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Effect of temperature and relative humidity on ultraviolet (UV₂₅₄) inactivation of airborne porcine respiratory and reproductive syndrome virus

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ABSTRACT

The objective of this research was to estimate the effects of temperature and relative humidity on the inactivation of airborne porcine reproductive and respiratory syndrome (PRRS) virus by ultraviolet light (UV₂₅₄). Aerosols of PRRS virus were exposed to one of four doses of UV₂₅₄ under nine combinations of temperature ($n=3$) and relative humidity ($n=3$). Inactivation constants (k), defined as the absolute value of the slope of the linear relationship between the survival fraction of the microbial population and the UV₂₅₄ exposure dose, were estimated using the random coefficient model. The associated UV₂₅₄ half-life dose for each combination of environmental factors was determined as $(\log_{10} 2/k)$ and expressed as UV₂₅₄ mJ per unit volume. The effects of UV₂₅₄ dose, temperature, and relative humidity were all statistically significant, as were the interactions between UV₂₅₄ dose \times temperature and UV₂₅₄ dose \times relative humidity. PRRS virus was more susceptible to ultraviolet as temperature decreased; most susceptible to ultraviolet inactivation at relative humidity between 25% and 79%, less susceptible at relative humidity $\leq 24\%$, and least susceptible at $\geq 80\%$ relative humidity. The current study allows for calculating the dose of UV₂₅₄ required to inactivate airborne PRRS virus under various laboratory and field conditions using the inactivation constants and UV₂₅₄ half-life doses reported therein.

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1. Introduction

Initially described in the late 1980s, porcine reproductive and respiratory syndrome (PRRS) is characterized by reproductive failure in sows, poor growth performance in growing pigs, and respiratory disease in pigs of all ages (Zimmerman et al., 2012). Since its emergence as a clinical entity in the late 1980s, PRRS virus has proven to be a persistent threat to the health and productivity of pig

herds and the economic well-being of pig producers. Neumann et al. (2005) estimated the annual cost of PRRS to U.S. pig producers at \$560.32 million per year. By comparison, prior to eradication, annual losses in the U.S. to classical swine fever (hog cholera) and pseudorabies virus were estimated at \$364.09 million (Wise, 1981) and \$36.27 million (Hallam et al., 1987), respectively (adjusted to year 2004 dollars).

Since the beginning of the PRRS virus pandemic in the 1980s, movement of the virus between neighboring herds in the apparent absence of direct contact (“area spread”) has been often reported (Robertson, 1991). Several epidemiological investigations showed that proximity to infected herds increased the risk of a herd acquiring PRRS virus. In France, Le Potier et al. (1997) found that 45% of herds suspected to have become infected through area

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spread were located within 500 m (0.3 miles) of the postulated source herd and only 2% were 1 km from the initial outbreak. In Denmark it was observed that the likelihood of herd positivity increased as the density and proximity of PRRS virus-positive neighboring herds increased (Mortensen et al., 2002).

In the last decade, researchers have confirmed the occurrence of airborne transmission of PRRS virus over significant distances. Initially, Dee et al. (2005b) demonstrated that infectious airborne PRRS virus could travel over distances ≥ 150 m. Thereafter, using a source population of 300 PRRS virus-infected pigs, Dee et al. (2009) demonstrated long-distance airborne movement by the successful recovery of infectious virus up to 4.7 km from the source. Subsequently, Otake et al. (2010) recovered infectious airborne PRRS virus at distance of 9.1 km from the source. Cumulatively, the epidemiological and experimental data suggest that airborne PRRS virus is a significant, and perhaps the primary, means of area spread.

Protection of pig barns from airborne spread of infectious agents is a recent concept. To date, researchers have primarily focused on preventing the introduction of airborne PRRS virus into barns using commercially available air filters, e.g., high-efficiency particulate air (HEPA) filters, minimum efficiency rating value (MERV) filters of various efficiencies, and fiberglass pre-filters. Overall, the results demonstrated that filtering incoming air with HEPA filters and MERV filters ≥ 14 prevented the transmission of PRRS virus (Dee et al., 2005, 2006a,b, 2010).

