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奈米生物性氣膠採樣與分析探討(III)(2/2)

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目錄

一、中文摘要

為達到對感染性病毒氣膠去活化之目的，紫外線之應用也就非常普遍。本研究在實驗室測試腔評估紫外線對空氣以及表面病毒的去活化效率，評估不同紫外線劑量、不同的病毒核酸形式以及不同的相對濕度對於病毒去活化效率之影響。對空氣中病毒來說，紫外線欲去除百分之九十之單股RNA病毒以及雙股RNA病毒別需要劑量 339-423 μW sec/cm^2 和 662-863 μW sec/cm^2。同時本研究也發現，欲對空氣以及表面病毒達到百分之九十九之去除效率所需之紫外線劑量為百分之九十去除效率之兩倍。另外，在相同病毒去除效率下，表面上的病毒所需之紫外線劑量為空氣中病毒的3.9至7.6倍。不論是空氣中或表面上的病毒，單股核酸病毒均比雙股核酸病毒對紫外線具有較佳之感受性。在相對濕度85 %時，紫外線對於病毒去活化效果較相對濕度55 %差，可能的原因是水氣附著於病毒表面遮蔽部分UVGI，導致RNA病毒在高相對濕度下，對紫外線之感受性降低。由以上結果可知，紫外線對於病毒來說具有非常好的去活化效果。

關鍵詞：生物氣膠、病毒氣膠、噬菌體
紫外線

Abstract

The increasing incidence of infectious diseases has prompted the application of Ultraviolet Germicidal Irradiation (UVGI) for the inactivation of viruses. This study evaluates UVGI effectiveness for both airborne and surface viruses in a laboratory test chamber by determining the effect of UV dosage, different nucleic acid type of virus, and relative humidity on virus survival fraction after UVGI exposure. For airborne viruses, the UVGI dose for 90% inactivation was 339-423 μW sec/cm^2 for ssRNA, and 662-863 μW sec/cm^2 for dsRNA. For all tested, the UVGI dose for 99% inactivation was 2 times higher than that for 90% inactivation, and the ratio of surface to airborne UVGI dose ranged from 3.9 to 7.6. Airborne and surface viruses with single-stranded nucleic acid were more susceptible to UV inactivation than were those with double-stranded ones. For all tested viruses at the same inactivation, the UVGI dose at 85% RH was higher than that at 55% RH, possibly because water sorption onto a virus surface provides protection against UV-induced RNA damage at higher RH. In summary, UVGI was an effective method for virus inactivation.

Keywords: bioaerosols ; virus aerosol; bacteriophage ; UVGI

二、前言

Viruses are obligate parasites that are biologically active only within their host. Viruses can be transmitted by various routes, including direct and indirect contact, vector transmission, and vehicle transmission. For deadly viruses such as Severe Acute Respiratory Syndrome (SARS) virus, influenza virus, and enterovirus, the vehicle transmission pathways include respiratory transmission by droplets and aerosols, as well as fecal-oral transmission via water, food, and environmental surfaces. To reduce infection risk from virus infection, control techniques for inactivating such viruses have been extensively researched (Jensen, 1964 ; Gerba et al., 2002 ; Shin et al., 2003; Thurston-Enriquez et al.,2003). Among these control techniques, ultraviolet germicidal irradiation (UVGI) was demonstrated to be extremely efficient for virus inactivation (Jensen, 1964；Galasso et al.,1965；Gerba et al., 2002；Nuanualsuwan et al., 2003; Thurston-Enriquez et al., 2003).

三、研究目的

This study evaluates UVGI effectiveness for both airborne and surface viruses in a laboratory test chamber by determining the effect of UV dosage, different nucleic acid type of virus (single-stranded RNA, ssRNA and double-stranded RNA, dsRNA), and relative humidity on virus survival fraction after UVGI exposure.

