, families regularly exchange bacterial and viral infections, and are also subject to allergens that include mold spores and indoor fungal contamination. Families with children are especially subject to regular infections, including colds and flus that are routinely brought home from school by the youngest children. The need for protection against airborne pathogens has not yet been formally recognized by any agencies and not only do few homes have any type of air cleaning devices (i.e. filtration) but many homes have no ventilation at all, other than natural ventilation or air conditioners. All such living environments can benefit from air cleaning systems, including reductions in illnesses symptoms, allergies, and asthma symptoms (Bernstein et al 2006, Menzies et al 2003).

Hotels and dormitories are often unique residential environments in that the living quarters are typically small and do not always have direct supply air, only air conditioning or heaters under occupant control. Hotels will typically have one or more central air handling units providing air to the lobbies, hallways, restaurants, and other large areas. This supply air is intended to infiltrate into the individual hotel rooms. Sometimes the individual air conditioning units may have individual outside air dampers.

Although the central air handling units in hotels may have medium-to-low efficiency filters, the room air conditioners rarely have anything more than a simple dust filter fabric. As a result, these air conditioners tend to accumulate spores over time. With the presence of condensation, these spores may even amplify and lead to air quality problems in rooms.

Regular maintenance of room air conditioners normally involves removing the unit and cleaning with an acid or fungicide once a year. In a 200-400 unit hotel where only one or two units can be cleaned per day, this means that about half the units will have months of accumulation during the times of the year they need it most – summer and fall. As a result, hotel patrons often discover that when they turn on the air conditioner it produces a somewhat unpleasant odor. Figure 9 shows the results of samples taken from a hotel central air handling unit cooling coil.

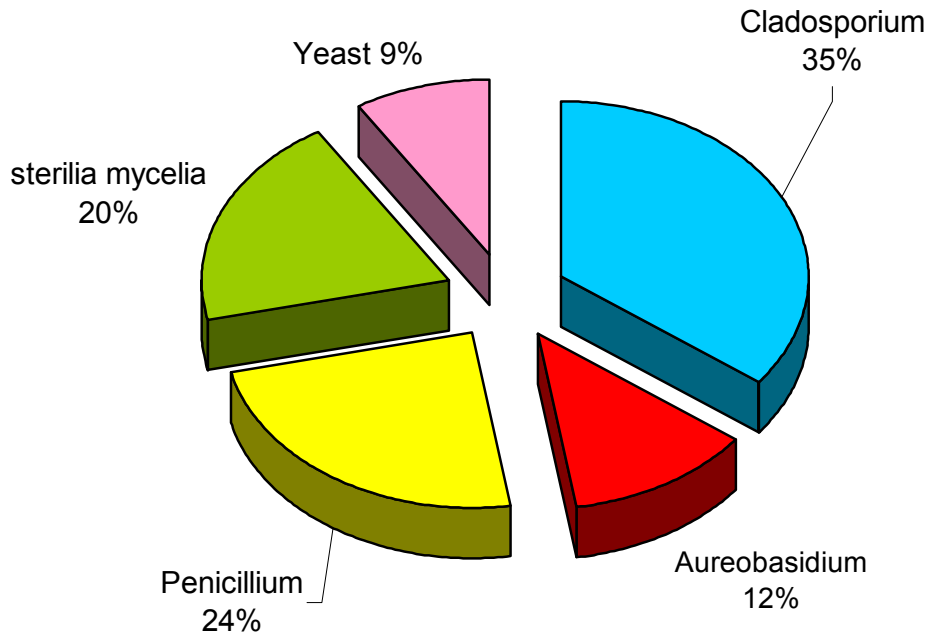


Figure 9: Fungal contamination of main air handling unit cooling coils of a large hotel. Based on author's data (Kowalski 2006).

Figure 10 shows the results of a survey taken by the author of the air in two hotel rooms in winter, in a hotel that had experienced water damage from a leaky roof. Although outdoor spore levels were less than  $10 \text{ cfu/m}^3$ , indoor spore levels in one of the rooms exceeded a few hundred  $\text{cfu/m}^3$ .

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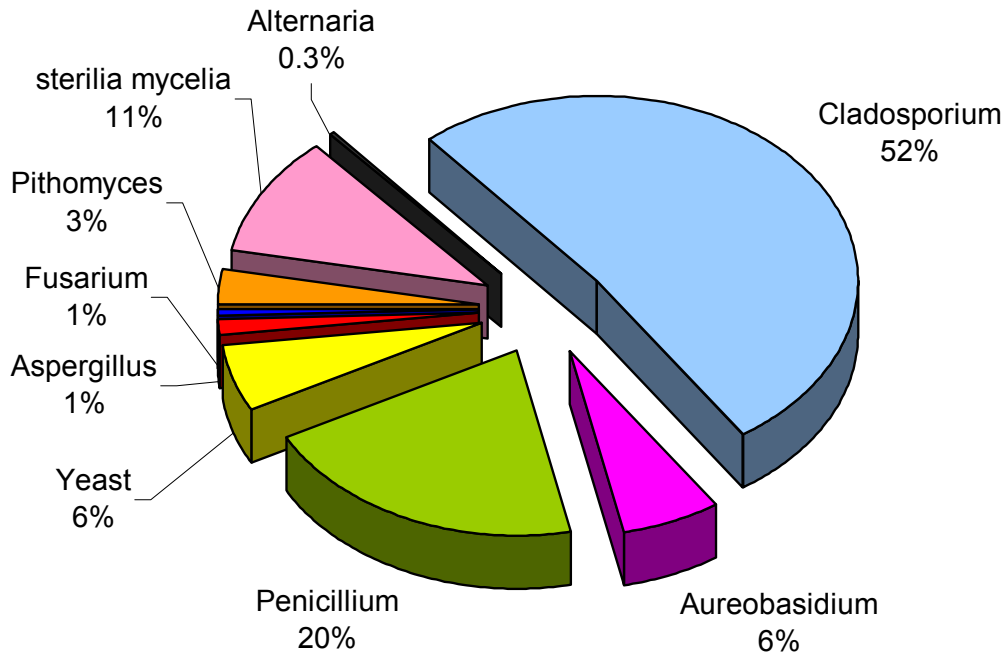


Figure 10: Airborne fungal spores in a hotel that had water damage. Based on average of two rooms from author's data (Kowalski 2006).

A UVGI lamp was installed in one room in the stand-alone A/C unit over the coils. Airborne fungal spores were measured before installation and after two weeks of operation. Figure 11 shows the results, in which airborne levels dropped significantly. Although there was no filter, other than a dust filter, on the A/C unit, this modification appeared to greatly reduce fungal spore levels in the room. UVGI has a very limited effect on fungal spores, which tend to be resistant to UV exposure, but the constant recirculation of the room air through the unit produces a 'chronic dosing, effect – that is, if a 1% kill rate is produced by a single pass, then after several hundred passes the total kill rate will approach 99%.

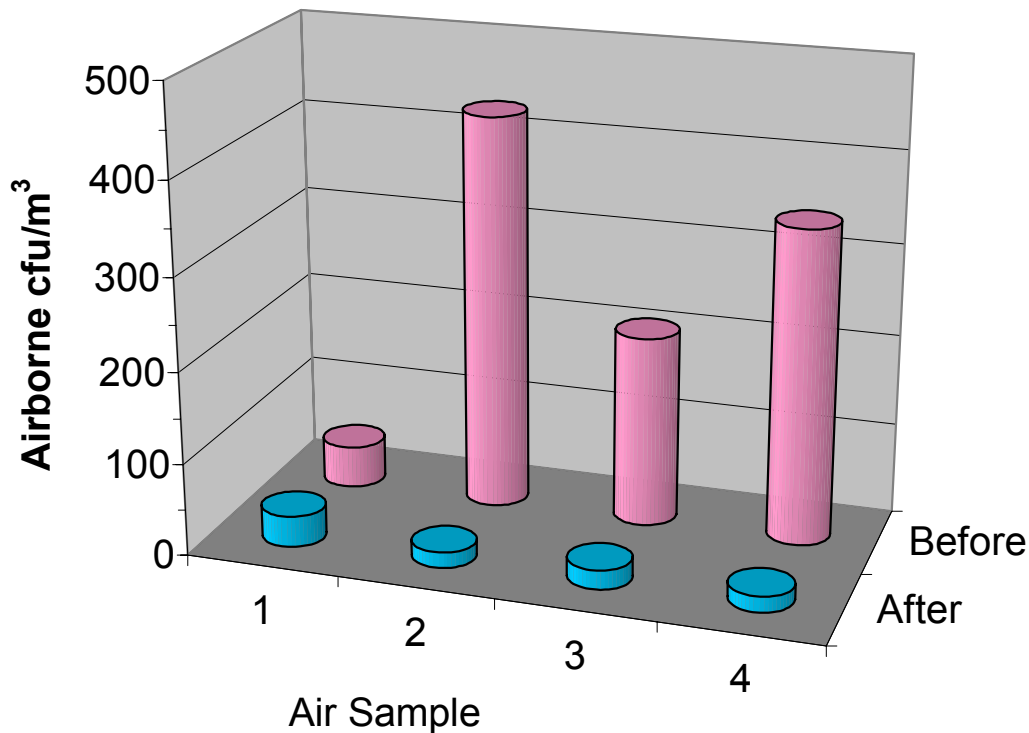


Figure 11: Airborne levels of fungi in a hotel room before and two weeks after UVGI installation. Based on author's data (Kowalski 2006).

One of the problems most frequently encountered in hotels is that the local room A/C units tend to accumulate mold spores on the coils and the dust filter. Condensation on the coils can then produce mold growth, which manifests itself as a foul smell when the A/C unit is turned on. These A/C units are typically subject to cleaning by maintenance personnel approximately once a year. Maintenance programs will often cycle through all the units in the hotel by removing them individually, cleaning them with steam or chemicals, and then reinstalling them. Such maintenance programs are not necessarily tied to the seasons, but may operate continuously throughout the year. What this means is that statistically up to one half of all A/C units will not have been cleaned in over six months, and if these six months are in the mold season (Spring through Fall) then up to one half the rooms may have moldy odors when the units are turned on. The odds are that many guests will experience moldy odors when they turn on the A/C units. Changes in maintenance programs may be one way to address the problem.

A more cost-effective way to control mold growth on local room A/C units, and also on central A/C units, is to install UVGI lamps around the cooling coils, provided there is sufficient space. This is relatively easy to accomplish for larger central air handling units with cooling coils, but can be problematic for local room A/C units due to the lack of space around the coils. Stand-alone A/C units



located in walls and overhead may have very limited space and UV lamps can be installed with appropriate reflectors to ensure coil exposure. Window A/C units may have no space to install UV lamps and might have to be modified to create such space, or else replaced with newer A/C units that can accommodate UV lamps.

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## **10. Electric Utilities and UV Air Disinfection**

The use of UV for air disinfection has long seen applications in the electric utilities, with the primary interest being energy savings from cooling coil disinfection systems. In fact, the first US installation of a UV cooling coil disinfection system was at an electric utility, Public Service of Omaha (PSO), who installed UV lamps in their air handling units in 1996 in an attempt to control mold growth around the cooling coils and drains pans. The system proved to be so effective in reducing odors and suppressing mold growth that all 28 air handling units were designated for the retrofit (Scheir 2000).

In 1996, the Central and Southwest Corporation (CSW), a holding company that owns America's second largest utility system and many electrical subsidiaries in Texas, Ohio, Oklahoma, Missouri, Louisiana, and Arkansas, began installing the first of 170 UV lamps in its building's air handling units (ELP 2000). By eliminating microbial growth as well as maintenance costs, the increase in cooling efficiency allowed the company to reduce its summertime energy consumption and save an estimated \$58,000 in the first year. They were able to switch to operating only three of their four 300 ton chillers, and saved an estimated 300 kW of power. There was a significant improvement in coil heat transfer efficiency and a reduction in airside pressure drop, which further resulted in a lowering of fan RPM -- thanks to the fortunate fact that they had variable frequency drives (VFD). That produced an estimated 28% reduction in total air conditioning system usage. Uncounted costs include the four-times-a-year coil cleaning maintenance program that is no longer needed since the coils stay permanently clean, and the fact that the change-out of the cooling coils has been pushed back indefinitely. The system energy savings has been particularly welcome to the utility due to costly, record-breaking heat waves in the South.

