ENGINEERING THE CONTROL OF AIRBORNE PATHOGENS

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1.0 INTRODUCTION

The airborne route of transmission is important for a number of pathogenic microorganisms. Tuberculosis (TB) is a classic example of a disease which is transmitted by the airborne passage of pathogens. Primary pulmonary TB is caused by inhalation of droplet nuclei, carrying *Mycobacterium tuberculosis* (MTB), of less than 5 μm diameter. In the clinical setting microorganisms such as *Aspergillus* spp. are known to be transmitted by an airborne route. Indeed, it has been calculated that the airborne route of transmission accounts for 10% of all sporadic cases of nosocomial infection (i.e. infection originating in hospital). The airborne link in the 'chain of infection' associated with diseases such as TB and aspergillosis, is the weakest 'link', and the one which gives hospital engineers and health care authorities the best opportunity to break the chain. Through the use of well designed engineering systems it is possible to control the spread of airborne pathogens in hospital buildings. Conversely, the opposite is also true. Poorly designed engineering systems are often implicated in spread of airborne pathogens around buildings. Consequently, there is a need to raise the general awareness of available engineering control measures and to carry out research into the optimisation of these measures in healthcare facilities.

2.0 DROPLET NUCLEI

When a person sneezes or coughs many thousands of droplets are vigorously expelled into the atmosphere. The velocities involved are high. In the case of sneezing, the most violent expiratory process, initial velocities can be as high as 100 m/s (1). During sneezing most of the droplets are approximately 10 μm in diameter, although some may be in excess of 100 μm (1). The larger droplets fall to the ground, while evaporation of the smaller droplets takes place and they rapidly decrease in size to become droplet nuclei. This process is illustrated in Table 1 which shows the evaporation times for a variety of water droplets (2). It should be noted that the precise rate of evaporation is dependent on the vapour pressure in the air which is governed by its temperature and humidity.

Diameter of Droplet (μm)	**Evaporation Time (Seconds)**	**Distance that droplet will fall Before evaporation (m)**
200	5.2	6.51
100	1.3	0.42
50	0.31	0.0255
25	0.08	1.59×10^{-3}
12	0.02	8.4×10^{-5}

Table 1 Evaporating time of water droplets and falling distance before evaporation in air at 22°C and 50 % RH (2)

It can be seen from Table 1 that the evaporation time for small droplets is very short indeed. This is because they have an enormous surface area compared with their mass. Consequently, most of the droplets produced by a sneeze quickly evaporate to form droplet nuclei. If the droplet nuclei are produced by an infectious patient, then they will contain pathogenic microorganisms which will be dispersed into the atmosphere.

Droplet nuclei are so small that they settle slowly and remain suspended in air for a considerable period of time. This can be illustrated by applying Stokes's Law to a 2 μm droplet nuclei settling in air in a calm room, as follows:

$$\text{Terminal velocity, } U = \frac{\rho.g.d^2}{18.\eta} \times c_s \qquad (1)$$

where;

ρ	=	Density of droplet nuclei (e.g. 1000 kg/m^3)
g	=	Acceleration due to gravity (i.e. 9.81 m/s^2)
d	=	Diameter of droplet nuclei (e.g. 2×10^{-6} m)
η	=	Viscosity of air (i.e. 1.78×10^{-5} kg/ms)
c_s	=	Cunningham slip correction factor (e.g. 1.033)

Under the above data condition the terminal velocity of the falling droplet nuclei is 0.127 mm/s, which means that under calm conditions the particle would take approximately 4.4 hours to fall a distance of 2 m. Given this long suspension time, it is not difficult to envisage particles being carried long distances by convection currents. Depending on ventilation-associated factors, droplet nuclei can travel over long distances and thus can be distributed widely throughout hospital and other buildings. The chain of infection is therefore very much influenced by the ventilation conditions which exist in any particular clinical setting.

3.0 VENTILATION

Of all the possible engineering techniques that can be employed to control airborne pathogens, good ventilation is probably the most effective. Although most people are familiar with the general concept of ventilation, very few understand the principles on which it is based. Indeed, most confuse room air movement with ventilation. The two are different! Most mechanical 'ventilation' and air conditioning systems supply air, a large percentage of which has been recirculated (i.e. reused), with only a small proportion (e.g. 20 % to 30 %) being 'fresh' outside air. Recirculation of extracted air is usually adopted in order to save energy, since it is expensive to throw away air which has been heated or cooled. The term ventilation should therefore only be applied to the supply of outside air to the room space.