Ultraviolet inactivation of PRRS virus may also offer promise. Wheeler et al. (1945) reported the use of ultraviolet to inactivate airborne rubella virus and *Streptococcus pyogenes* in Army and Navy barracks. Likewise, Perkins et al. (1947) reduced the spread of airborne viral pathogens (“measles”) in school classrooms using ultraviolet. Riley (1961) demonstrated that ventilated air from hospital tuberculosis wards produced tuberculosis in guinea pigs, but not when the air was irradiated with ultraviolet light. In recent years, UV₂₅₄ emitters have been engineered into areas where people congregate either by placing UV₂₅₄ light tube grids into existing ventilation ductwork or by installing free standing UVC emitters (Brickner et al., 2003; Dumyuhn and First, 1999; McDevitt et al., 2008; Menzies et al., 1999; Noakes et al., 2006). Ultraviolet technology is appealing due to its low cost as compared to HEPA filtration (Brickner et al., 2003). However, effective implementation must be based on achieving a dose of UV₂₅₄ sufficient to inactivate the target. No estimates of the effect of UV₂₅₄ on airborne PRRS virus have been published. Therefore, the objective of this experiment was to evaluate the capability of ultraviolet (UV₂₅₄) to inactivate airborne PRRS virus under varying conditions of temperature and relative humidity.

2. Materials and methods

2.1. Experimental design

The objective of this study was to evaluate the effect of temperature and relative humidity on the inactivation of

Table 1

Temperature and relative humidity combinations of PRRS virus aerosols.

Temperature	Relative humidity		
	$\leq 24\%$	25–79%	$\geq 80\%$
$\leq 15^\circ\text{C}$	Airborne PRRS virus received 4 levels of UV ₂₅₄ treatment at each combination of temperature and relative humidity. Each combination was replicated 3 times.		
16–29 °C			
$\geq 30^\circ\text{C}$			

airborne PRRS virus by ultraviolet irradiation (UV₂₅₄). Aerosols of PRRS virus were exposed to four levels of UV₂₅₄ under nine defined conditions of temperature and relative humidity (Table 1). Each combination of temperature and relative humidity was replicated 3 times. Samples of air collected after UV₂₅₄ treatment were titrated for infectious PRRS virus and the data used to calculate the UV₂₅₄ inactivation constants (*k*) and UV₂₅₄ half-life (*T* 1/2) exposure doses for each combination of temperature and relative humidity.

2.2. Porcine reproductive and respiratory syndrome virus

A type 2 PRRS virus isolate, MN-184 (kindly provided by Dr. Scott Dee, University of Minnesota, MN, USA) was propagated on MARC-145 cells, a clone of the African monkey kidney cell line MA-104 (Kim et al., 1993). Cells were grown in 162 cm² flasks (Corning Incorporated, Corning, NY, USA) using growth media; Dulbecco's Modified Eagles Medium (DMEM), (Mediatech Inc., Manassas, VA, USA) supplemented with 0.25 µg/ml Amphotericin B (Sigma Chemical Co., St. Louis, MO, USA), 50 µg/ml gentamicin (Sigma), 0.5 M L-glutamine (Fisher Scientific, Hampton, NH, USA), 300 international units (IU) per ml penicillin (Sigma), 300 µg/ml streptomycin (Sigma), 1.0% nonessential amino acids (HyClone, Logan, UT, USA), 25 mM HEPES buffer (Sigma Chemical Co.) and 10.0% heat-inactivated fetal bovine serum (FBS) (Sigma Chemical Co.). When cells were confluent (72–84 h), the medium was discarded and the flasks inoculated with 5 ml DMEM (without L-glutamine) containing PRRS virus isolate MN-184 at a virus titer of $1 \times 10^{3.5}$ median tissue culture infective dose (TCID₅₀) per ml. Flasks were placed on a rocking platform in a 37 °C humidified 5% CO₂ incubator for 90 min and then 40 ml of supplemented DMEM (Mediatech Inc.) growth medium (now with 4% FBS) was added and the flasks returned to the incubator for 36 h. Cell culture supernatant was harvested by flask freeze-thaw and centrifugation (3000 × *g* for 20 min at 4 °C). Virus stock was stored in 25 ml aliquots at –80 °C.