Utilities have also taken an interest in UV as a cost-effective method of solving health care problems. Consolidated Edison Company in New York is funding research to combat the spread of drug-resistant TB in the cities by installing Upper Room UVGI lamps in homeless shelters (Wald 1994). As part of a six-year plan, the effectiveness of the UV lamps will be monitored and studied by Dr. Philip Brickner of St. Vincent's Hospital and Dr. Ed Nardell (currently with the Harvard School of Public Health). The Electric Power Research Institute (EPRI) also has an interest in controlling TB with UV lights and in 1993 their Community Environmental Center funded the installation Upper Room fixtures in the VA Medical Center in Memphis (EPRI 1996). The Memphis VA hospital found that the UV installations provided cost-effective protection against airborne pathogens. New York Central Bronx Hospital installed Upper Room UVGI systems in 1995 to successfully control TB and nosocomial infections, in a project that was supported by The New York Power Authority (NYPA), who provide electricity to all the NYC Health and Hospitals Corporation facilities (EPRI 1997).

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## APPENDIX A: UV Rate Constants for Pathogens and Allergens

Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Acinetobacter baumannii	Bacteria	Veg	18	36	S	-	1.225	3598	Rastogi 2007
Acinetobacter baumannii	Bacteria	Veg	12	24	W	-	1.225	3598	Templeton 2009
Aeromonas	Bacteria	Veg	11	23	W	Wat	2.098	4740	Sako 1985
Aeromonas hydrophila	Bacteria	Veg	16	33	W	Wat	2.098	4740	Lilved 1996
B. atrophaeus (B. globigii)	Bacteria	Sp	144	288	Air	(Lo RH)	1.12	4140	EPA 2006
B. atrophaeus spores	Bacteria	Sp	1323	2647	W	Wat	1.12	4140	Shafaat 2006
Bacillus anthracis spores	Bacteria	Sp	411	822	W	Wat	1.118	5220	Nicholson 2003
Bacillus anthracis spores	Bacteria	Sp	45	90	S	-	1.118	5220	Sharp 1939
Bacillus anthracis spores	Bacteria	Sp	743	1486	S	-	1.118	5220	Knudson 1986
Bacillus cereus spores	Bacteria	Sp	267	534	S	-	1.118	5700	Weinberger 1984
Bacillus cereus spores	Bacteria	Sp	210	419	S	-	1.118	5700	Weisova 1966
Bacillus cereus spores	Bacteria	Sp	116	233	S	-	1.118	5700	Germaine 1973
Bacillus cereus spores	Bacteria	Sp	408	817	S	-	1.118	5700	Benoit 1990
Bacillus megatherium	Bacteria	Sp	273	546	S	-	1.12	4600	Hercik 1937
Bacillus megatherium	Bacteria	Veg	113	226	S	-	-	4600	Hercik 1937
Bacillus pumilus spores	Bacteria	Sp	50	100	W	Wat	-	-	Newcombe 2005
Bacillus subtilis	Bacteria	Veg	25	50	W	Wat	-	4210	Lojo 1995
Bacillus subtilis	Bacteria	Veg	14	27	Air	(Lo RH)	-	4210	Nakamura 1987
Bacillus subtilis spores	Bacteria	Sp	250	501	W	Wat	1.12	4140	Nicholson 2003
Bacillus subtilis spores	Bacteria	Sp	161	322	W	Wat	1.12	4140	Hoyer 2000
Bacillus subtilis spores	Bacteria	Sp	116	232	W	Wat	1.12	4140	Sommer 1989
Bacillus subtilis spores	Bacteria	Sp	220	440	W	Wat	1.12	4140	Sommer 1998
Bacillus subtilis spores	Bacteria	Sp	199	399	W	Wat	1.12	4140	Sommer 1999
Bacillus subtilis spores	Bacteria	Sp	77	154	W	Wat	1.12	4140	Qualis 1983
Bacillus subtilis spores	Bacteria	Sp	155	309	W	Wat	1.12	4140	Mamane-Gravetz 2005
Bacillus subtilis spores	Bacteria	Sp	89	178	W	Wat	1.12	4140	Horneck 1985
Bacillus subtilis spores	Bacteria	Sp	200	400	W	Wat	1.12	4140	Chang 1985
Bacillus subtilis spores	Bacteria	Sp	80	160	W	Wat	1.12	4140	DeGuchi 2005
Bacillus subtilis spores	Bacteria	Sp	94	187	S	-	1.12	4140	Rentschler 1941
Bacillus subtilis spores	Bacteria	Sp	68	137	S	-	1.12	4140	Munakata 1975
Bacillus subtilis spores	Bacteria	Sp	89	177	Air	Hi RH	1.12	4140	Peccia 2001a
Bacillus subtilis spores	Bacteria	Sp	85	171	Air	Lo RH	1.12	4140	Peccia 2001a
Bacillus subtilis spores	Bacteria	Sp	149	297	Air	Lo RH	1.12	4140	Ke 2009
Bacillus thuringiensis	Bacteria	Sp	2303	4605	W	Wat	-	-	Griego 1978
Burkholderia cenocepacia	Bacteria	Veg	58	116	W	Wat	0.707	7270	Abshire 1981
Burkholderia cepacia	Bacteria	Veg	11	22	Air	Lo RH	0.77	7700	Fletcher 2004
Burkholderia cepacia	Bacteria	Veg	22	44	Air	Hi RH	0.77	7700	Fletcher 2004
Campylobacter jejuni	Bacteria	Veg	11	22	W	Wat	2.12	1641	Wilson 1992
Campylobacter jejuni	Bacteria	Veg	29	58	W	Wat	2.12	1641	Butler 1987
Citrobacter diversus	Bacteria	Veg	32	64	W	Wat	1.2	-	Giese 2000
Citrobacter freundii	Bacteria	Veg	42	84	W	Wat	1.2	-	Zemke 1990
Citrobacter freundii	Bacteria	Veg	46	92	W	Wat	1.2	-	Giese 2000
Clostridium perfringens	Bacteria	Veg	38	77	W	Wat	5	3031	Hijnen 2006
Clostridium perfringens	Bacteria	Veg	135	271	-	-	5	3031	Jepson 1973
Clostridium tetani	Bacteria	Veg	49	98	-	-	5	2790	Jepson 1973
Corynebacterium diphtheriae	Bacteria	Veg	33	66	S	-	0.698	2480	Sharp 1939
Coxiella burnetii	Bacteria	Veg	15	30	W	Wat	0.283	2030	Little 1980
Deinococcus radiodurans	Bacteria	Veg	365	731	W	Wat	-	3280	Setlow 1964
Enterobacter cloacae	Bacteria	Veg	64	128	W	Wat	1.414	-	Zemke 1990
Escherichia coli	Bacteria	Veg	21	42	W	Wat	0.5	5490	Zelle 1955
Escherichia coli	Bacteria	Veg	53	107	W	Wat	0.5	5490	Tyrrell 1972
Escherichia coli	Bacteria	Veg	20	40	W	Wat	0.5	5490	Oguma 2001
Escherichia coli	Bacteria	Veg	47	93	W	Wat	0.5	5490	Kim 2002
Escherichia coli	Bacteria	Veg	43	87	W	Wat	0.5	5490	Hofmeister 1975
Escherichia coli	Bacteria	Veg	13	26	W	Wat	0.5	5490	Harris 1987
Escherichia coli	Bacteria	Veg	20	40	W	Wat	0.5	5490	Harm 1968
Escherichia coli	Bacteria	Veg	24	48	W	Wat	0.5	5490	David 1973
Escherichia coli	Bacteria	Veg	81	163	W	Wat	0.5	5490	Abshire 1981
Escherichia coli	Bacteria	Veg	25	49	S	-	0.5	5490	Sharp 1939
Escherichia coli	Bacteria	Veg	19	38	S	Hi RH	0.5	5490	Rentschler 1942
Escherichia coli	Bacteria	Veg	12	24	S	Lo RH	0.5	5490	Rentschler 1942
Escherichia coli	Bacteria	Veg	25	50	S	-	0.5	5490	Rentschler 1941

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Escherichia coli	Bacteria	Veg	20	39	S	-	0.5	5490	Quek 2008
Escherichia coli	Bacteria	Veg	34	69	S	-	0.5	5490	Kim 2002
Escherichia coli	Bacteria	Veg	55	110	S	-	0.5	5490	Hollaender 1955
Escherichia coli	Bacteria	Veg	8	16	S	-	0.5	5490	Collins 1971
Escherichia coli	Bacteria	Veg	3	6	Air	Lo RH	0.5	5490	Webb 1970
Escherichia coli	Bacteria	Veg	11	21	Air	Hi RH	0.5	5490	Webb 1970
Escherichia coli	Bacteria	Veg	11	21	Air	Hi RH	0.5	5490	Rentschler 1942
Escherichia coli	Bacteria	Veg	13	25	Air	Lo RH	0.5	5490	Rentschler 1942
Escherichia coli	Bacteria	Veg	15	29	Air	Lo RH	0.5	5490	Luckiesh 1949
Escherichia coli	Bacteria	Veg	2	5	Air	Lo RH	0.5	5490	Koller 1939
Escherichia coli	Bacteria	Veg	11	22	Air	Hi RH	0.5	5490	Koller 1939
Francisella tularensis	Bacteria	Veg	256	512	Air	Lo RH	0.2	1890	Beebe 1959
Francisella tularensis	Bacteria	Veg	288	576	Air	Hi RH	0.2	1890	Beebe 1959
Haemophilus influenzae	Bacteria	Veg	38	77	S	-	0.285	1910	Mongold 1992
Haemophilus influenzae Rd	Bacteria	Veg	13	26	W	Wat	0.285	1910	Barnhart 1970
Halobacterium sp. NRC-1	Bacteria	Veg	25	50	S	-	-	2571	Crowley 2006
Halobacterium salinarum	Bacteria	Veg	68	136	-	-	-	-	Martin 2000
Halomonas elongata	Bacteria	Veg	13	25	-	-	-	-	Martin 2000
Helicobacter pylori	Bacteria	Veg	33	67	W	Wat	2.1	1780	Hayes 2006
Klebsiella pneumoniae	Bacteria	Veg	42	84	W	Wat	0.671	5315	Zemke 1990
Klebsiella pneumoniae	Bacteria	Veg	68	136	W	Wat	0.671	5315	Giese 2000
Klebsiella terrigena	Bacteria	Veg	33	66	W	Wat	-	-	Wilson 1992
Legionella dumoffi	Bacteria	Veg	24	48	S	-	0.52	3400	Knudson 1985
Legionella bozemanii	Bacteria	Veg	19	26	W	Wat	0.52	3400	Yamamoto 1987
Legionella bozemanii	Bacteria	Veg	15	30	S	-	0.52	3400	Knudson 1985
Legionella gormanii	Bacteria	Veg	26	52	S	-	0.52	3400	Knudson 1985
Legionella jordanis	Bacteria	Veg	11	22	S	-	0.52	3400	Knudson 1985
Legionella longbeach	Bacteria	Veg	11	22	S	-	0.52	3400	Knudson 1985
Legionella micdadei	Bacteria	Veg	15	30	S	-	0.52	3400	Knudson 1985
Legionella oakridgensis	Bacteria	Veg	22	44	S	-	0.52	3400	Knudson 1985
Legionella pneumophila	Bacteria	Veg	13	26	W	Wat	0.52	3400	Yamamoto 1987
Legionella pneumophila	Bacteria	Veg	12	24	W	Wat	0.52	3400	Gilpin 1985
Legionella pneumophila	Bacteria	Veg	9	19	W	Wat	0.52	3400	Antopol 1979
Legionella pneumophila	Bacteria	Veg	5	10	S	-	0.52	3400	Knudson 1985
Legionella pneumophila	Bacteria	Veg	25	51	W	Wat	0.52	3400	Wilson 1992
Legionella pneumophila	Bacteria	Veg	16	32	W	Wat	0.52	3400	Oguma 2004 (LP)
Legionella pneumophila	Bacteria	Veg	19	38	W	Wat	0.52	3400	Oguma 2004 (MP)
Legionella wadsworthii	Bacteria	Veg	4	8	S	-	0.52	3400	Knudson 1985
Listeria monocytogenes	Bacteria	Veg	73	145	W	Wat	0.707	3130	Kim 2002
Listeria monocytogenes	Bacteria	Veg	156	311	S	-	0.707	3130	Kim 2002
Listeria monocytogenes	Bacteria	Veg	10	20	S	-	0.707	3130	Collins 1971
Micrococcus candidus	Bacteria	Veg	61	121	S	-	1.2	4050	Hollaender 1955
Micrococcus piltonensis	Bacteria	Veg	81	162	S	-	2.2	-	Rentschler 1941
Micrococcus sphaeroides	Bacteria	Veg	100	200	S	-	1.2	4050	Rentschler 1941
Moraxella	Bacteria	Veg	10965	20933	W	Wat	1.225	1940	Keller 1982
Mycobacterium avium-intra.	Bacteria	Veg	84	168	W	Wat	1.118	5470	David 1973
Mycobacterium avium	Bacteria	Veg	60	120	W	Wat	1.118	5470	Shin 2008
Mycobacterium avium	Bacteria	Veg	35	70	W	Wat	1.118	5470	McCarthy 1974
Mycobacterium bovis BCG	Bacteria	Veg	22	44	S	-	0.637	4340	Collins 1971
Mycobacterium bovis BCG	Bacteria	Veg	10	19	Air	50	0.637	4340	Riley 1976
Mycobacterium bovis BCG	Bacteria	Veg	12	24	Air	-	0.637	4340	Peccia 2002
Mycobacterium bovis BCG	Bacteria	Veg	19	38	Air	Lo RH	0.637	4340	Ko 2000
Mycobacterium bovis BCG	Bacteria	Veg	33	66	Air	Hi RH	0.637	4340	Ko 2000
Mycobacterium flavescens	Bacteria	Veg	120	240	W	Wat	0.637	-	David 1973
Mycobacterium fortuitum	Bacteria	Veg	68	136	W	Wat	0.637	5000	David 1973
Mycobacterium fortuitum	Bacteria	Veg	96	192	W	Wat	0.637	5000	David 1971
Mycobacterium kansasii	Bacteria	Veg	80	160	W	Wat	0.637	4345	David 1973
Mycobacterium marinum	Bacteria	Veg	76	152	W	Wat	0.637	6485	David 1973
Mycobacterium marinum	Bacteria	Veg	743	1486	W	Wat	0.637	6485	David 1971
Mycobacterium parafortuitum	Bacteria	Veg	13	26	Air	50	0.637	-	Peccia 2001
Mycobacterium parafortuitum	Bacteria	Veg	46	92	Air	95	0.637	-	Peccia 2001
Mycobacterium parafortuitum	Bacteria	Veg	19	38	Air	50	0.637	-	Xu 2003