四、文獻探討

The mechanisms of UVGI on microbes are uniquely vulnerable to light at wavelengths at or near 2537 Angstroms, because the maximum absorption wavelength of a DNA molecule is 253.7 nm. The pyrimidine of DNA base can strongly absorb UV light. After irradiation, the DNA sequence where pyrimidine and pyrimidine link can form pyrimidine dimers. These dimers can change the DNA double helix structure and interfere with DNA duplication, as well as lead to the destruction of the replicate ability of cells and thus render the cells non-infectious (Brickner et al., 2003). Until now, the application of UVGI has mainly focused on control of tuberculosis transmission, although the susceptibility to UVGI for different microorganism species widely differs (Brickner et al., 2003). The UVGI effectiveness for microorganisms is known to be significantly affected by the irradiation level, duration of irradiation, room configuration, lamp placement, lamp age, air movement patterns, and relative humidity (RH) (Summer, 1962; NIOSH, 1972; CDC, 1994), as well as by the mixing degree of room air (Nicas 1996).

Early research on UVGI applications focused mainly on airborne bacteria, such as *Bacillus subtilis* and *Mycobacterium tuberculosis* (Sharp et al., 1938；Rentschler et al., 1941), as well as fugal spores, such as *Fusarium*, *Penicillium*, and *Aspergillus* species (Luckiesh et al., 1946). Recent studies report that the UV susceptibility of these microorganisms is significantly reduced when the RH is increased (Peccia et al., 2000; Ko et al., 2000), that airborne microorganisms are much more susceptible to UV damage than those suspended in a liquid broth (Brickner et al., 2003), and that the UVGI dose between fungal spores and bacterial cells is as high as 80 times (Lin and Li, 2002). These previous studies reveal that the susceptibility of microbes is highly related to the presence or absence of a cell wall, to the cell-wall thickness, and to RH.

Until now, only limited data has been available on the inactivation of airborne viruses by UVGI. In 1942, the use of UVGI in schools greatly reduced the spread of measles, chickenpox, and mumps (Wells et al., 1942). Recently, adenovirus was reported less susceptible to UVGI, possibly due to dsDNA as its genetic material (Thurston-Enriquez et al., 2003). Moreover, the required dose of UVGI for viruses that

lack a cell wall is lower than that for bacteria and fungi (Jensen, 1964; Brickner et al., 2003). Virus inactivation by UVGI also depends on the type of nucleic acid; viruses with double-stranded genomes are less susceptible to UV inactivation (Thurston-Enriquez et al., 2003) possibly because only one strand of the nucleic is damaged during inactivation, and thus the undamaged strand might then serve as a template for repair by host enzymes (Kallenbach et al., 1989). In virus inactivation, UVGI predominately damages DNA and inhibits replication. However, only limited information is available about the mechanism of UVGI on RNA viruses.

For assessment of UVGI inactivation on viruses, bacteriophages have been used as surrogates for mammalian viruses. Among these phages, MS2 has been suggested as an adequate indicator for enteric virus UV inactivation, because the size, shape, and nucleic acid type of MS2 are similar to those of enteric virus (Havelaar et al., 1991). Therefore, MS2 has been used as a surrogate for poliovirus and other enteric viruses (Jones et al., 1991; Maillard et al., 1994). The susceptibility of these different nucleic acid types to UVGI inactivation might not be the same.

Viruses from fecal-oral transmission via different environmental surfaces are also directly related to the potential risk for human exposure, such as SARS virus and enterovirus. SARS virus and enterovirus are apparently most commonly spread by close person-to-person contact through exposure to infectious droplets and possibly by direct or indirect contact with infected body fluids (Tsang, et al., 2003). Emerging evidence indicates that these infectious viruses can also be acquired from contaminated inanimate objects in the environment (Poutanen et al., 2003). Therefore, applications of UVGI on surface viruses are equally important as on airborne viruses. Until now, however, only limited data has been available about the virus inactivation by UVGI to virus-coated solid surfaces. Most investigations of UVGI inactivation to surface viruses have involved virus stock solution in glass dishes for UVGI irradiation (Galasso et al., 1965；Ma et al., 1994). Although UVGI effectively inactivated a high titer (10^8 PFU/ml) of virus on the surface of water (> 99% of the virus was inactivated by UV light), a corresponding inactivation effect on viruses bound to a solid surface was not realized. Compared with airborne viruses, viruses bound to both water and solid surfaces were more difficult to inactivate (Galasso et al., 1965). Therefore, to simultaneously eliminate both airborne and surface viruses, understanding and quantifying the effective UVGI dose for surface virus inactivation is crucial.