3.1 DILUTION VENTILATION

Most ventilation systems push large quantities of 'clean' outside air into occupied spaces so that any contaminants in the room space are diluted and flushed out to atmosphere. To function properly good mixing of the air in the room space is essential. As 'clean' ventilation air is introduced into a room space, the level of the contaminants in the space drops until eventually, a contaminant equilibrium level is reached, below which it is impossible to go without a further increase in the ventilation rate.

The contaminant equilibrium level depends on the volume flow rate of the ventilation air and on the rate at which contaminants are introduced into the room space. Assuming that the volume flow rate of the contaminants is very small in comparison with the ventilation air flow rate, it is possible to determine the contaminant equilibrium level using equation 2.

$$C_e = \frac{Q_c}{Q} \qquad (2)$$

where: C_e = Contaminant equilibrium level ($\mu g/m^3$)
Q_c = Rate at which contaminants are introduced into the room space per second (μg /s)

Equation 2 applies equally to viable and non-viable contaminants and a variety of units can be used for the contaminant equilibrium level (e.g. ppm, $\mu g/m^3$ and cfu/m^3). Equation 2 can be modified, so that the ventilation air rate is expressed in terms of air changes per hour (ACH).

$$C_e = \frac{Q_{ch}}{N_{vent} \times V} \qquad (3)$$

Where: Q_{ch} = Rate at which contaminants are introduced into the room space per hour (μg /h)
N_{vent} = Number of air changes per hour due to ventilation system (h^{-1})
V = Room Volume (m^3)

By applying a range of ventilation rates to equation 3, it is possible to plot the theoretical reduction in the contaminant equilibrium level that is achieved (as shown in Figure 1).

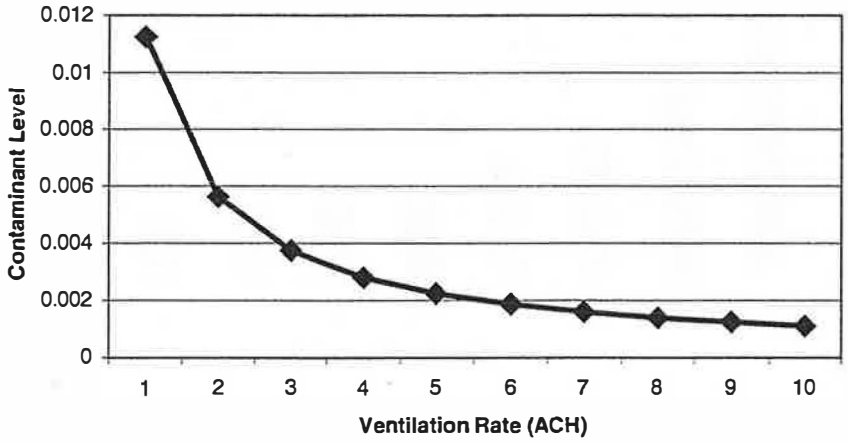

Figure 1 Effect of various ventilation rates on contaminant equilibrium level

It can be seen from Figure 1 that every time the ventilation rate is doubled, the contaminant equilibrium level decreases by 50%. Consequently, if very low contamination levels are required in a space, very large ventilation rates are involved. It is therefore often impractical to flush out the airborne pathogens simply by using mechanical ventilation, as this results in the installation of a disproportionately large mechanical ventilation system.

Through their experiments on TB transmission, Wells and Riley discovered that there is an epidemiological relationship between the number of infection cases that occur in an outbreak and ventilation rate (3, 4). The relationship is characterised by the Wells-Riley model (4, 5).

$$C = S \times (1 - e^{-(I.q.p.t/Q)}) \qquad (4)$$

Where:

C	=	Number of new infections
S	=	Number of susceptible people
I	=	Number of source cases (i.e. infectors)
q	=	Number of infectious doses generated per minute (quanta/min)
p	=	Pulmonary ventilation rate (l/min)
t	=	Duration of exposure to infection (min)
Q	=	Room ventilation rate (l/min)

By applying equation 4 to data compiled for a TB outbreak (5), Nardell *et al.* were able to predict the theoretical reduction in new infection cases which could be achieved by increasing the ventilation rate. Figure 2 illustrates a prediction made using the Wells-Riley model.