## APPENDIX A: UV Rate Constants for Pathogens and Allergens

Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Mycobacterium phlei	Bacteria	Veg	76	152	W	Wat	0.637	6000	David 1973
Mycobacterium phlei	Bacteria	Veg	63	126	Air	50	0.637	6000	Riley 1976
Mycobacterium phlei	Bacteria	Veg	23	46	Air	50	0.637	6000	Kethley 1973
Mycobacterium phlei	Bacteria	Veg	16	33	Air	50	0.637	6000	Gillis 1974
Mycobacterium smegmatis	Bacteria	Veg	108	216	W	Wat	0.637	6980	David 1973
Mycobacterium smegmatis	Bacteria	Veg	1047	2093	W	Wat	0.637	6980	David 1971
Mycobacterium smegmatis	Bacteria	Veg	68	135	W	Wat	0.637	6980	Boshoff 2003
Mycobacterium smegmatis	Bacteria	Veg	12	24	Air	50	0.637	6980	Gillis 1974
Mycobacterium terrae	Bacteria	Veg	50	100	W	Wat	0.637	-	Bohrerova 2006
Mycobacterium tuberculosis	Bacteria	Veg	28	56	W	Wat	0.637	4400	David 1973
Mycobacterium tuberculosis	Bacteria	Veg	77	154	W	Wat	0.637	4400	David 1971
Mycobacterium tuberculosis	Bacteria	Veg	74	149	W	Wat	0.637	4400	Boshoff 2003
Mycobacterium tuberculosis	Bacteria	Veg	11	22	S	-	0.637	4400	Collins 1971
Mycobacterium tuberculosis	Bacteria	Veg	5	10	Air	50	0.637	4400	Riley 1976
Mycoplasma arthritis	Bacteria	Veg	7	15	S	-	0.177	816	Furness 1977
Mycoplasma fermentans	Bacteria	Veg	9	18	S	-	0.177	816	Furness 1977
Mycoplasma hominis	Bacteria	Veg	7	14	S	-	0.177	816	Furness 1977
Mycoplasma Orale type 1	Bacteria	Veg	11	21	S	-	0.177	816	Furness 1977
Mycoplasma Orale type 2	Bacteria	Veg	6	12	S	-	0.177	816	Furness 1977
Mycoplasma pneumoniae	Bacteria	Veg	8	17	S	-	0.177	816	Furness 1977
Mycoplasma salivarium	Bacteria	Veg	11	22	S	-	0.177	816	Furness 1977
Neisseria catarrhalis	Bacteria	Veg	44	88	S	-	0.177	816	Rentschler 1941
Nocardia asteroides	Bacteria	Veg	280	560	S	-	1.118	6021	Chick 1963
Phytomonas tumefaciens	Bacteria	Veg	44	88	S	-	-	-	Rentschler 1941
Proteus mirabilis	Bacteria	Veg	8	16	W	Wat	0.494	4063	Hofmeister 1975
Proteus vulgaris	Bacteria	Veg	30	60	S	-	0.291	3462	Rentschler 1941
Pseudomonas aeruginosa	Bacteria	Veg	10	20	W	Wat	0.494	5900	Gilpin 1985
Pseudomonas aeruginosa	Bacteria	Veg	172	344	W	Wat	0.494	5900	Dolman 1989
Pseudomonas aeruginosa	Bacteria	Veg	36	70	W	Wat	0.494	5900	Abshire 1981
Pseudomonas aeruginosa	Bacteria	Veg	55	110	W	Wat	0.494	5900	Zelle 1955
Pseudomonas aeruginosa	Bacteria	Veg	55	110	S	-	0.494	5900	Hollaender 1955
Pseudomonas aeruginosa	Bacteria	Veg	22	44	S	-	0.494	5900	Elasri 1999
Pseudomonas aeruginosa	Bacteria	Veg	10	19	S	-	0.494	5900	Collins 1971
Pseudomonas aeruginosa	Bacteria	Veg	4	8	Air	(Lo RH)	0.494	5900	Sharp 1940
Pseudomonas diminuta	Bacteria	Veg	96	193	W	Wat	0.5	-	Abshire 1981
Pseudomonas fluorescens	Bacteria	Veg	35	70	S	-	0.5	6438	Rentschler 1941
Pseudomonas fluorescens	Bacteria	Veg	3	10	Air	50	0.5	6438	vanOsdell 2002
Pseudomonas maltophilia	Bacteria	Veg	70	140	W	Wat	0.5	-	Abshire 1981
Pseudomonas putrefaciens	Bacteria	Veg	87	173	W	Wat	0.5	-	Abshire 1981
Rickettsia prowazekii	Bacteria	Veg	13	26	W	Wat	0.6	1110	Allen 1954
Salmonella spp.	Bacteria	Veg	11	22	W	Wat	0.8	4746	Yaun 2003
Salmonella anatum	Bacteria	Veg	60	120	W	Wat	0.8	-	Tosa 1998
Salmonella derby	Bacteria	Veg	36	72	W	Wat	0.8	-	Tosa 1998
Salmonella enteritidis	Bacteria	Veg	10	21	S	-	0.8	4746	Collins 1971
Salmonella enteritidis	Bacteria	Veg	33	66	W	Wat	0.8	4746	Tosa 1998
Salmonella infantis	Bacteria	Veg	20	40	W	Wat	0.8	-	Tosa 1998
Salmonella typhi	Bacteria	Veg	21	43	W	Wat	0.806	4791	Zelle 1955
Salmonella typhi	Bacteria	Veg	30	60	W	Wat	0.806	4791	Chang 1985
Salmonella typhi	Bacteria	Veg	21	43	S	-	0.806	4791	Sharp 1939
Salmonella typhi	Bacteria	Veg	9	18	W	Wat	0.806	4791	Wilson 1992
Salmonella typhimurium	Bacteria	Veg	295	590	W	Wat	0.8	4950	Kim 2002
Salmonella typhimurium	Bacteria	Veg	18	36	W	Wat	0.8	-	Tosa 1998
Sarcina lutea	Bacteria	Veg	197	394	S	-	1.48	-	Rentschler 1941
Serratia indica	Bacteria	Veg	209	419	Air	42-51	0.632	-	Harstad 1954
Serratia marcescens	Bacteria	Veg	22	44	W	Wat	0.632	5114	Zelle 1955
Serratia marcescens	Bacteria	Veg	105	210	W	Wat	0.632	5114	Harris 1993
Serratia marcescens	Bacteria	Veg	22	44	S	-	0.632	5114	Sharp 1939
Serratia marcescens	Bacteria	Veg	22	44	S	-	0.632	5114	Rentschler 1941
Serratia marcescens	Bacteria	Veg	8	17	S	-	0.632	5114	Hollaender 1955
Serratia marcescens	Bacteria	Veg	10	21	S	-	0.632	5114	Collins 1971
Serratia marcescens	Bacteria	Veg	2	5	Air	Lo RH	0.632	5114	Fletcher 2003
Serratia marcescens	Bacteria	Veg	24	48	Air	Hi RH	0.632	5114	Fletcher 2003