In our current study, the effectiveness of UVGI was evaluated for both airborne and surface viruses in a laboratory test chamber by determining the effect of UV dosage, different nucleic acid type of virus (different bacteriophages with single-stranded RNA, and double-stranded RNA), and RH (55% and 85%) on virus survival fraction after UVGI exposure.

五、研究方法

Test Viruses

In this study, the test viruses were two different bacteriophages: single-stranded RNA, or ssRNA (MS2, ATCC 15597-B1), and double-stranded RNA, or dsRNA (phi 6 with envelope lipid, ATCC 21781-B1). The host bacteria were ***Escherichia coli*** for coliphages MS2 (ATCC 15597) and ***Pseudomonas syringae*** (ATCC 21781) for phi 6. A high titer stock of bacteriophages (10^9-10^{10} PFU/ml, where PFU is Plaque Forming Units) was prepared via plate lysis and elution. To allow the phage to attach to the host, the bacteriophages were mixed with their own respective host. First, 5 ml of top agar was added to a sterile tube of infected cells. Then, the contents of the tube were mixed by gentle tapping for 5 sec and poured

onto the center of a labeled agar plate. Finally, the plate was incubated for 24 h either at 37 °C for coliphages or at 26 °C for phi 6. After cultivation, 5 ml SM buffer (containing NaCl, $MgSO_4 \bullet 7H_2O$, Tris, and gelatin) was pipetted onto a plate that showed confluent lysis. Then, the plate was slowly rocked for 40 min and the buffer was transferred to a tube for centrifugation at 4,000 x g for 10 min. After the supernatant was removed, the resulting phage stock was stored at 4 °C.

Aerosol Test System

(I) Aerosol Generation Unit

A Collison three-jet nebulizer (BGI Inc., Waltham, MA) was used to nebulize the bacteriophage stock in deionized water at 3 L/min with dry, filtered, compressed laboratory air, then passed though a Kr-85 particle-charge neutralizer (model 3077, TSI). The aerosolized suspension was then diluted with filtered, compressed air at 57 L/min. The stock solutions of bacteriophages MS2 was diluted in sterile, deionized water for nebulization, and that of phi 6 phage was diluted in sterile, deionized water containing 0.03 % Tween 80 to preserve infectivity.

(II) RH Regulation Unit

A humidified gas stream was generated by passing pure compressed air through a humidity saturator. The water vapor content (i.e., RH) in the gas stream was adjusted by changing the flow rate ratio of humidified gas stream to dry gas stream, and finally measured using a hygrometer (Testo, Sekunden-Hygrometer 601) placed in the sampling chamber. For evaluating the effect of RH, the humidified gas stream was heated by adding a dry gas stream to reach the medial (RH 55%) or humid condition (85%).