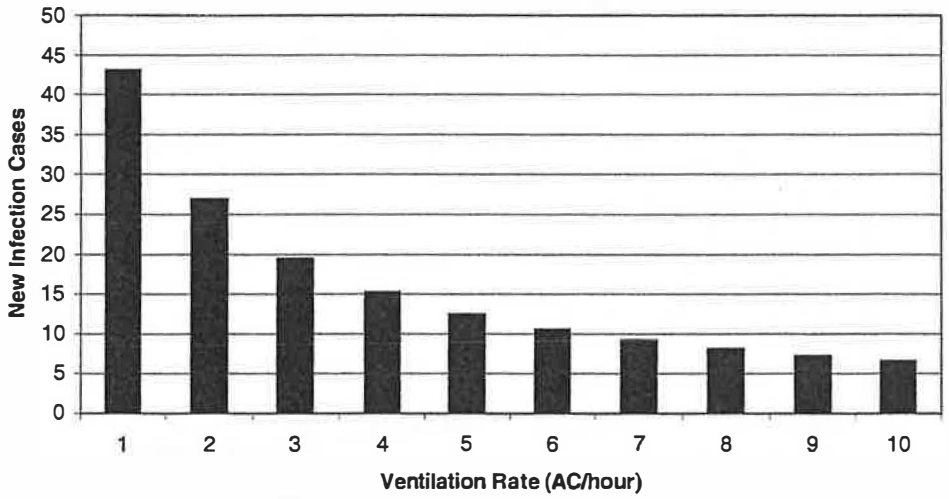

Figure 2 Effect of various ventilation rates on new infection cases

It can be seen that Figure 2 is similar in shape to Figure 1. As the ventilation rate is increased, so the number of new infectious cases decreases. However, it is important to note that graph in Figure 2 is

only indicative. The precise shape of the curve will vary with the particular characteristics of the outbreak, the number of infectious doses generated per minute and the duration of exposure to the infection being two critical factors which influence the shape of the curve (4, 5).

Equations 3 and 4 assume complete mixing of the room air, which in reality it is not possible to achieve. Nevertheless, a well designed dilution ventilation system should try to achieve as much mixing as possible and to avoid any 'short circuiting' since this may result in stagnant regions occurring in the room space. It is in stagnant regions that pathogens may persist and thus be a potential health hazard.

3.2 LAMINAR AND DISPLACEMENT VENTILATION

An alternative approach to simple dilution ventilation is to use carefully directed airflows to displace the contaminated air so that it is 'pushed' air out of the room space. In this way the contaminated air is continually being replaced by clean air. With this type of ventilation system it is undesirable to have any air mixing and so 'laminar' streams of air are often used. Such ventilation systems are commonplace in industrial clean-rooms and are frequently used in operating theatres and isolation rooms. Indeed, 'quasi' displacement ventilation systems are now becoming popular in commercial and public buildings. With infectious patients, laminar flow ventilation can be used to great effect. In isolation rooms, where healthcare workers are particularly vulnerable to airborne nosocomial infection, it is important to produce airflow patterns which reduce the risk of infection. Clean air should therefore be introduced into the room space, so that it passes over the healthcare worker before the infectious patient. The Centers for Disease Control and Prevention (CDC) recommend that airflows should *ideally* be laminar, with supply diffusers located in a wall opposite to the patient, and the exhaust located in a wall near the patient. Alternatively a ceiling supply can be used with the exhaust located at low level in the walls (6). In reality however, laminar flow is impossible to achieve due to spatial restrictions.

3.3 PRESSURE DIFFERENCES

By controlling the airflows within a building it is possible to create 'high' and 'low' pressure regions. This can be used to great advantage in isolation rooms, which can be negatively pressurised space so that airborne pathogens are unable to escape. Although the CDC in the USA recommend a minimum negative pressure of 0.25 Pa for isolation rooms (7, 8), Streifel and Marshall recommend a higher value of 2.5 Pa as an ideal (9) These negative pressures can be achieved by supplying less air to an isolation room than is extracted. This can be achieved by a supply to extract volume differential of between 10 % and 20 % (8, 9, 10). It should be noted that in many countries the location of a negatively pressurised isolation room directly adjacent to a corridor, directly contravenes the fire regulations. In such situations it is recommended that a positively pressurised anteroom be placed between the corridor and the isolation room (11).

