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Inactivation of Virus-Containing Aerosols by Ultraviolet Germicidal Irradiation

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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 airborne viruses in a laboratory test chamber by determining the effect of UV dosage, different nucleic acid type of virus (single-stranded RNA, ssRNA; single-stranded DNA, ssDNA; double-stranded RNA, dsRNA; and double-stranded DNA, dsDNA), 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, 444–494 μW sec/cm^2 for ssDNA, 662–863 μW sec/cm^2 for dsRNA, and 910–1196 μW sec/cm^2 for dsDNA. For all four tested, the UVGI dose for 99% inactivation was 2 times higher than that for 90% inactivation. Airborne viruses with single-stranded nucleic acid (ssRNA and ssDNA) were more susceptible to UV inactivation than were those with double-stranded ones (dsRNA and dsDNA). 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 DNA or RNA damage at higher RH. In summary, UVGI was an effective method for inactivation of airborne virus.

INTRODUCTION

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).

The mechanisms of UVGI on microbes are uniquely vulnerable to light at wavelengths at or near 253.7 nm, because the maximum absorption wavelength of a DNA molecule is 260 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. 2001; Ko et al. 2000), that airborne microorganisms are much more susceptible to UV damage than those suspended in a liquid suspension (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.

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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 double-stranded DNA as its genetic material (Meng and Gerba 1996; Thurston-Enriquez et al. 2003). Moreover, the required doses of UVGI for viruses were found to be lower than those for bacteria and fungi (Jensen 1964; Brickner et al. 2003). In addition, virus inactivation by UVGI was observed to depend on the type of nucleic acid, as well as viruses with double-stranded genomes are less susceptible to UV inactivation than those with single-stranded genomes (Thurston-Enriquez et al. 2003). That 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.

The major mechanism of UVGI inactivation on microbes is based on both physical and biochemical inactivation process. It was believed that radiation could restructure the nucleic acid of the microorganism and destroy its replication ability. Therefore, the type of the viral nucleic acid may play a critical role on virus inactivation by UVGI. According to the types of the nucleic acids, viruses could be divided into four groups such as single-stranded RNA (ssRNA); single-stranded DNA (ssDNA); double-stranded RNA (dsRNA); and double-stranded DNA (dsDNA). Until now, very few data are available regarding viruses inactivation by UVGI among the four groups. Therefore, it is necessary to evaluate the UVGI effectiveness on all the four nucleic acid groups of viruses.

For evaluating effectiveness of UVGI on viruses, bacteriophages have been used as indicators of viral pathogens in environmental virology applications, because phages are innocuous and allowing for expeditious and economical screening for the presence of pathogenic mammalian viruses (Chung and Sobsey 1993). In addition, bacteriophages can grow to higher titers than most mammalian viruses do. Therefore, bacteriophages could constitute a more sensitive assay. Among these phages, MS2 has been suggested as an adequate indicator because of high resistance to UGVI in water. Furthermore, the size, shape, and nucleic acid type of MS2 is similar to the characteristics 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).

In our study, the effectiveness of UVGI was evaluated for airborne viruses in a laboratory test chamber by determining the effect of UV dosage, different nucleic acid type of virus (four different bacteriophages with ssDNA, ssRNA, dsDNA, and dsRNA), and RH (55% and 85%) on virus survival fraction after UVGI exposure.

MATERIALS AND METHODS

Test Viruses

In this study, the test viruses were four different bacteriophages: ssRNA (MS2, ATCC 15597-B1), ssDNA (phi X174, ATCC 13706-B1), dsRNA (phi 6 with envelope lipid, ATCC 21781-B1) and dsDNA (T7, ATCC 11303-B1). The host bacteria were *Escherichia coli* F-amp (ATCC 15597) for MS2, *Escherichia coli* CN-13 (ATCC 13706) for phi X174, *Escherichia coli* 11303 (ATCC 11303) for T7, as well as *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 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. The medium for MS2, phi X174, T7, and phi 6 phage cultivation include Luria-Bertani Agar (Difco Laboratories, 244520), Nutrient Agar (Difco Laboratories, 213000) with 0.5% NaCl, Trypticase Soy Agar (Difco Laboratories, 236950), and NBY Agar (containing Nutrient Broth, Yeast extract, K_2HPO_4, KH_2PO_4, and $MgSO_4 \cdot 7H_2O$), respectively. 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, $MgSO_4 \cdot 7H_2O$, 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 × g for 10 min. After the supernatant was removed, the remaining phage stock was kept at −80°C. From our preliminary results (data not shown), virus infectivity could be maintained for 24 h at 4°C. For UVGI experiments, the virus titers were determined by plaque assay, and the virus suspension was stored at 4°C within 24 h.