(III) UV Exposure Unit

As shown in Fig. 1, the eight Germicidal lamps (Philips Germicidal Lamp, TUV 8W/G8 T5, Holland) were low-pressure mercury-vapor discharge lamps consisting of a tubular glass envelope that emitted short-wave UV radiation with a radiation peak at 253.7 nm (UV-C) for germicidal action. Each lamp was 28.8 cm long, and was two-ended with a two-pin base. The UV irradiance intensity was measured using a radiometer (P-97503-00, Cole-Parmer, France) with a 254nm sensor. Exposure of airborne virus to a given intensity of UV was carried out by passing the aerosolized suspension through a cylinder (5-cm diameter, 28-cm length, made of quartz) at a distance from 0 to 30 cm from the UV source (with a radiation peak at 254 nm). The UV irradiance intensity was measured using a radiometer (P-97503-00, Cole-Parmer) with a 254-nm sensor fixed inside the cylinder and oriented with its surface parallel to the germicidal lamps. Therefore, an average facial intensity (four faces) could be obtained. With an air flow rate of 60 L/min and UV exposure volume of 0.55 L, the exposure time was 0.55 sec. The evaluated parameter was UV dose, defined as the product of UV intensity and UV exposure time. Experiments were done at least in triplicate for each set of conditions with different UV intensity (60, 120, 180, or 240 $\mu W/cm^2$), RH (55% and 85%), and test virus. The test system was located in a chemical hood so that the exhausted gas was vented outside (Lin and Li, 2002).

(IV) Virus Aerosol Sampling

An Andersen one-stage viable impactor (Andersen Samplers, Inc., Atlanta, GA) was used to sample the virus aerosol. This stage has four hundred 0.25-mm holes and has a sampling flow rate of 28.3 L/min (corresponding to a velocity of 24 m/s) when 20 ml LB (Luria-Bertani) broth is used with 3% gelatin plates. The measured and theoretical cut-point diameters of this stage are 0.57 μm and 0.65 μm, respectively (Nevalainen et al. 1993). Because this impactor has only one sampling port, samples of each virus aerosol were taken in sequence first without and then with UVGI irradiation. To collect a sufficient

concentration of virus, the sampling times without UVGI exposure ranged from 30 sec to 1 min, and those with UVGI exposure ranged from 1 min to 5 min. After sampling, the plate with collection medium from the impactor was placed in an incubator at 37 °C for 10 min. All of the viral samples were subjected to plaque assay for coliphage at 37 °C and for phi 6 at 26 °C. Then, PFU per cubic meter (PFU/m^3) was calculated based on the dilution ratio, plated volume, sampling time, and sampling flow rate. Our results showed that the virus infectivity in the aerosolized suspension and aerosol phase (at 55% and 85% RH) could be maintained up to 90 min with a coefficient of concentration variation less than 25% (Tseng and Li., 2005). Therefore, the natural decay rates of the aerosolized suspension were found to be insignificant.

Surface Test System

(I) Solid Media on a Gelatin Agar Plate

A diluted culture of virus stock solution (0.1 ml) was spread on the surface of LB (Luria-Bertani) media plates (with 3% gelatin) and then dried for 20 min in laminar flow. The virus concentration in each plate was 10^8 PFU/ml. All of the viral samples (both UVGI-exposed and unexposed samples) were subjected to plaque assay for coliphage at 37 °C and for phi 6 at 26 °C. For UVGI-exposed samples, the UV-induced inactivation on virus growth was observed. For all viral samples, the observed incubation time period was 24 hours. The virus survival fraction was calculated as the ratio of the number of plaques forming on the UVGI-exposed plates compared to that on the UVGI-unexposed control plates. Based on our preliminary test (data not shown), we selected the UVGI intensity range from 60 μW/cm^2 to 240 μW/cm^2, and the exposure time range from 3 sec to 6 min. Experiments were done at least in triplicate for each set of conditions for different UV intensity (60, 120, 180, or 240 μW/cm^2), RH (55% and 85%), and test virus. The test system was located in a chemical hood so that the exhausted gas was vented outside.

(II) RH Regulation Unit

The experimental apparatus used for RH regulation was the same as that used in the aerosol test system described above, consisting of a compressed air system, RH conditioner, and an UV exposure chamber. The humidified gas stream was generated by passing pure compressed air through a humidity saturator. The air temperature and RH (55% and 85%) throughout the trials were monitored using a humidity/temperature sensor (Hygromer-A1, Rotronic) mounted inside the chamber.