One major problem associated with negatively pressurised isolation rooms, is that of maintaining negative pressure at all times. In a recent study in the USA (12), of isolation rooms in 5 hospitals, it was found that over the 5 day study period "none of the control ventilation parameters were met all of the time" and that, for a variety of reasons, all the isolation rooms lost their pressure differential for some period during the study. The most common reason for loss of the pressure differential in isolation room is the opening of doors. Other factors, however, including clogged filters and other adjacent negatively pressurised spaces can result in the pressure differential being lost. The use of a positively pressurised anteroom is one strategy which helps to overcome some of these problems.

The necessity of maintaining negative pressurisation, was graphically illustrated by a TB outbreak in a major teaching hospital in London, in which a patient with MDR TB was admitted to, and placed in a ward side-room, adjacent to a ward in which HIV positive patients were based (13). Unfortunately, the side-room was positively pressurised relative to the adjacent ward, and seven HIV-positive patients contracted MDR TB. This ultimately resulted in the deaths of the index patient and two of the contact patients.

3.4 MECHANICAL OR NATURAL VENTILATION?

Many hospital buildings, especially older facilities, rely heavily on natural ventilation. In many ways the natural ventilation of clinical spaces is a good solution. However, reliance solely on natural ventilation can have a number of drawbacks:

- Ventilation rates will be variable and are greatly dependent on the outside climatic conditions.
- Ventilation may be difficult to control, with airflows being uncomfortably high in some locations and stagnant in others.
- Energy and comfort criteria usually dictate that windows and vents remain closed in winter, when outside temperatures are low. This means that ventilation rates are often much lower in winter than during the summer months.
- Pathogens such as *Aspergillus* spp. which is widespread in the outdoor environment can easily enter ward spaces.

In addition, in deeper plan buildings it is often difficult to ventilate internal enclosed spaces by natural means. Mechanical ventilation systems are therefore often used, as they overcome many of the problems associated with natural ventilation, and can also be used to heat and cool room spaces. Mechanical ventilation is however, not without its disadvantages. There have been a number of notable cases where mechanical ventilation systems have been implicated in TB outbreaks (5, 14, 15). In each case a recirculating mechanical ventilation system was involved and droplet nuclei containing MTB were evenly distributed around the building (or ship in (15)). Since air velocities in excess of 5 m/s are often used in mechanical ventilation systems, it should come as no surprise that poorly designed or maintained systems can very easily become efficient pathogen distribution systems.

Elements in a mechanical ventilation system can become contaminated with microorganisms which form colonies which are then distributed around buildings via the ductwork system. Cooling coils and humidifiers are notorious for becoming contaminated with microorganisms such as *Legionella pneumophila*. Low air velocities (e.g. under 2.25 m/s) and eliminator plates should be employed in order to prevent aerosols from entering the air stream. In addition, Health Technical Memorandum 2025 requires filters to be placed down-stream of any cooling coils or humidifiers (16). While filters are supposed to prevent the spread of pathogens and dirt, they themselves can become contaminated and can become implicated in the spread of airborne pathogens (17, 18).

4.0 ROOM AIR CLEANING DEVICES

There are a variety of room air cleaning devices currently available, incorporating technologies such as high-efficiency particulate air (HEPA) filters, ultraviolet germicidal irradiation (UVGI) lamps and electrostatic filters. These devices are intended to be mounted within a room space and are designed to reduce the microbial level in the room air. They have the advantage that that they are relatively cheap and can be strategically positioned to protect vulnerable patients and staff.

Devices incorporating HEPA filters exhibit very high 'single-pass' efficiencies (i.e. 99.9 % efficient for particles ≥ 0.3 μm in diameter). Recent work by Miller-Leiden *et al.* has shown that, compared with a base condition of 2 ACH, the use of room mounted HEPA filters can achieve reductions in room droplet nuclei concentrations ranging from 30% to 90% (19). This study indicated that room mounted HEPA filtered devices have the potential to greatly reduce the risk of nosocomial infection if correctly utilised.