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
<i>Serratia marcescens</i>	Bacteria	Veg	4	8	Air	22-33	0.632	5114	Ko 2000
<i>Serratia marcescens</i>	Bacteria	Veg	115	230	Air	Hi RH	0.632	5114	Ko 2000
<i>Serratia marcescens</i>	Bacteria	Veg	5	10	Air	(Lo RH)	0.632	5114	Sharp 1940
<i>Serratia marcescens</i>	Bacteria	Veg	20	41	Air	(Lo RH)	0.632	5114	Nakamura 1987
<i>Serratia marcescens</i>	Bacteria	Veg	33	66	Air	95	0.632	5114	Peccia 2001
<i>Serratia marcescens</i>	Bacteria	Veg	3	5	Air	68	0.632	5114	Lai 2004
<i>Serratia marcescens</i>	Bacteria	Veg	3	11	Air	50	0.632	5114	vanOsdell 2002
<i>Serratia marcescens</i>	Bacteria	Veg	5	10	Air	50	0.632	5114	Peccia 2001
<i>Serratia marcescens</i>	Bacteria	Veg	1	2	Air	36	0.632	5114	Lai 2004
<i>Shigella dysenteriae</i>	Bacteria	Veg	18	35	W	Wat	0.801	4369	Wilson 1992
<i>Shigella paradysenteriae</i>	Bacteria	Veg	17	34	S	-	0.801	-	Sharp 1939
<i>Shigella sonnei</i>	Bacteria	Veg	18	37	W	Wat	0.801	-	Chang 1985
<i>Spirillum rubrum</i>	Bacteria	Veg	44	88	S	-	-	-	Rentschler 1941
<i>Staphylococcus albus</i>	Bacteria	Veg	18	37	S	-	1.06	2900	Sharp 1939
<i>Staphylococcus albus</i>	Bacteria	Veg	33	66	S	-	1.06	2900	Rentschler 1941
<i>Staphylococcus albus</i> (1)	Bacteria	Veg	23	46	Air	(Lo RH)	1.06	2900	Rentschler 1942
<i>Staphylococcus albus</i> (2)	Bacteria	Veg	52	105	Air	(Lo RH)	1.06	2900	Rentschler 1942
<i>Staphylococcus aureus</i>	Bacteria	Veg	52	105	W	Wat	0.866	2800	Dolman 1989
<i>Staphylococcus aureus</i>	Bacteria	Veg	27	54	W	Wat	0.866	2800	Chang 1985
<i>Staphylococcus aureus</i>	Bacteria	Veg	56	111	W	Wat	0.866	2800	Abshire 1981
<i>Staphylococcus aureus</i>	Bacteria	Veg	30	60	S	-	0.866	2800	Sturm 1932
<i>Staphylococcus aureus</i>	Bacteria	Veg	50	99	S	-	0.866	2800	Hollaender 1955
<i>Staphylococcus aureus</i>	Bacteria	Veg	66	132	S	-	0.866	2800	Gates 1934
<i>Staphylococcus aureus</i>	Bacteria	Veg	26	52	S	-	0.866	2800	Sharp 1939
<i>Staphylococcus aureus</i>	Bacteria	Veg	37	74	S	-	0.866	2800	Luckesh 1949
<i>Staphylococcus aureus</i>	Bacteria	Veg	19	39	S	-	0.866	2800	Gates 1929
<i>Staphylococcus aureus</i>	Bacteria	Veg	20	41	Air	(Lo RH)	0.866	2800	Nakamura 1987
<i>Staphylococcus aureus</i>	Bacteria	Veg	7	13	Air	(Lo RH)	0.866	2800	Sharp 1940
<i>Staphylococcus aureus</i>	Bacteria	Veg	2	5	Air	-	0.866	2800	Luckesh 1949
<i>Staphylococcus aureus</i>	Bacteria	Veg	2	5	Air	(Lo RH)	0.866	2800	Luckesh 1946
<i>Staphylococcus epidermis</i>	Bacteria	Veg	161	321	W	Wat	0.866	2640	Harris 1993
<i>Staphylococcus epidermis</i>	Bacteria	Veg	14	28	Air	50	0.866	2640	vanOsdell 2002
<i>Staphylococcus epidermis</i>	Bacteria	Veg	29	576	Air	85	0.866	2640	vanOsdell 2002
<i>Staphylococcus epidermis</i>	Bacteria	Veg	20	41	Air	(Lo RH)	0.866	2640	Nakamura 1987
<i>Staphylococcus epidermis</i>	Bacteria	Veg	22	44	Air	(Lo RH)	0.866	2640	Furuhashi 1989
<i>Streptococcus agalactiae</i>	Bacteria	Veg	5	11	Air	-	0.707	2127	Luckesh 1949
<i>Streptococcus faecalis</i>	Bacteria	Veg	55	50	W	Wat	0.707	-	Chang 1985
<i>Streptococcus faecalis</i>	Bacteria	Veg	195	390	W	Wat	0.707	-	Sanz 2007
<i>Streptococcus faecalis</i>	Bacteria	Veg	31	61	W	Wat	0.707	-	Harris 1987
<i>Streptococcus faecalis</i>	Bacteria	Veg	120	240	W	Wat	0.707	-	Abshire 1981
<i>Streptococcus faecium</i>	Bacteria	Veg	45	90	W	Wat	0.632	5114	Martiny 1988
<i>Streptococcus haemolyticus</i>	Bacteria	Veg	22	43	S	-	0.707	2680	Sharp 1939
<i>Streptococcus lactis</i>	Bacteria	Veg	62	123	S	-	0.707	-	Rentschler 1941
<i>Streptococcus pneumoniae</i>	Bacteria	Veg	468	936	S	-	0.707	-	Gritz 1990
<i>Streptococcus pyogenes</i>	Bacteria	Veg	4	75	S	-	0.894	1900	Lidwell 1950
<i>Streptococcus pyogenes</i>	Bacteria	Veg	1	3	Air	-	0.894	1900	Luckesh 1949
<i>Streptococcus viridans</i>	Bacteria	Veg	20	40	S	-	0.707	-	Sharp 1939
<i>Streptomyces coelicolor</i>	Bacteria	Veg	60	120	W	Wat	-	8667	Jagger 1970
<i>Streptomyces griseus</i>	Bacteria	Veg	129	259	W	Wat	-	8545	Kelner 1949
<i>Streptomyces griseus</i>	Bacteria	Veg	60	120	W	Wat	-	8545	Jagger 1970
<i>Vibrio cholerae</i>	Bacteria	Veg	17	34	W	Wat	2.12	4148	Wilson 1992
<i>Vibrio ordalii</i>	Bacteria	Veg	18	37	W	Wat	2.12	-	Sako 1985
<i>Vibrio parahaemolyticus</i>	Bacteria	Veg	8	15	W	Wat	2.12	5165	Nozu 1977
<i>Yersinia enterocolitica</i>	Bacteria	Veg	15	30	W	Wat	0.707	4615	Butler 1987
<i>Yersinia enterocolitica</i>	Bacteria	Veg	28	57	W	Wat	0.707	4615	Carlson 1975
<i>Yersinia enterocolitica</i>	Bacteria	Veg	11	23	W	Wat	0.707	4615	Butler 1987
<i>Yersinia enterocolitica</i>	Bacteria	Veg	13	27	W	Wat	0.707	4615	Wilson 1992

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Adenovirus	Virus	dsDNA	34	68	Air	Hi RH	0.079	35.937	Walker 2007
Adenovirus	Virus	dsDNA	59	118	Air	Lo RH	0.079	35.937	Walker 2007
Adenovirus	Virus	dsDNA	42	84	Air	50	0.079	36.001	Jensen 1964
Adenovirus	Virus	dsDNA	903	1806	W	Wat	0.079	36.001	Wasserman 1962
Adenovirus type 1	Virus	dsDNA	299	598	W	Wat	0.079	36.001	Battigelli 1993
Adenovirus type 1	Virus	dsDNA	350	700	W	Wat	0.079	36.001	Nwachuku 2005
Adenovirus type 2	Virus	dsDNA	400	800	S	-	0.079	35.937	Day 1974
Adenovirus type 2	Virus	dsDNA	640	1279	W	Wat	0.079	35.937	Rainbow 1970
Adenovirus type 2	Virus	dsDNA	490	980	W	Wat	0.079	36.001	Rainbow 1973
Adenovirus type 2	Virus	dsDNA	533	1066	W	Wat	0.079	35.937	Linden 2007 (LP lamp)
Adenovirus type 2	Virus	dsDNA	150	299	W	Wat	0.079	35.937	Linden 2007 (MP lamp)
Adenovirus type 2	Virus	dsDNA	300	600	W	Wat	0.079	35.937	Shin 2005
Adenovirus type 2	Virus	dsDNA	400	800	W	Wat	0.079	35.937	Gerba 2002
Adenovirus type 2	Virus	dsDNA	276	552	W	Wat	0.079	35.937	Ballester 2004
Adenovirus type 4	Virus	dsDNA	921	1842	W	Wat	0.079	35.937	Nwachuku 2005
Adenovirus type 15	Virus	dsDNA	396	793	W	Wat	0.079	35.937	Thompson 2003
Adenovirus type 40	Virus	dsDNA	300	600	S	-	0.069	36.001	Meng 1996
Adenovirus type 40	Virus	dsDNA	546	1091	W	Wat	0.069	36.001	Thurston-Enriquez 2003
Adenovirus type 41	Virus	dsDNA	240	472	S	-	0.069	36.001	Meng 1996
Adenovirus type 41	Virus	dsDNA	425	850	W	Wat	0.069	36.001	Malley 2004
Adenovirus type 41	Virus	dsDNA	555	1110	W	Wat	0.069	36.001	Ko 2005
Adenovirus type 41	Virus	dsDNA	600	1199	W	Wat	0.069	36.001	Durance 2005
Adenovirus type 5	Virus	dsDNA	400	800	W	Wat	0.084	35.938	Durance 2005
Adenovirus type 5	Virus	dsDNA	541	1081	W	Wat	0.084	36.598	Wang 2004
Adenovirus type 5	Virus	dsDNA	720	1439	W	Wat	0.084	35.598	Nwachuku 2005
Adenovirus type 6	Virus	dsDNA	390	780	W	Wat	0.079	35.937	Nwachuku 2005
Adenovirus type 6	Virus	dsDNA	400	800	W	Wat	0.079	35.937	Battigelli 1993
Avian Influenza virus	Virus	ssRNA	22	43	W	Wat	0.09	-	Lucio-Forster 2006
Avian Influenza virus	Virus	ssRNA	30	60	W	Wat	0.098	-	Deshmukh 1968
Avian Leukosis virus (RSA)	Virus	ssRNA	631	1262	W	Wat	0.107	7.286	Levinson 1966
Avian Sarcoma virus	Virus	ssDNA	155	309	W	Wat	0.098	7	Owada 1976
Avian Sarcoma virus	Virus	ssDNA	381	762	W	Wat	0.098	7	Bister 1977
Bacteriophage MS2	phage	ssRNA	26	96	Air	Hi RH	0.02	3.569	Walker 2007
Bacteriophage MS2	phage	ssRNA	61	121	Air	Lo RH	0.02	3.569	Walker 2007
Bacteriophage MS2	phage	ssRNA	3	6	Air	Lo RH	0.02	3.569	Tseng 2005
Bacteriophage MS2	phage	ssRNA	4	7	Air	Hi RH	0.02	3.569	Tseng 2005
Bacteriophage MS2	phage	ssRNA	135	269	W	Wat	0.02	3.569	Tree 1997
Bacteriophage MS2	phage	ssRNA	427	854	W	Wat	0.02	3.569	Sommer 2001
Bacteriophage MS2	phage	ssRNA	193	387	W	Wat	0.02	3.569	Sommer 1998
Bacteriophage MS2	phage	ssRNA	419	837	W	Wat	0.02	3.569	Mamane-Gravetz 2005
Bacteriophage MS2	phage	ssRNA	368	737	W	Wat	0.02	3.569	Templeton 2006
Bacteriophage MS2	phage	ssRNA	295	590	W	Wat	0.02	3.569	Ko 2005
Bacteriophage MS2	phage	ssRNA	40	80	W	Wat	0.02	3.569	Weidenmann 1993
Bacteriophage MS2	phage	ssRNA	173	346	W	Wat	0.02	3.569	Wilson 1992
Bacteriophage MS2	phage	ssRNA	275	550	W	Wat	0.02	3.569	Thurston-Enriquez 2003
Bacteriophage MS2	phage	ssRNA	217	434	W	Wat	0.02	3.569	Batch 2004
Bacteriophage MS2	phage	ssRNA	250	501	W	Wat	0.02	3.569	Battigelli 1993
Bacteriophage MS2	phage	ssRNA	217	434	W	Wat	0.02	3.569	Simonet 2006
Bacteriophage MS2	phage	ssRNA	217	433	W	Wat	0.02	3.569	deRodaHusman 2004
Bacteriophage MS2	phage	ssRNA	213	426	W	Wat	0.02	3.569	Butkus 2004
Bacteriophage MS2	phage	ssRNA	187	374	W	Wat	0.02	3.569	Oppenheimer 1997
Bacteriophage MS2	phage	ssRNA	169	339	W	Wat	0.02	3.569	Nuanualsuwan 2002
Bacteriophage MS2	phage	ssRNA	164	328	W	Wat	0.02	3.569	Rauth 1965
Bacteriophage MS2	phage	ssRNA	150	299	W	Wat	0.02	3.569	Shin 2005
Bacteriophage MS2	phage	ssRNA	140	281	W	Wat	0.02	3.569	Meng 1996
Bacteriophage MS2	phage	ssRNA	198	397	W	Wat	0.02	3.569	Nieuwstad 1994
Bacteriophage MS2	phage	ssRNA	228	456	W	Wat	0.02	3.569	Lazarova 2004
Bacteriophage MS2	phage	ssRNA	245	490	W	Wat	0.02	3.569	Thompson 2003