(III) UV Exposure Unit

The UV exposure chamber was approximately 26 liters in volume (26.5 cm x 30 cm x 33 cm). The exposed samples were irradiated with four 8W UV-C lamps (Philips Germicidal Lamp, TUV 8W/G8 T5, Holland), which were placed 30.5 cm above the surface of the media. Lamps were wrapped in a layer of cellophane to attenuate original irradiation magnitude. The intensity of UVGI on the surface of the media was measured using an UV-radiometer (P-97503-00, Cole-Parmer, France) with a 254-nm sensor. The UV intensity level was adjusted to 60, 120, 180, or 240 μW/cm^2 by changing the number of lamps. Finally, the UV dose was calculated as the product of the UV intensity and UV exposure time.

• Survival Fraction of Viruses vs. UVGI Exposure

The total dose to which an airborne virus was exposed was defined as the product of the UVGI intensity *I* on the microbe and the exposure time *t*. The survival fraction is a ratio that represents the virus concentration after UVGI exposure, and defined as

$$\frac{N_{a,uv}}{N_{a,0}} or \frac{N_{s,uv}}{N_{s,0}} = e^{-KIt}$$

where

$N_{a,uv}$ = concentration of airborne virus

surviving after exposure to UVGI by using one-stage Andersen sampler (PFU/m^3)

$N_{a,0}$ = concentration of airborne virus unexposed to UVGI by using one-stage Andersen sampler (PFU/m^3)

$N_{s,uv}$ = centration of surface virus surviving after exposure to UVGI (PFU/ml)

$N_{s,0}$= concentration of surface virus unexposed to UVGI (PFU/ml)

I = UV intensity (μW/cm^2)

t = UV exposure time

K = microorganism susceptibility factor (cm^2 /μW sec)

Statistics

The parameter exponential log of the survival fraction vs. UV dose for each experiment was used to perform regression analysis on the data for each virus. Comparisons of survival fraction among the viruses were performed using *t* test to evaluate statistically significant differences.

六、結果與討論

In this study, the germicidal effect of UVGI was evaluated for both airborne and surface viruses. The effect of UV dose and RH was evaluated for two different bacteriophages selected to represent virus nucleic acid: bacteriophages with ssRNA (MS2), or dsRNA (phi 6).

The effectiveness of UVGI on both airborne and surface viruses inactivation was fitted well with an exponential decay model where the logarithm of survival fraction of virus surviving with UVGI exposure was linearly proportional to UVGI dose. Our findings were also consistent with the Bunsen-Roscoe reciprocity law, which states that virus survival fraction with UVGI irradiation being dependent on UV dose, is not affected by reciprocal changes in UV intensity or to exposure time. In summary, the germicidal effects of UVGI for airborne and surface virus inactivation depended on UV dose, neither UV intensity nor exposure time. In this study, the survival fraction decreased exponentially with increasing UVGI dose.

Survival Fraction of Airborne Viruses after UVGI Exposure Figures 2 and 3 show the measured survival fraction of two viruses at the two RH conditions. For all four viruses, the survival fraction was inversely related to UVGI dose. To obtain 90% virus inactivation, the ssRNA virus (MS2) required only an extremely low dose (339-423 μW sec/cm^2), the the dsRNA virus (phi 6) required a relatively high dose (662-863 μW sec/cm^2). These results indicate that the UVGI dose for 90% inactivation of dsRNA and dsDNA viruses is approximately 2 times higher than that of ssRNA and ssDNA viruses.

To obtain 99% virus inactivation, the ssRNA virus (MS2) required a dose of 803-909 μW sec/cm^2, and the dsRNA virus (phi 6) required a dose of 1388-1771 μW sec/cm^2. Similar to the results for 90% inactivation, these results indicate that the UVGI dose for 99% inactivation of dsRNA viruses is approximately 2 times higher than that of ssRNA viruses.

The required doses for the two viruses evaluated in this study are similar to those reported for airborne fragile bacteria, ***E.coli*** (Lin and Li, 2002), but are significantly lower than those for endospore bacteria (***B. subtilis***), fungi (yeast), and fungi spore (***P. citrinum***). Therefore, UVGI is clearly more effective for inactivation of airborne virus than for inactivation of yeast and other spore-type microorganisms.