Aerosol Test System

(I) Aerosol Generation Unit

In general, virus droplets generated from sneezing or coughing typically are in the size of 1 μm to 100 μm, and evaporate to droplet nuclei that approach the size of the individual microbe in the air (Kowalski and Bahnfleth 1998). The droplet nuclei remain airborne for long periods of time with potential risk for retention in the respiratory tract (Ijaz et al. 1987). When a virus is encased in a droplet, its infectivity is enhanced because of shielding from drying, temperature, and sunlight (Tyrrell 1967). It was indicated that virus-containing aerosols less than 2 μm in size have higher infectivity than those of the virus itself (Couch et al. 1965). From the previous studies (Buckland et al. 1962; Couch et al. 1965; Benbough 1971), virus-containing aerosols were generated by Collison three-jet nebulizer to range from 0.5 μm to 3.0 μm.

In our current study, 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, phi X174, and T7 were diluted in sterile, deionized water for nebulization. For phi 6 phage, the stock solution was diluted in sterile, deionized water containing 0.03% Tween 80 to preserve infectivity. In all of the experiments, the phage concentrations in the nebulizer were ranged from 2×10^8 to 7×10^8 PFU/ml.

(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%) at 25–28°C.

(III) UV Exposure Unit

As shown in Figure 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 254 nm 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 μW/cm^2), RH (55% and 85%), and test virus (MS2, phi X174, T7, and phi 6). 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

From our previous investigation (Tseng and Li 2005), an aerodynamic particle sizer (APS, Model 3310A, TSI, Inc., St. Paul, MN) was used to measure the real-time number concentration and size distribution of the virus-containing aerosols in the test chamber. By using the Andersen 6-STG sampler, the measured geometric mean aerodynamic diameter of MS2, phi $\times$174, T7, and phi 6 was found to be 1.23 μm, 1.25 μm, 1.24 μm, and 1.25 μm, respectively, with a geometric standard deviation of 1.5. In addition, more than 95% of virus-containing aerosols were found to be less than 2.1 μm in diameter. For UVGI evaluation, an Andersen one-stage viable impactor (Andersen Samplers, Inc., Atlanta, GA) was used to collect virus-containing aerosols before and after UVGI treatment. 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. For collecting sufficient concentrations of virus on agar plate (at least 30 plaques) sampling times ranged from 30 sec to 1 min without UVGI exposure, and ranged from 1 min to 5 min with UVGI exposure. The lower limit of 30 plaques is necessary to obtain sufficient statistical power for comparison purposes (Lembke et al. 1981; Thorne et al. 1992).

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 coliphages at 37°C and for phi 6 at 26°C. Then, PFU per cubic meter (PFU/m^3) was calculated based on plaque numbers, 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.

From our previous study (Lin and Li 2002), it was revealed that UV could induce delays in the growth of microbes (several hours for *E. coli*, one day for *B. subtilis* and yeast, as well as two or three days for *P. citrinum*). This delayed growth effect occurs because visible light or near-UV light (about 330 nm to 480 nm) can help repair photochemical damage as much as 80% in microbes (Ko et al. 2000). In our current experiments, each medium plate removed from the Andersen sampler was immediately stored in a dark incubator to prevent photoreactivation. From our preliminary results (data not shown), there were no delayed growth effect for the four evaluated viruses.

Survival Fraction of Viruses versus 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