2.3. Experimental procedures

2.3.1. Overview

The system was constructed such that, throughout the 45 min experiment, aerosolized PRRS virus continuously flowed from Reservoir One to Reservoir Two and then across a UV₂₅₄ exposure field. A manifold in Reservoir Two functioned to equally distribute aerosolized PRRS virus into four quartz tubes placed parallel to each other in the

field of ultraviolet irradiation. Each quartz tube represented a different level of UV₂₅₄ treatment.

Airflow through the system was impelled by negative pressure generated by four AGI-30 glass impingers (Ace Glass, Vineland, NJ, USA), each operating at a flow rate of 12.5 l/min. Thus, air flow through the system totaled 50 l/min. Temperatures in Reservoir One and Reservoir Two were adjusted to achieve targeted temperatures and relative humidities at the UV₂₅₄ irradiation field. Air samples collected by the impingers downstream of the UV₂₅₄ exposure field were titrated for infectious PRRS virus.

2.3.2. Aerosolized PRRS virus

The virus suspension to be aerosolized consisted of 25 ml of stock PRRS virus (1×10^7 TCID₅₀/ml), 50 ml of sterile $1 \times$ PBS (Thermo Fisher, Rockford IL, USA), and 0.1% (v/v) antifoam A emulsion (Sigma Chemical Co., A5758). Previous research showed that antifoam A emulsion innocuous for cultured cells and PRRS virus (Hermann et al., 2006). The virus suspension was maintained on ice and shielded from light until nebulization. The solution was aerosolized using a 24-jet Collision nebulizer (BGI Inc., CN60, Waltham, MA, USA) operating on compressed air at 1.55 kg/cm² (22 psi). Under these parameters approximately 1.0 ml of virus solution was nebulized each minute into particles of approximately 1.9 μm diameter (Hermann et al., 2007).

2.3.3. Relative humidity and temperature

For each 45 min replicate, virus was continuously nebulized into Reservoir One (16 l; aerosol residence time ~19 s). Reservoir One was housed in a chamber capable of maintaining temperatures between -20 °C and room temperature (Carroll Coolers Inc., Carroll, IA). Air was drawn from Reservoir One into Reservoir Two (13 l; aerosol residence time ~16 s). Reservoir Two was housed in a chamber capable of maintaining temperatures between 0 °C and 40 °C (SS Series 600, 1695-03-36231, Terra Universal Inc., Anaheim, CA, USA).

Based on the rate of nebulization (1.0 ml/min at 1.55 kg/cm) and the temperature capabilities of Reservoirs One and Two, psychrometric calculations determined that the system was theoretically capable of producing an air flow with the RH (%) values listed in Table 2, with temperature at the UV₂₅₄ field of exposure approximating the temperature in Reservoir Two.

For each replicate, the actual temperature and relative humidity of the aerosol was measured (Vaisala, HMI41

indicator and HMP46 temperature probe, Helsinki, Finland) inside a manifold located within Reservoir Two and immediately upstream of the UV₂₅₄ exposure field. Temperature and relative humidity measurements were taken prior to starting the replicate and then at the beginning, midpoint and end of each replicate to confirm that the system operated at targeted parameters. All targeted (Table 1) combinations of temperature ($n = 3$) and relative humidity ($n = 3$) were conducted in triplicate.