In our study, the survival fraction decreased exponentially with increasing UVGI dose. Based on simple exponential regression analyses, the microorganism susceptibility factor, K (expressed in cm^2 /μWs), which is a commonly used indicator of the sensitivity of the test microorganism, varied widely. MS2 showed the highest K (0.0054-0.0068) and phi6 the lowest (0.0031-0.0043).

Previous findings (Thurston-Enriquez et al., 2003) suggest that viruses with dsRNA or dsDNA are less susceptible to UV inactivation. The reason is that only one strand of the nucleic acid is damaged during inactivation, and the undamaged strand might then serve as a template for repair by host enzymes (Kallenbach et al., 1989). For DNA viruses, host cells can contain the enzymatic machinery to repair damage by excision or recombinational repair. This has been suggested as a reason for the shouldering effect observed in UV inactivation experiments involving dsRNA viruses. Except for the complex nucleic acid, the capsid structure and the lipid component of virus might act as barriers that prevent UVGI penetration. Therefore, shielding or absorption of UV irradiation before reaching the nucleic acids might occur. Based on our results (as shown in Figures 2 and 3), phi6 with high-resolution capsid (Bamford et al., 2002) and lipid envelope showed higher susceptibility to UVGI than did isosahedral bacteriophages (MS2) without lipid.

The *K* of airborne viruses studied here ranged from 0.0031 to 0.0081, similar to that reported (Lin and Li, 2002) for bacterial aerosol of *E. coli* (0.0032–0.0054), but much higher than that for a fungal aerosol of yeast (0.00036-0.00050),*B.subtilis*(0.00039-0.0005 0), and *P.citrinum* (0.000092-0.00015). These findings reveal that the susceptibility to UVGI of viruses is similar to that of fragile bacteria, but is higher than that for endospore bacteria, yeast, and fungi spores. These results can be explained as follows; the susceptibility of microorganisms to UV irradiation is highly related to the presence or absence of a cell wall, to the cell-wall thickness, and to the type of nucleic acid. Because viruses lack a cell wall, they might be more susceptible to UVGI irradiation.

For all viruses tested here, *K* (0.0031-0.0064) at 85% RH was lower than that (0.0043-0.0081) at 55% RH (Figs. 2 and 3), indicating that a higher UVGI dose was required to inactivate a virus at higher RH conditions. At higher RH, the UVGI was apparently attenuated by water vapor. The water sorption onto a virus surface might also provide protection against UV-induced RNA damage when RH is increased (Peccia et. al, 2001).

Survival Fraction of Surface Viruses after UVGI Exposure

Figures 4 and 5 show the virus survival fraction and UV exposure dose for the two tested viruses at 55% and 85% RH, respectively, for surface evaluation. For 90% inactivation, the ssRNA virus (MS2) required an extremely low UV dose (658 to 1332 μWsec/cm^2), and dsRNA (phi 6) required a relatively higher dose (1294 to 4352 μW sec/cm^2). These results clearly indicate that dsRNA viruses are more resistant to UV light inactivation than are ssRNA viruses. The measured UVGI dose for the ssRNA virus (658 to 1332 μWsec/cm^2) agrees with that previously reported for poliovirus type 1 (ssRNA), 960 μWsec/cm^2 necessary for 90% inactivation on clear suspending medium (Nuanualsuwan and Cliver, 2003).

For 99% surface virus inactivation, the UVGI dose for MS2 ranged from 2376 to 3310 μW sec/cm^2, and for ph6 from 7644 to 8915 μW sec/cm^2. These results indicate that the dose for surface virus inactivation for dsRNA viruses is approximately 3 times higher than that for ssRNA viruses. These UV doses for 99% surface virus inactivation are similar to that previously reported for *E.coli* (4160 to 5530 μW sec/cm^2), but much lower than that for *subtilis* (24920 to 40310 μW sec/cm^2), yeast (12260 to 13700 μW sec/cm^2), and *P. citrinum* (30160 to 41520 μW sec/cm^2) (Lin and Li, 2005). The K of the surface viruses studied here was the highest for MS2 (0.0007-0.0017) and lowest for T7 (0.0002-0.0004), indicating that dsRNA viruse is more resistant to UV irradiation than are ssRNA viruse. Comparison with the K of airborne viruses measured here reveals that the apparent UV lethal radiation doses required for airborne