The 'single pass' efficiency of a room air cleaning mounted device should not be confused with its overall room effectiveness. Although a device may have a single pass efficiency in excess of 99 %, its overall room effectiveness may be much lower, simply because very little of the room air passes through the device. Therefore in order to increase the effectiveness of a room air cleaning device, it is important to maximise the air flow that passes through it. The background room ventilation rate also influences the extent to which a device will be able to reduce the microbial bioburden. For a room air cleaning device the theoretical equilibrium microbial level that can be achieved in a room space can be determined by using equation 5.

$$C_e = \frac{Q_c}{(N_{vent} + \eta_{sp} \cdot N_{uv}) \times V} \qquad (5)$$

where:

C_e	=	Bioburden equilibrium level (cfu/m^3)
Q_c	=	Rate at which microorganisms are introduced room space (cfu/h)
N_{vent}	=	Number of air changes per hour due to ventilation system (h^{-1})
N_{uv}	=	Equivalent number of air changes per hour due to UV device (h^{-1})
η_{sp}	=	Single pass efficiency of UV device
V	=	Room Volume (m^3)

From equation 5 it can be seen that for a room with a background ventilation rate of 2 ACH, the introduction of an air cleaning device having an equivalent air change rate of 2 ACH, will result in a maximum theoretical reduction in the room bioburden of 50 % (assuming a single pass efficiency of 100 % and that complete mixing of the room air takes place). However, if the background ventilation rate were increased to 4 ACH, then the theoretical maximum reduction in bioburden due to the introduction of the UV device will be only 33.3 %. From this we can deduce that although air cleaning devices may produce an initial rapid decrease in pathogen concentrations, the disinfection rate falls off as the contaminant equilibrium level is achieved, and more air must be entrained if lower levels of contamination are to be achieved.

All room air cleaning devices are vulnerable to adverse room air currents and to the short-circuiting of the airflow in the vicinity of the unit. Little is known about the optimum location for room air cleaning devices or how they interact with mechanical ventilation systems. Therefore more research needs to be undertaken in this field. In addition, there are some specific drawbacks associated with devices which contain HEPA filters. HEPA filters have a high resistance to airflow and so larger fans are required to promote air movement, which can create noise problems. Also, as filters become dirty, so the discharge rate of the device falls, which can have a detrimental impact on the room effectiveness of the device.

5.0 UVGI

The lethal effect of UV-C radiation on bacteria has been known for approximately 100 years. The activation spectrum peaks in the range 260 to 270 nm and is similar to the absorption spectrum of nucleic acids, thus deoxyribonucleic acid (DNA) is the main target. Conventional low and medium pressure mercury discharge UV lamps have a strong spectral emission at 253.7 nm, close to the peak of the action spectrum, and can be used as an effective bactericidal agent. UV light at this wavelength is absorbed by nucleic acids with the formation of pyrimidine dimers, resulting in damage to the DNA of the micro-organism which is lethal.

UV lamps can be used in a variety of ways to disinfect air in buildings.

- They can be installed in fan driven room mounted air cleaning devices. In this capacity, they have the advantage over HEPA filters that they offer less resistance to the airflow and so smaller quieter fans can be used.
- They can be installed in return air ducts to purge the extract air of pathogens. Again in this application, they have advantage over HEPA filters that they offer less resistance to the airflow and so can be easily retrofitted to existing mechanical ventilation systems.
- They can be used to produce an upper room UV field, though which pathogens may pass as a result of natural convection through the room space.

The use of upper room UV fields has been and is currently being investigated in the USA. A number of investigators (20, 21) have found it to be particularly effective in rooms where there are strong convection currents and there is a lot of air movement between the lower and upper 'levels' of a room space. Riley *et al.* (20) in particular found that the results achieved by a 17 watt (UV power) fitting when disinfecting BCG, were equivalent to that achieved by a ventilation rate of 12 ACH in a test chamber with a test volume of approximately 61 m^3.

5.1 UV SUSCEPTIBILITY CONSTANTS

The *effective dose* H_{eff} received by an airborne particle can be represented by the expression:

$$H_{eff} = E \times t \qquad (6)$$

where: E = UV irradiance (W/m^2)
t = Duration of exposure to irradiation (s)

The percentage pathogen kill rate can be determined from the H_{eff} value by using equation 7:

$$\frac{N_t}{N_o} = e^{-kH_{eff}} \tag{7}$$

where: k = UV susceptibility constant for pathogen (m^2/J)
N_t = Number of pathogens at time t
N_o = Number of pathogens at t = 0 s

Equation 7 incorporates a term k, which is the UV susceptibility constant for a pathogen. The value of this constant varies with the particular pathogen under examination, and is the gradient of the line produced when the natural logarithm of the survival fraction (N_t / N_o) is plotted against *effective dose* $H_{eff.}$, as represented by equation 8 and shown in Figure 3. The higher the value of k, the more susceptible is the pathogen to damage from UV light.