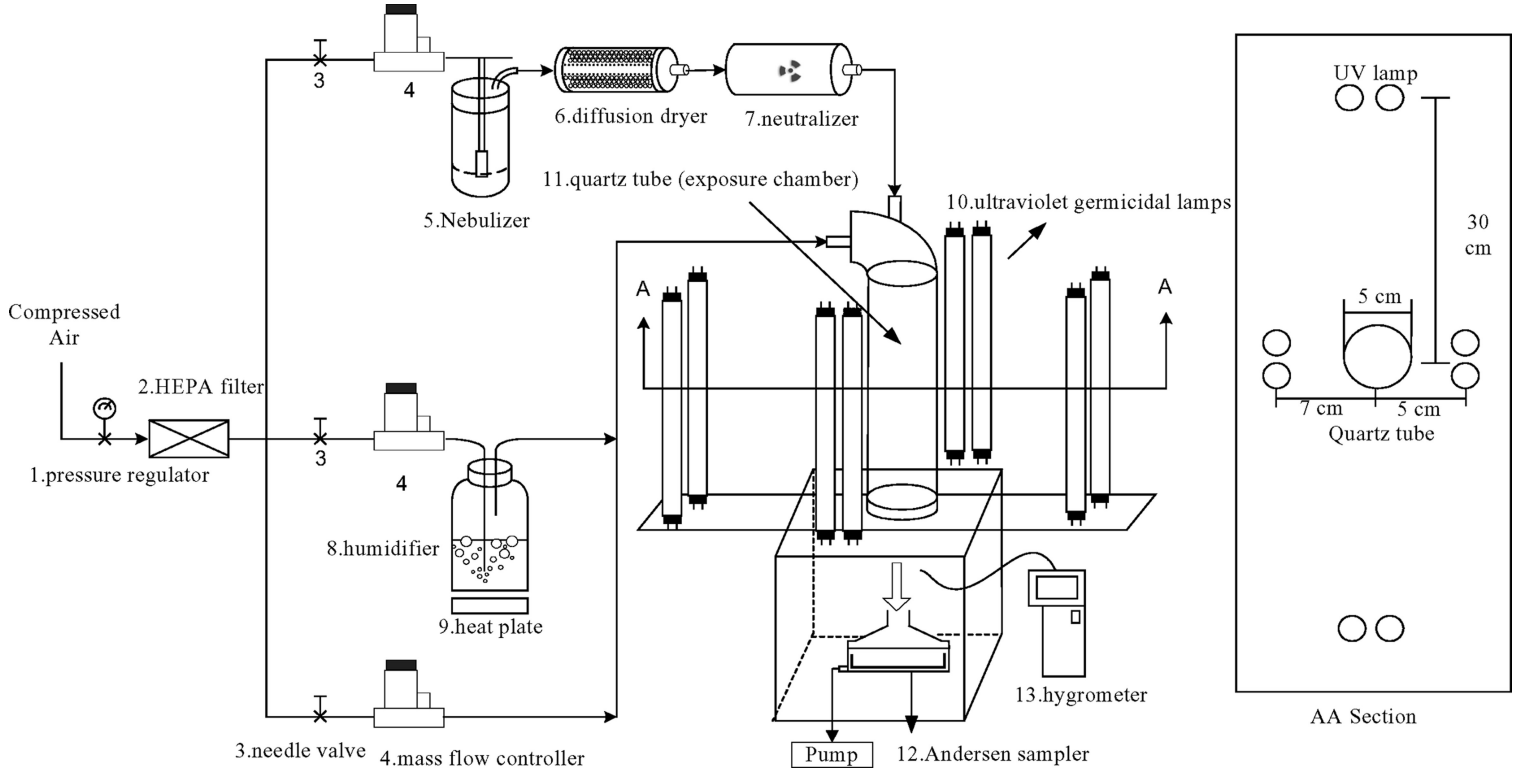

FIG. 1. Experimental apparatus to evaluate UVGI on virus-containing aerosols. 1. pressure regulator; 2. HEPA filter; 3. needle valve; 4. mass flow controller; 5. nebulizer; 6. diffusion dryer; 7. neutralizer; 8. humidifier; 9. heat plate; 10. ultraviolet germicidal lamps; 11. quartz tube (exposure chamber);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

defined as

$$\frac{N_{uv}}{N_0} = e^{-KIt}$$

where

$N_{,uv}$ = concentration of airborne virus surviving after exposure to UVGI by using one-stage Andersen sampler (PFU/m^3)
N_0 = concentration of airborne virus unexposed to UVGI by using one-stage Andersen sampler (PFU/m^3)
I = UV intensity (μW/cm^2)
t = UV exposure time (sec)
K = microorganism susceptibility factor (cm^2/μW sec)

Statistics

The parameter exponential log of the survival fraction versus 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.

RESULTS AND DISCUSSION

In this study, the inactivated effect of UVGI was evaluated for airborne viruses. The effect of UV dose and RH was evaluated for four different bacteriophages selected to represent all types of virus nucleic acid: bacteriophages with ssDNA (phi X174), ssRNA (MS2), dsDNA (T7), or dsRNA (phi 6).

The effectiveness of UVGI on airborne virus inactivation was fitted well with an exponential decay model. Our findings were also consistent with the Bunsen-Roscoe reciprocity law, which states that the survival fraction of virus with UVGI irradiation being dependent on UV dose, is not affected by reciprocal changes in UV intensity or to exposure time. In summary, the UVGI effects for airborne virus inactivation depended on UV dose, neither UV intensity nor exposure time. For all nucleic acid types of virus evaluated in this study, the survival fraction decreased exponentially with increasing UVGI dose.