viruses are lower than those for surface viruses. With respect to RH effect on surface virus inactivation, for all viruses tested here the survival fraction at 85% RH was higher than that 55% RH. The humid conditions possibly promote water sorption onto a virus surface that might provide protection against UV-induced DNA damage.

Comparison of UVGI Dose for Airborne and Surface Virus Inactivation

Our results show the UV lethal radiation doses required for airborne viruses (Figs. 2 and 3) were lower than those for surface viruses (Figs. 4 and 5). Furthermore, the ratio of the inactivation dose for surface viruses to airborne viruses for 90% inactivation ranged from 3.9 to 7.6 for MS2, and from 5.7 to 6.2 for phi 6. One explanation for the higher dose required for surface virus inactivation might be that only one side of the surface virus was exposed to UVGI on the medium surface, whereas the entire surface of the airborne virus was exposed to UVGI. Furthermore, because the viruses on a surface might be aggregated, a higher UVGI dose might be needed to inactivate a surface virus. Therefore, not only the UVGI intensity and the exposure time but also the degree of virus aggregation affected the UV effectiveness on surface viruses (Galasso et al., 1965). For bacteria and fungi (Lin and Li, 2002; Lin and Li, 2005), the ratio of the inactivation dose for surface viruses to airborne viruses ranged from 4.18 to 5.62 for *E. coli*, from 2.95 to 3.38 for *B. subtilis*, from 1.18 to 1.57 for yeast, and from 0.69 to 1.35 for *P. citrinum.* Based on our results and these previous results, the UVGI dose that can inactivate surface microorganisms will be more than adequate to inactivate airborne microorganisms, especially for viruses and fragile bacteria.

In summary, our current results agreed with the previous extensive laboratory and model-room studies that the germicidal effects of UVGI irradiation on viral nucleic acid are related to the UV intensity and exposure time (Ko et al., 2000；Ko et al., 2002). UVGI inactivation of ssRNA was easier than that of dsRNA and dsDNA viruses, regardless of whether the viruses were suspended in air or on a surface. In addition, viruses could be protected from the UV light inactivation by a complex nucleic acid, by strong capsid structures, by host cell repair mechanisms, and by lipid content. For all viruses evaluated here, the survival fraction at 85% RH was higher than that at 55% RH. Finally, the UVGI dose that can inactivate surface viruses will be more than adequate to inactivate airborne viruses.

七、計畫成果自評

本計畫已在實驗室中建立病毒氣膠採樣與控制的評估系統，並已運用此系統評估紫外線對空氣以及表面病毒之去除效果，此成果可運用於環境病毒之控制，並進一步達到預防病毒感染之成效。

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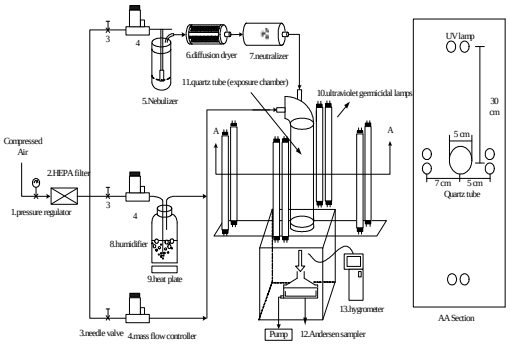

Fig. 1. Experimental apparatus to evaluate UVGI on virus-containing aerosols. 1. pressure regulator; 2. HEPA filter; 3. needle valve; 4. mass flow controller; 5. nebulizer; 6. diffusion dryer; 7. neutralizer; 8. humidifier; 9. heat plate; 10. ultraviolet germicidal lamps; 11. quartz tube (exposure chamber); 12. Andersen sampler; 13. hygrometer. b. AA Section. The distance between UV lamps and quartz tube is adjustable (from 0 to 30 cm).