2.3.4. Ultraviolet (UV₂₅₄) field of exposure

The UV₂₅₄ emitting apparatus consisted of six low-pressure, mercury-vapor discharge lamps 28.8 cm in length (American UV Company, Lebanon, IN, USA). Lamps were mounted in three 2-lamp, reflective, surface-mounted, 110 V, fixtures (American UV Company). To avoid fluctuation in UV₂₅₄ intensity, lamps were operated at their maximum UV₂₅₄ emission capacity for 10 min before the start of each experiment. UV₂₅₄ emission was measured using three calibrated radiometers (Model 1700, International Light Inc., Newburyport, MA, USA; VLX3W Technika, Phoenix, AZ, USA).

Exposure of airborne PRRS virus to UV₂₅₄ was done by passing the airborne virus through 4 quartz tubes (10 mm internal diameter \times 12.75 external diameter \times 14 cm in length) connected to a manifold within Reservoir Two. Quartz tubes were placed 25 cm from, and parallel to, the UV₂₅₄ emitters. Between each replicate, quartz tubes were cleaned with commercial quartz cleaner following the manufacturer's recommendations (Hellmanex[®] II, Hellma GmbH & Co., Mülheim, Germany). Quartz tubing was evaluated for cleanliness and UV₂₅₄ absorbance prior to each replicate by measuring UV₂₅₄ intensity beneath and beside the tubing with the impingers in operation (mock aerosol). The UV₂₅₄ exposure dose measured directly below the quartz tubing.

2.3.5. Ultraviolet (UV₂₅₄) treatment of PRRS virus aerosol

Each of the four quartz tubes delivered a different UV₂₅₄ treatment. This was achieved by shielding all but a specific length of each tube, i.e., 1.3 cm (residence time of 0.07 s); 3.2 cm (residence time of 0.14 s); and 5.2 cm (residence time of 0.25 s). A completely shielded tube served as a non-exposed (positive) control. Treatment (shielding) was randomized to quartz tubes before each replicate. UV₂₅₄ irradiance was measured at the unshielded area of each quartz tube at the start, midpoint, and end of each replicate

Table 2
Calculated relative humidity at the UV₂₅₄ field of exposure.^a

	Reservoir One (°C)										Reservoir Two (°C)
	-20	-15	-10	0	5	10	15	20	25	30	
Relative humidity (%)	20	31	47	100	-	-	-	-	-	-	0
	14	22	32	70	100	-	-	-	-	-	5
	10	16	23	50	71	100	-	-	-	-	10
	7	11	17	36	50	72	100	-	-	-	15
	5	8	13	26	37	53	73	100	-	-	20
	4	6	9	20	28	38	54	74	100	-	25
	3	5	7	14	21	30	40	55	75	100	30

^a Air temperature approximated by temperature of Reservoir Two.

and averaged. For each treatment, the UV₂₅₄ dose delivered to airborne PRRS virus was calculated as:

$$D = I \times T$$

where D is the ultraviolet dose (mJ/cm²), I is irradiance (mW/cm²), and T is residence time(s). I was calculated as the average of the irradiance measured at the start, midpoint, and end of each replicate.

Based on the parameters described above, the mean UV₂₅₄ exposure doses across all replicates for the four treatments were calculated as: zero for the non-exposed (positive) control, 0.05 (S.D. 0.009) mJ/cm²; 0.12 (S.D. 0.016) mJ/cm², and 0.20 (0.039) mJ/cm².

2.3.6. Sampling of UV₂₅₄-treated airborne PRRS virus

Each quartz tube was independently connected to an all-glass impinger (AGI-30, Ace Glass Inc., Vineland, NJ, USA) containing 25 ml of 1× PBS and shielded against ultraviolet. Impingers were placed on ice throughout the 45 min experiment to avoid desiccation of PBS and preserve virus viability. Impingers were operated at a constant flow rate of 12.5 l/min. Vacuum pressure was maintained using oil-less pumps (Fisher Scientific, S413801, Hampton, NH). All connections were sealed and checked for leakage prior to each replicate. Air from the system was exhausted through a biosafety level 2 (BL2) cabinet (NuAire Laboratory Equipment Supply, Plymouth, MN, USA).