From survival fraction at two RH conditions (as shown in Figures 2 and 3), the survival fraction of all four viruses 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 ssDNA virus (phi X174) a relatively low dose (444–494 μW sec/cm^2), the dsRNA virus (phi 6) a moderate dose (662–863 μW sec/cm^2), whereas dsDNA (T7) required a relatively high dose (910–1196 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 ($p < 0.05$).

To obtain 99% virus inactivation, the ssRNA virus (MS2) required a dose of 803–909 μW sec/cm^2, the ssDNA virus (phi X174) a dose of 974–1031 μW sec/cm^2, the dsRNA virus (phi 6) a dose of 1388–1771 μW sec/cm^2, and dsDNA (T7) a dose

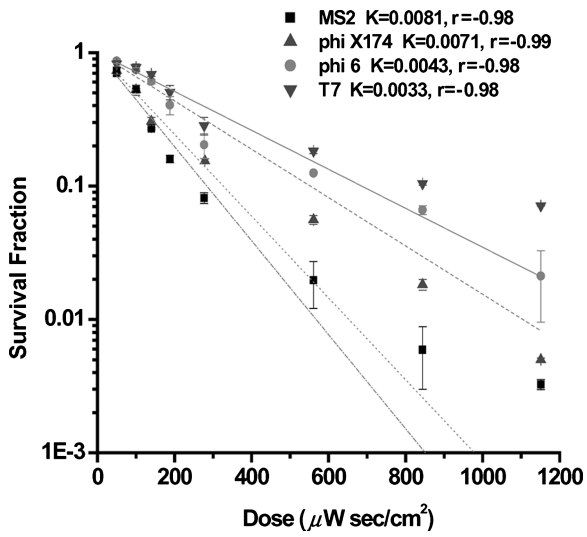

FIG. 2. Survival fraction of airborne viruses (MS2, phi X174, phi 6, and T7) exposed to UVGI at RH 55%. Error bars represent one standard deviation of the mean of at least three trials.

of 1906–2005 μW sec/cm^2. Similar to the results for 90% inactivation, these results indicate that the UVGI dose for 99% inactivation of dsRNA and dsDNA viruses is approximately 2 times higher than that of ssRNA and ssDNA viruses ($p < 0.05$). From the previous studies (Wells 1955; Kundsin 1968; David et al. 1973; Keller 1982; Mongold 1992; Lin and Li 2002), the UVGI doses for the four evaluated viruses in this study are similar to those reported for airborne *E. coli* (10^3 μW s/cm^2), but significantly lower than those for gram positive or negative bacteria (10^2–10^5 μW sec/cm^2), fungi (10^4–10^5 μW sec/cm^2), and spore type microorganisms (10^4 μW sec/cm^2). On the other hand, the American Conference of Governmental Industrial Hygienists

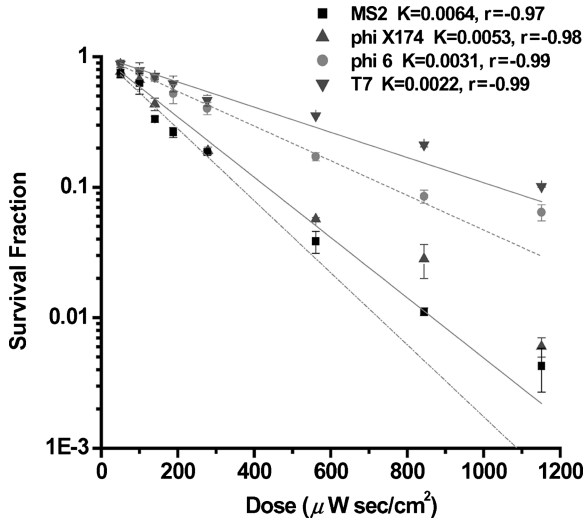

FIG. 3. Survival fraction of airborne viruses (MS2, phi X174, phi 6, and T7) exposed to UVGI at RH 85%. Error bars represent one standard deviation of the mean of at least three trials.

currently recommends that UV irradiance in the room should be less than 0.2 μW/cm^2 for 8 h (ACGIH, 1999). Therefore, our data demonstrated that the recommended UVGI dose could effectively inactivate airborne virus.