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Berne virus	Virus	ssRNA	13	25	W	Wat	0.13	20	Weiss 1986
BLV	Virus	ssRNA	1799	3598	W	Wat	0.1	8.419	Shimizu 2004
BLV	Virus	ssRNA	221	443	W	Wat	0.1	8.419	Guillemin 1981
Borna virus	Virus	ssRNA	79	158	W	Wat	0.09	8.91	Danner 1979
Bovine Calicivirus	Virus	ssDNA	95	190	W	Wat	0.02	7.45	Malley 2004
Bovine Parvovirus	Virus	ssDNA	35	70	W	-	0.02	5.517	vonBrodorotti 1982
Canine Calicivirus	Virus	ssRNA	67	133	W	Wat	0.037	8.513	deRodaHusman 2004
Canine hepatic Adenovirus	Virus	dsDNA	265	530	W	Wat	0.08	36.5	vonBrodorotti 1982
Coronavirus	Virus	ssRNA	3	12	Air	50	0.113	30.738	Walker 2007
Coronavirus	Virus	ssRNA	7	14	W	Wat	0.113	30.738	Weiss 1986
Coronavirus (SARS)	Virus	ssRNA	226	461	W	Wat	0.113	29.751	Kariw a 2004
Coronavirus (SARS)	Virus	ssRNA	3046	6091	W	Wat	0.113	29.751	Darnell 2004
Coxsackievirus	Virus	ssRNA	21	41	Air	60	0.027	7.413	Jensen 1964
Coxsackievirus	Virus	ssRNA	128	230	W	Wat	0.027	7.413	Hill 1970
Coxsackievirus	Virus	ssRNA	86	172	W	Wat	0.027	7.413	Havelaar 1987
Coxsackievirus B3	Virus	ssRNA	80	160	W	Wat	0.027	7.413	Gerba 2002
Coxsackievirus B4	Virus	ssRNA	60	120	W	Wat	0.027	7.413	Shin 2005
Coxsackievirus B5	Virus	ssRNA	95	190	W	Wat	0.027	7.413	Gerba 2002
Coxsackievirus B5	Virus	ssRNA	72	145	W	Wat	0.027	7.413	Battigelli 1993
Echovirus (Parechovirus)	Virus	ssRNA	106	210	W	Wat	0.024	7.354	Hill 1970
Echovirus 1	Virus	ssRNA	80	160	W	Wat	0.024	7.354	Gerba 2002
Echovirus 2	Virus	ssRNA	70	140	W	Wat	0.024	7.354	Gerba 2002
Encephalomyocarditis virus	Virus	ssRNA	50	99	W	Wat	0.025	7.835	Ross 1971
Encephalomyocarditis virus	Virus	ssRNA	52	103	W	Wat	0.025	7.835	Rauth 1965
Encephalomyocarditis virus	Virus	ssRNA	65	130	W	Wat	0.025	7.835	Zavadova 1968
Epstein-Barr virus (EBV)	Virus	ssDNA	162	324	W	Wat	-	-	Henderson 1978
Feline Calicivirus (FeCV)	Virus	ssRNA	434	869	W	Wat	0.034	7.683	Nuanualsuw an 2002
Feline Calicivirus (FeCV)	Virus	ssRNA	80	160	W	Wat	0.034	7.683	Thurston-Enriquez 2003
Feline Calicivirus (FeCV)	Virus	ssRNA	40	80	W	Wat	0.034	7.683	deRodaHusman 2004
Feline Calicivirus (FeCV)	Virus	ssRNA	44	87	W	Wat	0.034	7.683	Tree 2005
Friend Murine Leukemia	Virus	ssRNA	320	640	W	Wat	0.094	8.323	Yoshikura 1971
Hepatitis A virus	Virus	dsDNA	40	80	W	Wat	0.027	7.478	Battigelli 1993
Hepatitis A virus	Virus	dsDNA	45	90	W	Wat	0.027	7.478	Wang 2004
Hepatitis A virus	Virus	dsDNA	50	100	W	Wat	0.027	7.478	Weidenmann 1993
Hepatitis A virus	Virus	dsDNA	92	184	W	Wat	0.027	7.478	Wang 1995
Hepatitis A virus	Virus	dsDNA	98	197	W	Wat	0.027	7.478	Wilson 1992
Hepatitis A virus	Virus	dsDNA	307	614	W	Wat	0.027	7.478	Nuanualsuw an 2002
Herpes simplex virus (HRE)	Virus	dsDNA	40	80	W	Wat	0.18	152.261	Powell 1959
Herpes simplex virus Type 1	Virus	dsDNA	71	141	W	Wat	0.184	152.261	Bockstahler 1976
Herpes simplex virus Type 1	Virus	dsDNA	110	220	W	Wat	0.184	152.261	Selsky 1978
Herpes simplex virus Type 1	Virus	dsDNA	25	49	W	Wat	0.184	152.261	Lytle 1971
Herpes Simplex virus Type 1	Virus	dsDNA	35	70	W	Wat	0.184	152.261	Ross 1971
Herpes Simplex virus Type 1	Virus	dsDNA	21	42	W	Wat	0.184	152.261	Albrecht 1974
Herpes Simplex virus Type 1	Virus	dsDNA	41	81	W	Wat	0.184	152.261	Henderson 1978
Herpes Simplex virus Type 2	Virus	dsDNA	40	80	W	Wat	0.173	154.746	Wolff 1973
Herpes Simplex virus Type 2	Virus	dsDNA	41	82	W	Wat	0.173	154.746	Ross 1971
Herpes Simplex virus Type 2	Virus	dsDNA	75	150	W	Wat	0.173	154.746	Ryan 1986
Herpes Simplex virus Type 2	Virus	dsDNA	20	39	W	Wat	0.173	154.746	Albrecht 1974
HIV-1	Virus	ssRNA	280	560	W	Wat	0.125	9.181	Yoshikura 1989
HTLV-1	Virus	ssRNA	20	40	W	Wat	0.102	8.507	Shimizu 2004
Human Cytomegalovirus	Virus	dsDNA	658	1316	S	-	0.1	-	Hirai 1977
Human Cytomegalovirus	Virus	dsDNA	50	100	S	-	0.1	-	Albrecht 1974
Influenza A virus	Virus	ssRNA	19	39	Air	68	0.098	13.498	Jensen 1964
Influenza A virus	Virus	ssRNA	20	39	W	Wat	0.098	13.498	Ross 1971
Influenza A virus	Virus	ssRNA	48	96	W	Wat	0.098	13.498	Hollaender 1944
Influenza A virus	Virus	ssRNA	17	33	W	Wat	0.098	13.498	Abraham 1979
Kilham Rat Virus (parvovirus)	Virus	ssDNA	30	60	W	Wat	0.022	5	Proctor 1972

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Measles virus	Virus	ssRNA	22	44	W	Wat	0.329	15.894	DiStefano 1976
Mengovirus	Virus	dsRNA	162	324	W	Wat	-	6.1	Miller 1974
Minute Virus of Mice (MVM)	Virus	ssDNA	28	56	W	Wat	0.022	5.081	Vos 1981
Minute Virus of Mice (MVM)	Virus	ssDNA	17	34	W	Wat	0.022	5.081	Rommelaere 1981
Murine Cytomegalovirus	Virus	dsDNA	46	92	W	Wat	0.104	230.278	Shanley 1982
Moloney Murine Leukemia v.	Virus	ssRNA	115	230	W	Wat	0.094	8.332	Nomura 1972
Moloney Murine Leukemia v.	Virus	ssRNA	370	740	W	Wat	0.094	8.332	Guillemain 1981
Moloney Murine Leukemia v.	Virus	ssRNA	280	560	W	Wat	0.094	8.332	Yoshikura 1989
Murine Norovirus (MNV)	Virus	ssRNA	76	151	W	Wat	0.032	7.382	Lee 2008
Murine sarcoma virus	Virus	ssRNA	237	475	W	Wat	0.12	5.833	Nomura 1972
Murine sarcoma virus	Virus	ssRNA	144	288	W	Wat	0.12	5.833	Kelloff 1970
Murine sarcoma virus	Virus	ssRNA	299	598	W	Wat	0.12	5.833	Yoshikura 1971
Newcastle Disease Virus	Virus	ssRNA	8	17	W	Wat	0.212	15.186	vonBrodorotti 1982
Newcastle Disease Virus	Virus	ssRNA	45	90	W	Wat	0.212	15.186	Levinson 1966
Newcastle Disease Virus	Virus	ssRNA	16	32	S	-	0.212	15.186	Rubin 1959
Parvovirus H-1	Virus	ssDNA	25	50	W	Wat	0.022	6.194	Cornellis 1982
Poliovirus	Virus	dsRNA	44	88	S	-	0.0248	7.44	Ma 1994
Poliovirus type 1	Virus	dsRNA	41	82	S	-	0.0248	7.44	Meng 1996
Poliovirus	Virus	dsRNA	71	142	W	Wat	0.0248	7.44	Helenjaris 1977
Poliovirus	Virus	dsRNA	75	150	W	Wat	0.0248	7.44	Shin 2005
Poliovirus	Virus	dsRNA	95	190	W	Wat	0.0248	7.44	Bishop 1967
Poliovirus	Virus	dsRNA	52	103	W	Wat	0.0248	7.44	Dulbecco 1955
Poliovirus type 1	Virus	dsRNA	67	133	W	Wat	0.0248	7.44	Chang 1985
Poliovirus type 1	Virus	dsRNA	72	144	W	Wat	0.0248	7.44	Wilson 1992
Poliovirus type 1	Virus	dsRNA	96	192	W	Wat	0.0248	7.44	Wetz 1982
Poliovirus type 1	Virus	dsRNA	100	200	W	Wat	0.0248	7.44	Thompson 2003
Poliovirus type 1	Virus	dsRNA	125	250	W	Wat	0.0248	7.44	Oppenheimer 1997
Poliovirus type 1	Virus	dsRNA	224	447	W	Wat	0.0248	7.44	Nuanualsuw an 2003
Poliovirus type 1	Virus	dsRNA	240	480	W	Wat	0.0248	7.44	Nuanualsuw an 2002
Poliovirus type 1	Virus	dsRNA	111	221	W	Wat	0.0248	7.44	Hill 1970
Poliovirus type 1	Virus	dsRNA	77	154	W	Wat	0.0248	7.44	Harris 1987
Poliovirus type 1	Virus	dsRNA	80	160	W	Wat	0.0248	7.44	Gerba 2002
Poliovirus type 1	Virus	dsRNA	83	167	W	Wat	0.0248	7.44	Simonet 2006
Poliovirus type 1	Virus	dsRNA	57	115	W	Wat	0.0248	7.44	Tree 2005
Poliovirus type 2	Virus	dsRNA	121	241	W	Wat	0.0248	7.44	Hill 1970
Poliovirus type 3	Virus	dsRNA	103	206	W	Wat	0.0248	7.44	Hill 1970
Polyomavirus	Virus	dsDNA	480	959	W	Wat	0.0424	5	vander Eb 1967
Polyomavirus	Virus	dsDNA	640	1279	W	Wat	0.0424	5	Defendi 1967
Polyomavirus	Virus	dsDNA	696	1391	W	Wat	0.0424	5	Rauth 1965
Polyomavirus	Virus	dsDNA	501	1001	W	Wat	0.0424	5	Latarjet 1967
Polyomavirus (ssDNA)	Virus	ssDNA	120	240	W	Wat	0.045	5	vander Eb 1967
Porcine Parvovirus (PPV)	Virus	ssDNA	23	45	W	Wat	0.021	6.194	Wang 2004
Pseudorabies (PRV)	Virus	dsDNA	34	68	W	Wat	0.194	-	Ross 1971
Rabies virus (env)	Virus	ssRNA	10	21	W	Wat	0.07	11.932	Weiss 1986
Rauscher Murine Leukemia v.	Virus	ssRNA	157	313	W	Wat	0.094	8.282	Kelloff 1970
Rauscher Murine Leukemia v.	Virus	ssRNA	480	959	W	Wat	0.094	8.282	Lovinger 1975
Rauscher Murine Leukemia v.	Virus	ssRNA	959	1919	S	-	0.094	8.282	Stull 1976
Reovirus	Virus	dsRNA	175	350	W	Wat	0.075	11	Hill 1970
Reovirus	Virus	dsRNA	186	371	W	Wat	0.075	11	Wang 2004
Reovirus	Virus	dsRNA	69	137	W	Wat	0.075	11	vonBrodorotti 1982
Reovirus	Virus	dsRNA	245	490	W	Wat	0.075	11	Shaw 1973
Reovirus	Virus	dsRNA	121	241	W	Wat	0.075	11	Rauth 1965
Reovirus	Virus	dsRNA	270	540	W	Wat	0.075	11	McClain 1966
Reovirus	Virus	dsRNA	174	349	W	Wat	0.075	11	Hill 1970
Reovirus type 1	Virus	dsRNA	153	305	W	Wat	0.075	11	Harris 1987
Reovirus 3	Virus	dsRNA	334	667	W	Wat	0.075	11	Zavadova 1975