$$k = \frac{\ln(N_t/N_0)_a - \ln(N_t/N_0)_b}{H_{eff\ b} - H_{eff\ a}} \tag{8}$$

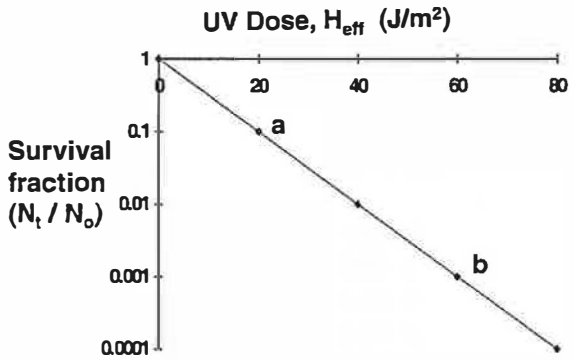

Figure 3 : UV dose verses logarithm of pathogen survival fraction.

UV susceptibility constants have been determined experimentally by a number of researchers. Early studies of the UV susceptibility of micro-organisms were conducted by placing solid surface grown organisms or stirred liquid suspensions under a UV lamp (emitting UVGI at 254 nm) at a fixed distance and intensity (22, 23, 24). Table 2 shows some typical k values determined by using a static approach.

Pathogen	*k* value (m^2/J)	Comments	Source
M. tuberculosis	0.0960	On surface of Agar Medium	(22)
M. tuberculosis	0.0700 - 0.1000	In 3 mm deep Liquid 7H9	(23)
M. bovis BCG	0.0960	On surface of Agar Medium	(22)
M. phlei	0.0500	On surface of Agar Medium	(22)
M. phlei	0.0500	In 3 mm deep Liquid 7H9	(23)
B. subtilis	0.0380	Based on 90 % kill UV dose	(25)
B. subtilis spores	0.0230	Based on 90 % kill UV dose	(25)
Staphylococcus aureus	0.1040	Based on 90 % kill UV dose	(25)
Pseudomonas aeruginosa	0.0490	Based on 90 % kill UV dose	(25)
Pseudomonas aeruginosa	0.2590	On surface of Agar Medium	(22)
Escherichia coli	0.0900	Based on 90 % kill UV dose	(25)
Escherichia coli	0.3000	On surface of Agar Medium	(22)
Escherichia coli	0.2500	In 3 mm deep Liquid 7H9	(23)
Shigella spp.	0.1230	Based on 90 % kill UV dose	(25)
Serratia marcescens	0.2200	On surface of Agar Medium	(22)
Aspergillus niger spores	0.0017	Based on 90 % kill UV dose	(20)
Aspergillus fumigatus spores	0.0043	Based on 90 % kill UV dose	(26)

Table 2 UV susceptibility constants

Riley *et al.* (20) used an entirely different approach to other researchers, by nebulizing bacteria and exposing the resultant aerosols to UVGI. This approach mimics the reality of airborne pathogens, which are atomised in air. Riley *et al.* found that no 'shouldered' graphs were obtained and the calculated *k* values were considerably greater than those found by Collins *et al.*(22) or David *et al.* (23, 24). Riley's results are presented in Table 2.

Pathogen	*k* value (m^2/J	Strain
M. tuberculosis	0.3300	Erdman
M, tuberculosis	0.4800	199RB
M. bovis BCG	0.3700	Culture No. 1
M. bovis BCG	0.2500	Culture No. 2
M. phlei	0.0400	
Serratia marcescens	2.1400	

Table 3 UV susceptibility constants determined by Riley *et al.*(20)

By comparing the results in Table 3 with that in Table 2, it can be seen that the nebulization of the bacteria has a considerable impact on the calculated *k* values. For example, by nebulizing MTB bacilli, it becomes approximately 4 times more susceptible to UVGI. Riley's results therefore suggest that pathogens are more susceptible to UV when in an aerosol form than they are on solid media.

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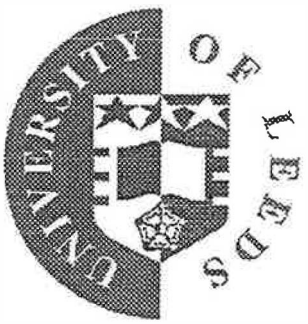

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