2.3.7. PRRS virus microinfectivity assay (TCID₅₀)

Impinger collection fluid was assayed for the concentration of infectious PRRS virus immediately following each replicate. Virus titrations were done on confluent monolayers of MARC-145 cells in 96-well plates (Corning Incorporated). Cell monolayers were prepared by inoculating 100 μl of cells suspended in growth medium into each well, and then incubating the plates at 37 °C in a humidified 5% CO₂ incubator for 24 h. Each sample was serially 10-fold diluted in DMEM, with five wells were inoculated with 100 μl of each sample dilution. Thereafter, plates were incubated at 37 °C in a humidified 5% CO₂ incubator for 2 h, after which the inoculum was discarded and 100 μl per well of DMEM supplemented with 4% FBS was added. Plates were incubated at 37 °C in a humidified

5% CO₂ incubator for 24 h, after which the cells were fixed with aqueous 80% acetone solution and stained with a fluorescein isothiocyanate-conjugated monoclonal antibody specific for PRRS virus (SDOW17, Rural Technologies Inc., Brookings, SD, USA). Virus titers were calculated using the Spearman–Kärber method on the basis of the number of wells showing PRRS virus-specific fluorescence at each dilution and expressed as tissue culture infection dose 50 (TCID₅₀)/ml of impinger fluid.

2.3.8. Statistical analysis

To study the main effects (UV₂₅₄ dose, temperature, and relative humidity) and their interactions, the TCID₅₀ data were log₁₀-transformed and analyzed using a random-coefficient ANCOVA (analysis of covariance) model with the quantitative explanatory variable “UV₂₅₄ dose” and categorical explanatory variables of “temperature” and “relative humidity” in SAS[®] Version 9.2 (SAS[®] Institute Inc., Cary, NC, USA). Inactivation constants (k), defined as the absolute value of the slope describing the linear relationship between the survival fraction of the microbial population and the UV₂₅₄ exposure dose (Goldberg et al., 1958), were estimated using the random coefficient model. The associated UV₂₅₄ half-life ($T_{1/2}$) dose for each combination of environmental factors was determined as $(\log_{10} 2/k)$ and expressed as UV₂₅₄ mJ per unit volume.

3. Results

The effects of UV₂₅₄ dose, temperature, and relative humidity on the concentration of PRRS virus (TCID₅₀) recovered in air samples were all statistically significant ($p < 0.001$), as were the interactions between UV₂₅₄ dose × temperature ($p = 0.0475$), and UV₂₅₄ dose × relative relative humidity ($p = 0.0204$). Inactivation constant estimates and UV₂₅₄ half-life dose estimates are given in Table 3. Comparisons among the three temperature groups detected a significant difference in the UV₂₅₄ inactivation constant for PRRS virus at low vs. high temperatures ($p = 0.0167$), but not between low vs. medium ($p = 0.1044$) or medium vs. high temperatures ($p = 0.4635$). Comparisons among the three levels of relative humidity revealed a statistically significant difference in inactivation constants between medium vs. high relative humidity

Table 3
Inactivation constants and UV₂₅₄ half-life doses by temperature and relative humidity.

		Relative Humidity			
		≤ 24%	25% to 79%	≥ 80%	
Temperature		Main effects	4.25 (0.071)*	5.87 (0.051)	3.41 (0.088)
	≤ 15°C	5.68 (0.053)	5.42 (0.055)	7.04 (0.043)	4.58 (0.070)
	16°C to 29°C	4.25 (0.071)	3.99 (0.075)	5.61 (0.054)	3.15 (0.096)
	≥ 30°C	3.59 (0.084)	3.33 (0.090)	4.96 (0.061)	2.49 (0.121)

*Inactivation constant and (half-life). Inactivation constant (k) is the absolute value of the slope of the PRRS virus survival fraction and the UV₂₅₄ exposure dose. Larger k values indicate more rapid PRRS virus inactivation. Half-life dose is expressed as UV₂₅₄ mJ per unit volume.

