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Inactivation of Human Rhinovirus due to Heat, UV Irradiation and Chemical Disinfectants

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Abstract

To evaluate and understand inactivation of HRV under many physical conditions and chemical agents, HRV86 were selected to expose with temperature, ultraviolet light (UV), Sodium hypochlorite, Virkon S, Peracetic acid (PAA), Glutaraldehyde and Ethanolin, respectively. The inactivation of HRV was analyzed by infectivity of the viral strains on the HeLa cells. Our research found the rhinovirus was very sensitive to temperature changes. Viral infectivity thoroughly lost after HRV86 was treated at 60°C for 10 min or UV irradiation for 45 min or longer. Virus also was completely inactivated after exposure to sodium hypochlorite (0.1 g/L) beyond 10 min, glutaraldehyde (10 g/L) for 5 min, Virkon-S (5 g/L) for 10 min, PAA (3 g/L) for 2 min, or 75% alcohol for 5 min or longer. The results provided the essential information for prevention and intervention of common cold.

Keywords: Chemical disinfectants; Rhinovirus; Inactivation; Thermal; UV irradiation

Introduction

Human Rhinovirus (HRV) is a major pathogeny associated with the common cold and is responsible for induction or exacerbation of asthma [1]. In recent years, studies have reported that HRV infection in early childhood markedly increased risk of wheezing and developing asthma [2-4]. The magnitude of this association appears larger than that of respiratory syncytial virus (RSV), the classic respiratory pathogen of infancy [5,6]. HRV infections could increase the severity of infection in patients with immunodeficiency diseases and abnormal immune function. HRV is highly transmissible, leading to high infection rates in the respiratory tract particularly in younger children and older men. This infection not only causes severe morbidity and mortality but also results in economic and social burden. The high prevalence of HRV infections and high association between HRV and asthma, severe upper respiratory tract infection, and bacterial super-infection underscores a need to prevent HRV infection and reduce its prevalence globally. However, HRV is highly heterogeneous and have high mutation rates, thus making it challenging to develop effective vaccines against these evolving viruses. Therefore, there is a need to identify effective measure for the immediate containment of the infection and prevention of potential future outbreaks. In addition to good sanitation and hygienic practices, effective disinfection strategies are also necessary. Understanding the potential effects of chemical and physical treatments on inactivation of HRV is significant for the implementation of proper public intervention measures. This study was designed to evaluate the effectiveness of various physical conditions (temperature, ultraviolet light (UV)) and chemical agents (sodium hypochlorite, PAA, Virkon-S, ethanol) against HRV.

Materials and Methods

Cell culture and HRV virus

HeLa cells were cultured in Dulbecco's modified Eagle's medium supplemented with 10% FCS, glutamine, and antibiotics as described previously [7]. HRV86 was propagated in HeLa cells with 2% serum for maintenance and infection. $2 \times 10^{-6.59}$/ml TCID50 HRV86 was used in this study.

Infectivity and inactivation of heat

To determine the effect of heat on infectivity, HRV86 viruses firstly were exposed to various temperatures. After 100 µl of viral stock solutions containing $1 \times TCID_{50}$, $10 \times TCID_{50}$, $100 \times TCID_{50}$ were incubated Room temperature, 37°C for 6 h, 12 h, 18 h, 24 h, 36 h, 48 h and 72 h. Treated virus was inoculated into HeLa cells respectively. For thermal inactivation, 100 µl of viral stock solutions containing $1 \times TCID_{50}$, $10 \times TCID_{50}$, $100 \times TCID_{50}$, $1000 \times TCID_{50}$ were respectively exposed at 60°C for 5 min, 10 min, 15 min, 30 min, 1 h and 2 h. The heat-inactivated HRV inoculated into HeLa cells. Then cytopathic effect (CPE) in HeLa cells was observed for 24 h after passaging three times.

UV inactivation

A 100 µl virus stock solution containing $1 \times TCID_{50}$, $10 \times TCID_{50}$, $100 \times TCID_{50}$, or $1000 \times TCID_{50}$ was exposed to UV light (distance of 30 cm and wavelength of 250-270 nm) in a biosafety cabinet (Thermo Company) at room temperature for 5, 10, 15, 20, 30, 45, 60, and 120 min, respectively. UV treated virus was inoculated into HeLa cells. CPE was observed as above described.

Disinfectants inactivation

Commercial disinfectants, including sodium hypochlorite (Beijing Yasite LTD. China), peracetic acid (Xilong Chemical Co., Ltd. China), glutaraldehyde (Xilong Chemical Co., Ltd. China), Virkon-S (Antec™ International, UK), ethanol (Dezhou ANNJET disinfection products Co. Ltd. China) were assessed for their effectiveness in the inactivating HRV86. Briefly, all disinfectants were diluted with distilled water following the manufacturers' protocol. A 100 µl virus stock solution containing 100 × $TCID_{50}$ was absorbed into filter and exposed with above disinfectants, respectively for 2, 5, 10, 20 or 30 min at room temperature. Then filter were washed with 1 ml PBS for three times. Treated virus was inoculated into HeLa cells and CPE was observed as described.

Cytotoxicity

The cytotoxic effects of HRV after exposure to various disinfectants were assessed as previously described [8] except for the replacement of the virus by PBS.