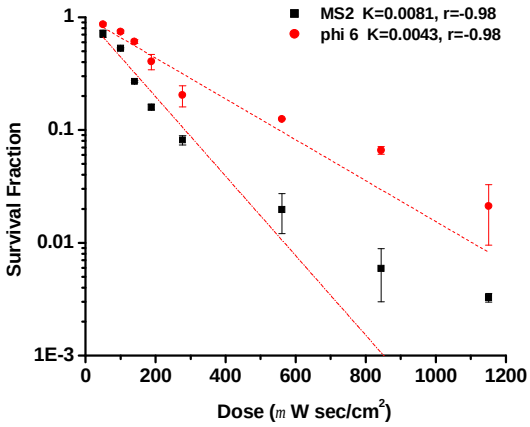

Fig. 2. Survival fraction of airborne viruses (MS2, and phi 6) exposed to UVGI at RH 55%. Error bars represent one standard deviation of the mean of at least three trials.

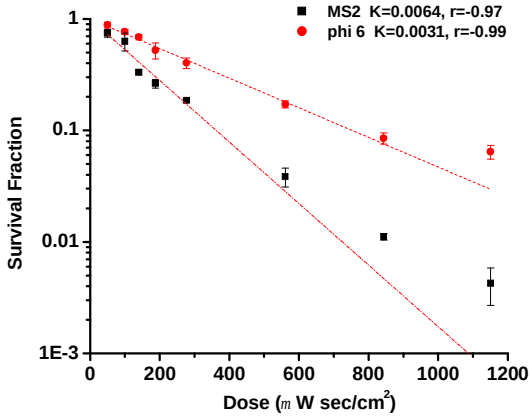

Fig. 3. Survival fraction of airborne viruses (MS2, and phi 6) exposed to UVGI at RH 85%. Error bars represent one standard deviation of the mean of at least three trials.

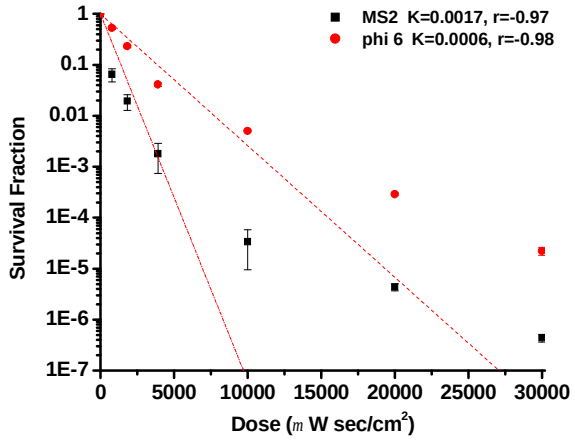

Fig. 4. Survival fraction of surface viruses (MS2, and phi 6) exposed to UVGI at RH 55%. Error bars represent one standard deviation of the mean of at least three trials.

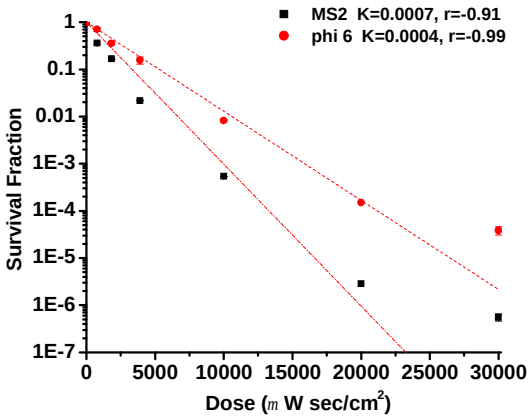

Fig. 5. Survival fraction of surface viruses (MS2, and phi 6) exposed to UVGI at RH 85%. Error bars represent one standard deviation of the mean of at least three trials.