For all four types of viruses evaluated 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/μWsec), which is a commonly used indicator of the sensitivity of the test microorganism, varied widely. MS2 showed the highest K (0.0064–0.0081) and T7 the lowest (0.0022–0.0033), indicating that dsDNA (T7) was the most resistant virus to UVGI inactivation. Our findings agreed well with those observed in the previous findings which indicated that the K values of MS2 and T7 were very close to those of Vaccinia virus (ssRNA, 0.0047) and Adenovirus (dsDNA, 0.0018), respectively (Jensen 1964).

The K of airborne viruses studied here ranged from 0.0022 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* (0.00039–0.00050), 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.

For the four types of viruses tested here, K values (0.0022–0.0064) at 85% RH were lower than those (0.0033–0.0081) at 55% RH (Figures 2 and 3), which indicated that a higher UVGI dose was required to inactivate virus at higher RH conditions in this study ($p < 0.05$). Moreover, the RH effects to UV effectiveness were also related to the type of virus nucleic acid. The RH effects to UVGI inactivation of ssRNA and ssDNA airborne viruses were greater than those of dsDNA and dsRNA ones. Our current virus observations agreed with our previous one which indicated that microorganism susceptibilities at 80% RH were lower than those found at 50% RH for fragile bacteria (*E. coli*), endospore bacteria (*B. subtilis*), fungi (yeast), and fungi spore (*P. citrinum*) (Lin and Li 2002). In this study, the generated virus-containing aerosols are in droplet phase which have water sorption onto a virus surface to provide protection against UV-induced DNA damage (Peccia and Hernandez 2001). If virus-containing aerosols evaporate water vapor and approach the size of virus itself, the resistance to UV may become lower because of the lack of water shielding.

Up to now, there are very limited data available on the inactivation of airborne viruses by UVGI. However, UVGI inactivation of viruses in liquid suspension has been well characterized. It was demonstrated that complex nucleic acid, capsid structure, and host cell repair mechanisms of viruses could affect the UVGI effectiveness, as well as viruses with double-stranded genomes are less susceptible than single-stranded ones to UVGI (Thurston-Enriquez et al. 2003; Kallenbach et al. 1989). On the other hand, host cell repair mechanisms and capsid structure of virus were found to play important role (Thurston-Enriquez et al. 2003). For host cell repair mechanisms, 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 (Thurston-Enriquez et al. 2003). Regarding capsid structure, it directly packaged and associated with virus nucleic acid, therefore, shielding or consumption of UVGI before reaching the nucleic acid may occur.

In suspension, MS2 is a suggested indicator of viral inactivation by UVGI, because it had high resistance to UVGI than other ssRNA viruses (feline calicivirus, Ecovirus, Coxsackie virus, and poliovirus) or dsDNA virus (PRD1). In airborne phase in this current study, MS2 aerosol was observed to be more susceptible to UVGI than those of phi 6 and T7 aerosols. For both phi 6 and T7, complex nucleic acids (double-stranded genomes) could make these two phages use the host cell enzymes to repair damages. Moreover, T7 capsid consists of 415 molecules capsid protein (Bamford et al. 2002), whereas the MS2 only with 180 ones. Therefore, T7 capsid could provide more protection to UVGI.

In summary, our current results demonstrated that effectiveness of UVGI on airborne virus is related to the UV intensity and exposure time. In this study, UVGI inactivation of ssRNA and ssDNA viruses was easier than that of dsRNA and dsDNA viruses. In addition, viruses could be protected from the UV light inactivation by a complex nucleic acid, by strong capsid structures and by host cell repair mechanisms (Thurston-Enriquez et al. 2003). For all four viruses evaluated here, the survival fraction at 85% RH was higher than that at 55% RH. Finally, the UVGI can inactivate airborne viruses effectively.

CONCLUSION

The effect of UV dose, type of virus nucleic acid, and RH on the effectiveness of UVGI to inactivate airborne viruses was evaluated using in a laboratory test chamber. For airborne virus inactivation, the effectiveness of UVGI strongly depended on the type of virus nucleic acid. In this study, viruses with dsRNA or dsDNA are significantly less susceptible to UV inactivation. For 90% airborne virus inactivation, the UVGI dose for dsRNA and dsDNA viruses was approximately 2 times higher than ssRNA and ssDNA viruses, respectively. The microorganism susceptibility factor was the highest for the viruses, similar to that for fragile bacteria, but 13–20 times higher than that for endospore bacteria or fungal spores. The susceptibility factor for the viruses was higher at 55% RH than that at 85% RH, possibly because when RH is increased, water sorption onto the virus surface might provide protection against UV-induced DNA or RNA damage.

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