($p = 0.0060$), but not between and low vs. medium ($p = 0.0700$) or low vs. high relative humidity ($p = 0.3291$).

4. Discussion

In this study, UV_{254} inactivation constants for PRRS virus were derived for three temperature ranges, three relative humidity ranges, and their combinations. The effects of temperature and relative humidity on the UV_{254} inactivation of PRRS virus were statistically significant, but the interaction of temperature and relative humidity was not. Ultraviolet inactivation constants decreased in a linear fashion as temperature increased, with statistically significant differences in inactivation constants detected in low vs. high temperatures, but not between low vs. medium or medium vs. high temperatures. The effect of relative humidity on UV_{254} inactivation was more complex. For any temperature, the rate of PRRS virus inactivation was highest at relative humidity between 25% and 79% and lowest at relative humidity $\geq 80\%$. These observations were reflected in statistically significant differences in inactivation constants in medium vs. high relative humidity, but not between other comparisons.

A search of the refereed literature found no publications describing UV_{254} inactivation of airborne viruses under varying conditions of relative humidity and temperature and only three publications on UV_{254} inactivation of airborne viruses under varying conditions of relative humidity. In agreement with the results of this study, Tseng and Li (2005) reported that UV_{254} inactivation of four bacteriophages (MS2, phi X174, phi 6, T7) decreased as relative humidity increased at temperatures of 25 °C to 28 °C and speculated that decreased UV_{254} susceptibility under higher relative humidity conditions resulted from attenuation of UV_{254} by water sorption onto the viral surface. In contrast, in a study involving bacteriophage MS2, respiratory adenovirus serotype 2, and mouse hepatitis virus (coronavirus), Walker and Ko (2007) reported that UV_{254} inactivation increased as relative humidity increased (temperature conditions not reported). This same general trend was reported by McDevitt et al. (2007) who stated there was an increase in UV_{254} susceptibility with an increase in relative humidity (temperature conditions not reported). Walker and Ko (2007) hypothesized that increased UV_{254} susceptibility at higher relative humidity could have been a function of larger droplet size at higher relative humidities. Given the overall paucity of data, fruitful hypothesis generation regarding the mechanisms underlying the interactions between UV_{254} inactivation, temperature, and relative humidity must await additional data on the ultraviolet inactivation of a greater diversity of micro-organisms.

The current study allows for calculating the dose of UV_{254} required to inactivate airborne PRRS virus under various laboratory and field conditions using the inactivation constants and their associated UV_{254} half-life doses from Table 3. Since inactivation constants vary by temperature and relative humidity, a conservative estimate of the necessary UV_{254} dose can be made using the smallest inactivation constant ($k = 2.49$, Table 3) and its associated UV_{254} half-life dose. The percent of the

infectious viral population remaining after n UV_{254} half-life doses may be described as $(1/2^n)$; thus, the percent of infectious PRRS virus remaining after 10 UV_{254} doses may be calculated as $(1/2^{10}) = 0.1\%$. From this, the dose of UV_{254} required to inactivate 99.9% of airborne PRRS virus may be calculated using the value from Table 3 as $(0.121 \text{ mJ/area}^2 / \text{half-life}) \times (10 \text{ half-lives}) = 1.21 \text{ mJ/area}^2$. This exposure dose may be achieved under different conditions of ultraviolet intensity and airflow. As given by the Bunsen–Roscoe Law of Reciprocity, if achieved, this dose will be effective regardless of UV_{254} intensity, distance, or residence time (Riley and Kaufman, 1972).

Conflict of interest statement

The authors declare no conflict of interest.

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