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Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Rotavirus	Virus	dsRNA	200	400	W	Wat	0.07	-	Caballero 2004
Rotavirus SA11	Virus	dsRNA	89	177	W	Wat	0.07	-	Wilson 1992
Rotavirus SA11	Virus	dsRNA	75	150	W	Wat	0.07	-	Meng 1987
Rotavirus SA11	Virus	dsRNA	105	210	W	Wat	0.07	-	Battigelli 1993
Rotavirus SA11	Virus	dsRNA	100	200	W	Wat	0.07	-	Chang 1985
Rotavirus SA11	Virus	dsRNA	84	168	W	Wat	0.07	-	Sommer 1989
Rous Sarcoma virus (RSV)	Virus	ssRNA	720	1439	W	Wat	0.127	9.392	Levinson 1966
Rous Sarcoma virus (RSV)	Virus	ssRNA	240	480	W	Wat	0.127	9.392	Golde 1961
Rous Sarcoma virus (RSV)	Virus	ssRNA	200	400	S	-	0.127	9.392	Rubin 1959
Semliki forest virus	Virus	ssRNA	25	50	W	Wat	0.061	11.442	Weiss 1986
Simian virus 40	Virus	dsDNA	2503	5006	W	Wat	0.045	5.243	Bourre 1989
Simian virus 40	Virus	dsDNA	1599	3198	W	Wat	0.045	5.243	Seemayer 1973
Simian virus 40	Virus	dsDNA	1439	2878	W	Wat	0.045	5.243	Cornellis 1981
Simian virus 40	Virus	dsDNA	1245	2489	W	Wat	0.045	5.243	Bockstahler 1977
Simian virus 40	Virus	dsDNA	886	1771	W	Wat	0.045	5.243	Defendi 1967
Simian virus 40	Virus	dsDNA	650	1301	W	Wat	0.045	5.243	Sarasin 1978
Simian virus 40	Virus	dsDNA	443	886	W	Wat	0.045	5.243	Aaronson 1970
Simian virus 40	Virus	dsDNA	23	46	W	Wat	0.045	5.243	Cornellis 1982
Simian virus 40	Virus	dsDNA	17	35	W	Wat	0.045	5.243	Wang 2004
Sindbis virus	Virus	ssRNA	22	44	Air	62	0.075	11.703	Jensen 1964
Sindbis virus	Virus	ssRNA	60	119	W	Wat	0.075	11.703	vonBrodorotti 1982
Sindbis virus	Virus	ssRNA	113	227	W	Wat	0.075	11.703	Wang 2004
Sindbis virus	Virus	ssRNA	50	100	W	Wat	0.075	11.703	Zavadova 1975
Vaccinia virus	Virus	dsDNA	1	2	Air	60	0.307	195.815	McDevitt 2007
Vaccinia virus	Virus	dsDNA	15	30	Air	65	0.307	195.815	Jensen 1964
Vaccinia virus	Virus	dsDNA	7	13	W	Wat	0.307	195.815	Galasso 1965
Vaccinia virus	Virus	dsDNA	14	28	W	Wat	0.307	195.815	Bossart 1978
Vaccinia virus	Virus	dsDNA	14	29	W	Wat	0.307	195.815	Ross 1971
Vaccinia virus	Virus	dsDNA	18	36	W	Wat	0.307	195.815	Klein 1994
Vaccinia virus	Virus	dsDNA	22	44	W	Wat	0.307	195.815	Zavadova 1971
Vaccinia virus	Virus	dsDNA	28	56	W	Wat	0.307	195.815	Rauth 1965
Vaccinia virus	Virus	dsDNA	715	1430	W	Wat	0.307	195.815	Davidovich 1991
Vaccinia virus	Virus	dsDNA	677	1354	W	Wat	0.307	195.815	Collier 1955
VEE	Virus	ssRNA	55	110	W	Wat	0.065	11.444	Smirnov 1992
Vesicular Stomatitis virus	Virus	ssRNA	13	25	W	Wat	0.104	11.161	Rauth 1965
Vesicular Stomatitis virus	Virus	ssRNA	12	24	W	Wat	0.104	11.161	Helentjaris 1977
Vesicular Stomatitis virus	Virus	ssRNA	100	200	W	Wat	0.104	11.161	Bay 1979
Vesicular Stomatitis virus	Virus	ssRNA	6	12	W	Wat	0.104	11.161	Shimizu 2004
WEE	Virus	ssRNA	54	107	W	Wat	0.07	11.484	Dubin 1975
NOTES for Appendix A			Type: Sp = Spore, Veg = Vegetative, VegY = Vegetative yeast D <sub>90</sub> : UV Dose for 90% inactivation (10% survival) D <sub>99</sub> : UV Dose for 99% inactivation (1% survival) Media: A = Air, S = Surface, W = Water RH = Relative Humidity Dia.: Logmean diameter in microns, including envelope for viruses if any MP: Medium Pressure UV lamp, LP: Low Pressure UV lamp						

## APPENDIX A: UV Rate Constants for Pathogens and Allergens

Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Aspergillus amstelodami	Fungi	Sp	700	1400	W	Wat	3.354	35900	Jepson 1973
Aspergillus amstelodami	Fungi	Sp	258	516	S	-	3.354	35900	Luckiesh 1949
Aspergillus amstelodami	Fungi	Sp	669	1339	Air	67	3.354	35900	Luckiesh 1949
Aspergillus flavus	Fungi	Sp	349	698	S	-	4.24	35900	Green 2004
Aspergillus flavus	Fungi	Sp	600	1200	-	-	4.24	35900	Nagy 1964
Aspergillus flavus	Fungi	Sp	853	1706	W	Wat	4.24	35900	Begum 2009
Aspergillus fumigatus	Fungi	Sp	535	1071	S	-	4.24	35900	Green 2004
Aspergillus fumigatus	Fungi	Veg	560	1120	S	-	24.5	35900	Chick 1963
Aspergillus fumigatus	Fungi	Sp	2240	4480	S	-	2.64	35900	Chick 1963
Aspergillus glaucus	Fungi	Sp	440	880	-	-	3.354	35900	Nagy 1964
Aspergillus niger	Fungi	Sp	1771	3542	S	Lo RH	3.354	35900	Zahl 1939
Aspergillus niger	Fungi	Sp	1439	2878	S	-	3.354	35900	Fulton 1929
Aspergillus niger	Fungi	Veg	4480	8960	S	-	3.354	35900	Chick 1963
Aspergillus niger	Fungi	Sp	1000	2000	W	Wat	3.354	35900	Jepson 1973
Aspergillus niger	Fungi	Sp	315	1316	S	-	3.354	35900	Kowalski 2001
Aspergillus niger	Fungi	Sp	1387	2774	S	-	3.354	35900	Luckiesh 1949
Aspergillus niger	Fungi	Sp	750	1193	S	-	3.354	35900	Gritz 1990
Aspergillus niger	Fungi	Sp	4480	8960	S	-	3.354	35900	Chick 1963
Aspergillus niger	Fungi	Sp	3984	7967	Air	55	3.354	35900	Luckiesh 1949
Aspergillus niger	Fungi	Sp	1320	2640	-	-	3.354	35900	Nagy 1964
Aspergillus niger	Fungi	Sp	1681	3361	W	Wat	3.354	35900	Begum 2009
Aspergillus versicolor	Fungi	Sp	384	768	Air	85	3.354	35900	vanOsdell 2002
Aspergillus versicolor	Fungi	Sp	768	1535	Air	55	3.354	35900	vanOsdell 2002
Aspergillus versicolor	Fungi	Sp	139	277	Air	50	3.354	35900	vanOsdell 2002
Aspergillus versicolor	Fungi	Veg	96	192	Air	(Lo RH)	3.354	35900	Nakamura 1987
Blastomyces dermatitidis	Fungi	VegY	140	280	S	-	11.000	23000	Chick 1963
Botrytis cinerea	Fungi	Sp	250	501	S	-	11.180	42660	Marquenie 2002
Candida albicans	Fungi	VegY	230	419	W	Wat	4.899	20000	Dolman 1989
Candida albicans	Fungi	VegY	447	894	W	Wat	4.899	20000	Abshire 1981
Candida albicans	Fungi	VegY	750	1131	S	-	4.899	20000	Gritz 1990
Candida albicans	Fungi	VegY	280	560	S	-	4.899	20000	Chick 1963
Candida parapsilosis	Fungi	VegY	98	195	W	Wat	-	-	Severin 1983
Cladosporium herbarum	Fungi	Sp	500	100	W	Wat	8.062	36000	Jepson 1973
Cladosporium herbarum	Fungi	Sp	189	377	S	-	8.062	36000	Luckiesh 1949
Cladosporium herbarum	Fungi	Sp	622	1245	Air	53	8.062	36000	Luckiesh 1949
Cladosporium trichoides	Fungi	Veg	560	1120	S	-	8.062	36000	Chick 1963
Cladosporium trichoides	Fungi	Sp	1120	2240	S	-	8.062	36000	Chick 1963
C. sphaerospermum	Fungi	Sp	1439	2193	Air	50	8.062	36000	vanOsdell 2002
Cladosporium werneckii	Fungi	Sp	4480	8960	S	-	8.062	36000	Chick 1963
Cladosporium werneckii	Fungi	Veg	560	1120	S	-	8.062	36000	Chick 1963
Cryptococcus neoformans	Fungi	Sp	138	276	S	-	4.899	23000	Wang 1994
Cryptococcus neoformans	Fungi	VegY	280	560	S	-	4.899	23000	Chick 1963
Curvularia lunata	Fungi	Veg	560	1120	S	-	17.100	29700	Chick 1963
Eurotium rubrum	Fungi	Sp	434	867	W	Wat	5.612	-	Begum 2009
Fusarium oxysporum	Fungi	Sp	260	324	W	Wat	11.225	43000	Asthana 1992
Fusarium solani	Fungi	Sp	313	627	W	Wat	11.225	43000	Asthana 1992
Fusarium spp.	Fungi	Sp	560	1120	S	-	11.225	43000	Chick 1963
Fusarium spp.	Fungi	Veg	1120	2240	S	-	34.300	43000	Chick 1963
Histoplasma capsulatum	Fungi	Veg	140	280	S	-	2.550	23000	Chick 1963
Monilinia fructigena	Fungi	Sp	167	334	S	-	10.300	-	Marquenie 2002
Mucor mucedo	Fungi	Sp	600	1200	W	Wat	7.071	39000	Jepson 1973
Mucor mucedo	Fungi	Sp	180	360	S	-	7.071	39000	Luckiesh 1949
Mucor mucedo	Fungi	Sp	577	1154	Air	63	7.071	39000	Luckiesh 1949
Mucor racemosus	Fungi	Sp	170	340	-	-	7.071	39000	Nagy 1964
Mucor spp.	Fungi	Sp	140	280	S	-	7.071	39000	Chick 1963
Mucor spp.	Fungi	Veg	280	560	S	-	31.600	39000	Chick 1963
Penicillium chrysogenum	Fungi	Sp	400	800	W	Wat	3.262	34000	Jepson 1973
Penicillium chrysogenum	Fungi	Sp	148	295	S	-	3.262	34000	Luckiesh 1949
Penicillium chrysogenum	Fungi	Sp	1645	2558	Air	50	3.262	34000	vanOsdell 2002
Penicillium chrysogenum	Fungi	Sp	531	1061	Air	41	3.262	34000	Luckiesh 1949
Penicillium corylophilum	Fungi	Sp	381	762	W	Wat	3.262	34000	Begum 2009
Penicillium digitatum	Fungi	Sp	321	641	W	Wat	3.262	34000	Asthana 1992

## APPENDIX A: UV Rate Constants for Pathogens and Allergens

Microbe	Group	Type	D <sub>90</sub> J/m <sup>2</sup>	D <sub>99</sub> J/m <sup>2</sup>	Media	RH %	Dia. μm	Base Pairs kb	Source
Penicillium digitatum	Fungi	Sp	440	880	-	-	3.262	34000	Nagy 1964
Penicillium expansum	Fungi	Sp	130	260	-	-	3.262	34000	Nagy 1964
Penicillium italicum	Fungi	Sp	321	404	W	Wat	3.262	34000	Asthana 1992
Penicillium roquefortii	Fungi	Sp	130	260	-	-	3.262	34000	Nagy 1964
Penicillium spp.	Fungi	Sp	2240	4480	S	-	3.262	34000	Chick 1963
Penicillium spp.	Fungi	Veg	280	560	S	-	8.800	34000	Chick 1963
Rhizopus nigricans	Fungi	Sp	3000	6000	W	Wat	6.928	54178	Jepson 1973
Rhizopus nigricans	Fungi	Sp	267	535	Air	62	6.928	54178	Luckiesh 1949
Rhizopus nigricans	Fungi	Sp	1110	2220	-	-	6.928	54178	Nagy 1964
Rhizopus nigricans	Fungi	Sp	173	346	S	-	6.928	54178	Kow alski 2001
Rhizopus oryzae	Fungi	Sp	4480	8960	S	-	6.928	-	Chick 1963
Rhodotorula spp.	Fungi	VegY	1120	2240	S	-	5.900	-	Chick 1963
Saccharomyces spp.	Fungi	VegY	44	88	-	-	-	-	Nagy 1964
Saccharomyces ellipsoideus	Fungi	VegY	33	66	-	-	-	-	Nagy 1964
Scopulariopsis brevicaulis	Fungi	Sp	650	250	W	Wat	5.916	-	Jepson 1973
Scopulariopsis brevicaulis	Fungi	Sp	226	451	S	-	5.916	-	Luckiesh 1949
Scopulariopsis brevicaulis	Fungi	Sp	2890	1339	Air	79	5.916	-	Luckiesh 1949
Sporotrichum schenkii	Fungi	VegY	280	560	S	-	5.500	-	Chick 1963
Stachybotrys chartarum	Fungi	Sp	5575	11151	S	-	5.623	-	Green 2005
Torula bergeri	Fungi	Veg	4480	8960	S	-	40	-	Chick 1963
Torula sphaerica	Fungi	VegY	23	46	Air	65	40	-	Luckiesh 1949
Torula sphaerica	Fungi	VegY	78	157	S	-	40	-	Luckiesh 1949
Trichophyton rubrum	Fungi	Veg	560	1120	S	-	4.899	-	Chick 1963
Trichophyton rubrum	Fungi	Sp	560	1120	S	-	4.899	-	Chick 1963
Ustilago zeae	Fungi	VegY	1120	2240	S	-	5.916	20500	Chick 1963
Ustilago zeae	Fungi	Sp	35	70	-	-	5.916	20500	Sussman 1966
Yeast	Fungi	VegY	40	80	W	Wat	-	-	Jepson 1973
Acanthameoba	Rhizopod	-	999	219	W	Wat	-	-	Maya 2003
Acanthameoba castellani	Rhizopod	-	992	1985	S	-	-	-	Gritz 1990
Algae	-	-	1000	2000	-	-	-	-	Summer 1962
Algae, blue-green	Algae	-	450	900	-	-	-	-	Jepson 1973
Cryptosporidium hominis	Protoz	-	30	59	-	-	-	-	Johnson 2005
Cryptosporidium parvum	Protoz	-	7	15	W	Wat	3	-	Oguma 2001
Cryptosporidium parvum	Protoz	-	20	40	W	Wat	3	-	Zimmer 2003
Cryptosporidium parvum	Protoz	-	10	20	W	Wat	3	-	Shin 2001
Cryptosporidium parvum	Protoz	-	50	100	W	Wat	3	-	Craik 2001
Cryptosporidium parvum	Protoz	-	10	20	W	Wat	3	-	Bukhari 2004
Cryptosporidium parvum	Protoz	-	5	10	W	Wat	3	-	Morita 2002
Encephalitozoon intestinalis	Protoz	-	29	59	W	Wat	-	-	.....
Encephalitozoon intestinalis	Protoz	-	15	30	W	Wat	-	-	Huffman 2002
Encephalitozoon cuniculi	Protoz	-	43	87	W	Wat	-	-	Marshall 2003
Encephalitozoon hellem	Protoz	-	80	160	W	Wat	-	-	Marshall 2003
Giardia lamblia cysts	Protoz	-	50	100	W	Wat	-	-	Campbell 2002
Giardia lamblia cysts	Protoz	-	3	5	W	Wat	-	-	Shin 2005
Giardia lamblia cysts	Protoz	-	20	40	W	Wat	-	-	Li 2007
Giardia muris cysts	Protoz	-	10	20	W	Wat	-	-	Craik 2001
Giardia muris cysts	Protoz	-	7	13	W	Wat	-	-	Hayes 2000
Protozoa	Protoz	-	80	160	-	-	-	-	Jepson 1973
Protozoa	Protoz	-	240	480	-	-	-	-	Summer 1962
Prions (scrapie)	Prion	-	24315	48629	-	-	-	-	Bellinger-Kawahara 1987
Prions (scrapie)	Prion	-	55618	111236	-	-	-	-	Alper 1967



### Appendix B: Common Indoor Bacteria

PATHOGEN	GROUP	TYPE	DISEASE GROUP	BIOSAFETY LEVEL
Acinetobacter	Bacteria	Gram-	Endogenous	Risk Group 2
Actinomyces israelii	Bacteria	Gram+	Endogenous	Risk Group 2
Aeromonas	Bacteria	Gram-	Non-communicable	Risk Group 2
Alcaligenes	Bacteria	Gram-	Endogenous	Risk Group 2
Bacteroides fragilis	Bacteria	Gram-	Endogenous	Risk Group 2
Bordetella pertussis	Bacteria	Gram-	Communicable	Risk Group 2
Brucella	Bacteria	Gram-	Non-communicable	Risk Group 2-3
Burkholderia cepacia	Bacteria	Gram-	Non-communicable	Risk Group 1
Burkholderia mallei	Bacteria	Gram-	Non-communicable	Risk Group 3
Burkholderia pseudomallei	Bacteria	Gram-	Non-communicable	Risk Group 2-3
Cardiobacterium	Bacteria	Gram-	Endogenous	Risk Group 2
Chlamydia pneumoniae	Bacteria	Gram-	Communicable	Risk Group 2
Chlamydophila psittaci	Bacteria	Gram-	Non-communicable	Risk Group 2-3
Clostridium botulinum	Bacteria	Gram+	Non-communicable	Risk Group 2-4
Clostridium perfringens	Bacteria	Gram+	Non-communicable	Risk Group 2
Corynebacterium diphtheriae	Bacteria	Gram+	Communicable	Risk Group 2
Enterobacter cloacae	Bacteria	Gram-	Endogenous	Risk Group 1
Enterococcus	Bacteria	Gram+	Non-communicable	Risk Group 1-2
Enterococcus faecalis	Bacteria	Gram+	Endogenous	Risk Group 1
Francisella tularensis	Bacteria	Gram-	Non-communicable	Risk Group 2-3
Haemophilus influenzae	Bacteria	Gram-	Communicable	Risk Group 2
Haemophilus parainfluenzae	Bacteria	Gram-	Endogenous	Risk Group 2
Klebsiella pneumoniae	Bacteria	Gram-	Endogenous	Risk Group 2
Legionella pneumophila	Bacteria	Gram-	Non-communicable	Risk Group 2
Listeria monocytogenes	Bacteria	Gram+	Non-communicable	
Moraxella	Bacteria	Gram-	Endogenous	Risk Group 2
Mycobacterium avium	Bacteria	Gram+	Non-communicable	Risk Group 3
Mycobacterium kansasii	Bacteria	Gram+	Non-communicable	Risk Group 2
Mycobacterium tuberculosis	Bacteria	Gram+ (acid fast)	Communicable	Risk Group 2-3
Mycoplasma pneumoniae	Bacteria	no wall	Endogenous	Risk Group 2
Neisseria meningitidis	Bacteria	Gram-	Endogenous	Risk Group 2
Proteus mirabilis	Bacteria	Gram-	Endogenous	Risk Group 2
Pseudomonas aeruginosa	Bacteria	Gram-	Non-communicable	Risk Group 1
Rickettsia prowazeki	Bacteria	Gram-	Vector-borne	Risk Group 2-3
Rickettsia rickettsii	Bacteria	Gram-	Vector-borne	Risk Group 2-3
Salmonella typhi	Bacteria	Gram-	Food-borne	Risk Group 2
Serratia marcescens	Bacteria	Gram-	Endogenous	Risk Group 1
Shigella	Bacteria	Gram-	Food-borne	Risk Group 2
Staphylococcus aureus	Bacteria	Gram+	Endogenous	Risk Group 2
Staphylococcus epidermidis	Bacteria	Gram+	Endogenous	Risk Group 1
Streptococcus pneumoniae	Bacteria	Gram+	Communicable	Risk Group 2
Streptococcus pyogenes	Bacteria	Gram+	Communicable	Risk Group 2
Vibrio cholerae	Bacteria	Gram-	Food-borne	Risk Group 2
Yersinia pestis	Bacteria	Gram-	Communicable	Risk Group 2-3
Coxiella burnetii	Bacteria / Rickettsiae	Gram-	Non-communicable	Risk Group 2-3
Bacillus anthracis	Bacterial Spore	Gram+	Non-communicable	Risk Group 2-3
Micromonospora faeni	Bacterial Spore	Micromonosporaceae	Non-communicable	-
Nocardia asteroides	Bacterial Spore	Nocardiaceae	Non-communicable	Risk Group 2
Nocardia brasiliensis	Bacterial Spore	Nocardiaceae	Non-communicable	Risk Group 2
Saccharopolyspora rectivirgula	Bacterial Spore	Micromonosporaceae	Non-communicable	Risk Group 2
Thermoactinomyces sacchari	Bacterial Spore	Micromonosporaceae	Non-communicable	Risk Group 2
Thermoactinomyces vulgaris	Bacterial Spore	Micromonosporaceae	Non-communicable	Risk Group 1
Thermomonospora viridis	Bacterial Spore	Micromonosporaceae	Non-communicable	Risk Group 1

### Appendix C: Common Indoor Fungi

PATHOGEN	GROUP	PHYLUM	DISEASE GROUP	BIOSAFETY LEVEL
Absidia	Fungal Spore	Zygomycetes	Non-communicable	Risk Group 2
Acremonium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1-2
Alternaria alternata	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Arthrinium phaeospermum	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Aspergillus	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Aureobasidium pullulans	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Blastomyces dermatitidis	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2-3
Botrytis cinerea	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 3
Candida	Fungal Spore	Hyphomycetes	Endogenous	Risk Group 1
Chaetomium globosum	Fungal Spore	Ascomycetes	Non-communicable	Risk Group 1
Cladosporium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Coccidioides immitis	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 3
Cryptosporidium parvum	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Curvularia	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Drechslera	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Emmericella nidulans	Fungal Spore	Ascomycetes	Non-communicable	Risk Group 1
Epicoccum nigrum	Fungal Spore	Ascomycetes	Non-communicable	Risk Group 1
Eurotium	Fungal Spore	Ascomycetes	Non-communicable	Risk Group 1
Exophiala	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Fusarium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Helminthosporium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Histoplasma capsulatum	Fungal Spore	Ascomycetes	Non-communicable	Risk Group 3
Mucor plumbeus	Fungal Spore	Zygomycetes	Non-communicable	Risk Group 1
Paecilomyces variotii	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Paracoccidioides	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Penicillium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Phialophora	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Phoma	Fungal Spore	Coelomycetes	Non-communicable	Risk Group 1
Pneumocystis carinii	Fungal Spore	Protozoal	Communicable	Risk Group 1
Rhizomucor pusillus	Fungal Spore	Zygomycetes	Non-communicable	Risk Group 1
Rhizopus stolonifer	Fungal Spore	Zygomycetes	Non-communicable	Risk Group 2
Rhodoturula	Fungal Spore	Blastomycetes	Non-communicable	-
Scopulariopsis	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Sporothrix schenckii	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 2
Stachybotrys chartarum	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1-2
Trichoderma	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Trichophyton	Fungal Spore	Hyphomycetes	Non-communicable	
Ulocladium	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Ustilago	Fungal Spore	Basidiomycetes	Non-communicable	Risk Group 1
Verticillium	Fungal Spore	Hyphomycetes	Non-communicable	
Wallemia sebi	Fungal Spore	Hyphomycetes	Non-communicable	Risk Group 1
Cryptococcus neoformans	Fungal Yeast	Hyphomycetes	Non-communicable	Risk Group 2
Trichosporon cutaneum	Fungi/Yeast	Basidiomycetes	Non-communicable	

**Appendix D: Health Care Facility Cost Estimates -- Part 1**

Category	Facility 1	Facility 2	Facility 3	Facility 4	Facility 5	Facility 6
General Facility						
Age	21 years	13 years	31 years	40 years	63 years	27 years
Area	193,200 SF	353,000 SF	294,619 SF	134,988 SF	1,000,000 SF	364,400 SF
No. Licensed Beds	149	247	228	108	345	401
Conditions	Good	Good	Poor	Adequate	Poor	Fair
Inpatient						
No. Staffed Beds	172	187	204	74	303	340
Private	38	110	0	10	0	0
Semi-private	84	57	0	44	0	401
Ward	7	7pr.+ 13 NICU	204	20	345	0
Total Admissions	6,864	7,890	6,088	2,637	12,154	21,246
Inpatient Days	30,880	44,513	31,553	15,287	87,354	99,503
Average Daily Census	83	122	149	39	239	273
Percent Occupancy	57	65	73	52	79	80
Routine Beds	108	131	108	54	176	331
Special Care	22	56	22	10	169	46
Nursery	26	44	26	0	0	42
Outpatient						
No. of Exam Rooms	57	124	12	38	190	62
Clinic Visits	110,232	119,399	118,234	61,181	257,795	98,897
Visits per exam room	1,934	1,592	9,852	1,610	1,357	1,595
List of Clinics	13	7	12	23	13	2
ER Visits	57,390	41,822	76,875	42,287	95,951	81,890
ER Treatment Rooms	16	7	26	10	80	20
Facility Needs	Space	Space	Space	Space	Space	Space, toilets
Area	13,205 SF	36,000 SF	51,000 SF	6,715 SF	53,540 SF	
Visits per Room	3,587	5,975	2,956	4,229	1,194	4,095
Clinical						
No. Operating Rooms	4	10	5	4	16	12
O.R. Cases	2,424	3,068	4,780	2,202	11,189	8,959
Cases per O.R.	606	307	956	550	699	747
No. Delivery Rooms	3	3	3	0	0	9
Live Births	948	1,638	1,603	0	0	3,579
Births per Room	316	546	534	0	0	398
No. X-Ray Rooms	9	13	8	6	37	12
X-Ray Equipment	See attached	See attached	See attached	See attached	See attached	See attached
Estimated # of air handlers	23	23	17	23	200	17
Facility Adequacy	Adequate	Adequate	Inadequate	Adequate	Inadequate	Adequate
Major Needs	Labor & Delivery, NICU		Labor & Delivery, NICU, MRI	ER, Clinic, Parking, Storage	New Facility	Space, Privacy, CT Scanner

## Appendix D: Health Care Facility Cost Estimates -- Part 2

Category	Facility 1	Facility 2	Facility 3	Facility 4	Facility 5	Facility 6	TOTALS
<b>ESTIMATED COSTS</b>							
Coil Irradiation	\$ 38,318.00	\$ 63,864.00	\$ 54,603.00	\$ 34,486.00	\$ 178,819.00	\$ 64,928.00	\$ 435,018.00
Duct Treatment	\$ 30,959.00	\$ 56,565.00	\$ 47,210.00	\$ 21,631.00	\$ 160,242.00	\$ 58,392.00	\$ 374,999.00
Operating Room	\$ 56,000.00	\$ 140,000.00	\$ 70,000.00	\$ 56,000.00	\$ 224,000.00	\$ 168,000.00	\$ 714,000.00
TOTAL COSTS	\$ 125,277.00	\$ 260,429.00	\$ 171,813.00	\$ 112,117.00	\$ 563,061.00	\$ 291,320.00	\$ 1,524,017.00
Annual Cost	\$ 20,044.32	\$ 41,668.64	\$ 27,490.08	\$ 17,938.72	\$ 90,089.76	\$ 46,611.20	\$ 243,842.72
ANNUAL MAINTENANCE							
ESTIMATED SAVINGS							
PATIENTS							
Nosocomial Infections	\$ 154,400.00	\$ 222,565.00	\$ 157,765.00	\$ 76,435.00	\$ 436,770.00	\$ 497,515.00	\$ 1,545,450.00
Operating Infections	\$ 12,120.00	\$ 15,340.00	\$ 23,900.00	\$ 11,010.00	\$ 55,945.00	\$ 44,795.00	\$ 163,110.00
Clinic Infections	\$ 220,464.00	\$ 238,798.00	\$ 236,468.00	\$ 122,362.00	\$ 515,590.00	\$ 197,794.00	\$ 1,531,476.00
STAFF							\$ -
Nosocomial Infections	3% reduction*	3% reduction*	3% reduction*	3% reduction*	3% reduction*	3% reduction*	\$ -
Health Care	20% reduction*	20% reduction*	20% reduction*	20% reduction*	20% reduction*	20% reduction*	\$ -
Illness Absentee rate	20% reduction*	20% reduction*	20% reduction*	20% reduction*	20% reduction*	20% reduction*	\$ -
COIL & DUCT CLEANING	\$ 1,725.00	\$ 1,725.00	\$ 1,275.00	\$ 1,725.00	\$ 15,000.00	\$ 1,275.00	\$ 22,725.00
ENERGY SAVINGS	10 - 14%*	10 - 14%*	10 - 14%*	10 - 14%*	10 - 14%*	10 - 14%*	\$ -
EXTENDED LIFE OF UNITS	10%*	10%*	10%*	10%*	10%*	10%*	\$ -
							\$ -
TOTAL SAVINGS YEAR 1	\$ 388,709.00	\$ 478,428.00	\$ 419,408.00	\$ 211,532.00	\$ 1,023,305.00	\$ 741,379.00	\$ 3,262,761.00
TOTAL SAVINGS YEAR 2	\$ 388,709.00	\$ 478,428.00	\$ 419,408.00	\$ 211,532.00	\$ 1,023,305.00	\$ 741,379.00	\$ 1,708,560.00
TOTAL SAVINGS YEAR 3	\$ 388,709.00	\$ 478,428.00	\$ 419,408.00	\$ 211,532.00	\$ 1,023,305.00	\$ 741,379.00	\$ 1,708,560.00
LIFETIME SAVINGS 10 YRS	\$ 3,887,090.00	\$4,784,280.00	\$4,194,080.00	\$2,115,320.00	\$10,233,050.00	\$ 7,413,790.00	\$32,627,610.00

Note: Above estimates are based on SSI rate of 3% (NNIS 2000) and an assumed nosocomial infection rate of 0.5% for airborne pathogens.

\*(Savings may vary based on local utility rates, age of equipment, and actual measured before and after results).

### **APPENDIX E: Reports from articles and new sources on UVC effectiveness for improving IAQ and realizing savings from reduced energy and maintenance costs**

A recent laboratory study has shown that ultraviolet (UV) light can effectively kill the Severe Acute Respiratory Syndrome (SARS) virus, according to FP Technologies. The company, an engineering firm that uses UV radiation to sterilize air and surfaces, designed the SARS testing. Tests were performed at ZeptoMetrix Inc., a biotechnology lab.<sup>1</sup>

Florida Hospital has been installing high-output ultraviolet C-band (UVC) lights in its air-handling units (AHUs), and found that this has reduced or, in some cases, eliminated coil-cleaning programs. The lights also offer IAQ and infection control benefits.

The air handler was essentially returned to its original performance specifications. The coil and drain pan areas have maintained their clean condition, and eliminating the necessity for routine cleaning.<sup>2</sup>

Exposing cooling coils to UVC will eventually kill all mold, and keep the drain pan clean, and keeping the coil clean will increase equipment efficiency, up to the design rating of the equipment."<sup>3</sup>

PSO installed UV lights in 1996-97 to eliminate a persistent mold and IAQ problem in the majority of its HVAC systems. The firm found that, by bathing the coil and drain pan areas from the downstream side was able to eradicate the microbial growth and its related problems. The lights eliminated most of the customary coil cleaning maintenance. It translated into a big energy consumption reduction."<sup>4</sup>

Placing a UV light close to the air conditioning coil can prevent microbes from breeding in this typically moist area, keeping the coil clean and preventing that yeasty odor that accompanies the growth of these microorganisms.<sup>5</sup>

UVC light can penetrate the cellular wall of a microbe and damage DNA. UVC renders bacteria and spores unable to spread. Application of UVC at a distance of 12 inches for 15 minutes resulted in a 74 percent spore count reduction as compared to the control sample.<sup>6</sup>

UVC lights can significantly reduce annual cleaning of evaporator coils and condenser coils and can significantly minimize the maintenance staff's exposure to a variety of chemicals.<sup>7</sup>

Sickness among office workers in industrialized countries could be reduced by using ultraviolet lamps to kill germs in ventilation systems. Ultraviolet germicidal irradiation, or UVGI, is sometimes used in hospital ventilation systems to disinfect the air but is rarely incorporated into office or other building ducts. In a study published this week in The Lancet medical journal, Canadian scientists found that the technique reduced overall worker sickness by about 20%, including a 40% drop in breathing problems. The cost of UVGI installations could prove cost-effective compared with the yearly losses from absenteeism.<sup>8</sup>

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In one example, after a few weeks of UVC operation, static pressure over the coil decreased from 1.8 in.wg to just 0.7 in. wg. Air velocity over the coil more than doubled, from 230 fpm to 520 fpm. The coil and drain pan areas had no visible evidence of mold. The air exit wet bulb temperature decreased significantly, from 57° F (before UVC) to 53° (with UVC). It was estimated a total of \$4,867 in savings accrued for this one unit. This hospital is saving approximately 15% in HVAC energy costs. Results from this and other studies indicate that just a one-micron buildup of dirt or debris on coil surfaces can lead to a 15% loss in efficiency.

The ability of UVC to inactivate all types of bacteria and viruses is well documented. And, by destroying microbes trapped on cooling coils or in air filters, UVC light may increase the service life of these components and may facilitate safer changeout.<sup>9</sup>

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<sup>1</sup> Testing Shows UV Effective At Killing SARS Virus /08/16/2004

<sup>2</sup> Retrofits Boost IAQ, Save Energy /Barb Checket-Hanks / Service, Maintenance and Troubleshooting Editor /08/16/2004

<sup>3</sup> Mechanical Inspectors Learn From IAQ Pro /by John R. Hall / Business Management Editor /09/24/2004

<sup>4</sup> UV technology sheds light on IAQ problems. Ultraviolet lights clear the air, reduce costs. /07/01/2000

<sup>5</sup> Contractor puts UV lighting system to the test. /by Ed Bas / Special Editor. /08/03/2000

<sup>6</sup> UVC Light: A Tool For Fighting Airborne Contaminants. /by John R. Hall / Business Management Editor. /07/17/2002

<sup>7</sup> IAQ Where We Work, Shop, And Save. /by Barb Checket-Hanks / Service/Maintenance and Troubleshooting Editor. /03/14/2003

<sup>8</sup> UV Lamps could reduce worker sickness. LONDON (AP) /11/28/2003

<sup>9</sup> UVC: Florida Hospital Puts HVAC Maintenance Under A New Light. Firouz Keikavousi - a mechanical engineer in charge of facilities management for Florida Hospital. /02/24/2004