Results

Heat inactivation

The infectivity of HRV86 after exposure to various environmental temperatures (RT (25 ± 2°C), 37°C, or 60°C) was determined. The effects of temperature on HRV inactivation were time-and titers-dependent. RT impact greatly on HRV virus infectivity of 1 × $TCID_{50}$. Although HRV was able to retained activation at RT for 24 h, its infectivity was decreased from 18 h when used at 1 × $TCID_{50}$. And, the virus was able to retained activation at RT for 48 h at both 10 × $TCID_{50}$ and 100 × $TCID_{50}$ titers (Table 1). However, we found that the validation of HRV was impaired severally at 37°C. Complete loss of viral infectivity at both 1 × $TCID_{50}$ titers and validation of HRV only maintained for 24 h in 100 × $TCID_{50}$ titers (1/4) (Table 1). Complete loss of viral infectivity was observed at 36 h in the 100 × $TCID_{50}$ titers when kept at 37°C. However, complete loss of viral infectivity were observed both 1 × $TCID_{50}$, 10 × $TCID_{50}$ titers and 100 × $TCID_{50}$ titers at 37°C after exposed for 6 h or longer.

Treat	RT			37°C			60°C		
Time	1 × $TCID_{50}$	10 × $TCID_{50}$	100 × $TCID_{50}$	1 × $TCID_{50}$	10 × $TCID_{50}$	100 × $TCID_{50}$	1 × $TCID_{50}$	10 × $TCID_{50}$	100 × $TCID_{50}$
06 h	4/4	4/4	4/4	0/4	2/4	4/4	0/4	0/4	0/4
12 h	4/4	4/4	4/4	0/4	2/4	2/4	0/4	0/4	0/4
18 h	3/4	4/4	4/4	0/4	1/4	2/4	0/4	0/4	0/4
24 h	1/4	4/4	4/4	0/4	0/4	1/4	0/4	0/4	0/4
36 h	0/4	3/4	3/4	0/4	0/4	0/4	0/4	0/4	0/4
48 h	0/4	2/4	1/4	0/4	0/4	0/4	0/4	0/4	0/4
60 h	0/4	0/4	0/4	0/4	0/4	0/4	0/4	0/4	0/4
72 h	0/4	0/4	0/4	0/4	0/4	0/4	0/4	0/4	0/4

Note: Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.

Table 1: Effect of temperatures on infectivity of HRV86.

Treat Time	1 × $TCID_{50}$	10 × $TCID_{50}$	100 × $TCID_{50}$	1000 × $TCID_{50}$
05 min	0/4	0/4	1/4	2/4
10 min	0/4	0/4	0/4	0/4
20 min	0/4	0/4	0/4	0/4
30 min	0/4	0/4	0/4	0/4
01 h	0/4	0/4	0/4	0/4
02 h	0/4	0/4	0/4	0/4

Note: Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.

Table 2: Heat inactivation of HRV86 with 60°C.

To find appropriately heat inactivation of viruses, exposure time was shorted within 2 h and virus titers were also increased to 1000 × $TCID_{50}$ (Table 2). It was observed that HRV86 was completely inactivated in the 1 × $TCID_{50}$ or 10 × $TCID_{50}$ titers when exposed at

60°C for 5 min or longer (Table 2). However, 100 × $TCID_{50}$ or 1000 × $TCID_{50}$ titers remain partial vitality when exposed at 60°C for 5 min, in which 2 of 4 wells and 1 of 4 wells CPE was respectively observed at 5 min in the 100 × $TCID_{50}$ or 1000×$TCID_{50}$ titers (Table 2). However, no CPE was observed when HRV was treated over 10 min at 60°C (Table 2). These results showed that HRV was completely inactivated at 60°C after exposure for 10 min.

UV inactivation

To examine the effects of continuous UV irradiation on HRV infectivity, 100 µl aliquots of HRV stock (1 × $TCID_{50}$, 10 × $TCID_{50}$, 100 × $TCID_{50}$, or 1000 × $TCID_{50}$) were exposed to UV light irradiation at different time points. After 1 × $TCID_{50}$, 10 × $TCID_{50}$, 100 × $TCID_{50}$, or 1000 × $TCID_{50}$ of HRV were treated with UV radiation for 5, 10, 15, 20, 30, 45, 60, and 120 min, infectivity of HRV decreased in a time-dependent manner. When 100 µl of 1 × $TCID_{50}$ HRV86 were exposed under UV radiation for 10 min, 2 of 4 wells appeared CPE (Table 3). HRV was completely inactivated by UV radiation for 15 min at the same titer. When 100 µl of 10 × $TCID_{50}$ HRV were exposed under UV radiation for 15 min or 20 min, 2 of 4 wells and 1 of 4 wells appeared CPE (Table 3), respectively. Additionally, at this titer, exposure of up to or over 30 min, HRV infectivity on cells lost thoroughly (Table 3). However, HRV titers at 100 × $TCID_{50}$ or 1000 × $TCID_{50}$ were completely inactivated by UV radiation after exposure for 45 min or longer (Table 3).

Treat Time	1 × $TCID_{50}$	10 × $TCID_{50}$	100 × $TCID_{50}$	1000 × $TCID_{50}$
05 min	4/4	4/4	4/4	4/4
10 min	2/4	4/4	4/4	4/4
15 min	0/4	2/4	2/4	4/4
20 min	0/4	1/4	2/4	4/4
30 min	0/4	0/4	1/4	2/4
45 min	0/4	0/4	0/4	0/4
Note: Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.				

Table 3: Inactivation of HRV86 with UV irradiation.

Treat time	Sodium hypochlorite			
	0.5 g/L	0.25 g/L	0.2 g/L	0.1 g/L
02 min	0/4	0/4	0/4	2/4
10 min	0/4	0/4	0/4	0/4
20 min	0/4	0/4	0/4	0/4
30 min	0/4	0/4	0/4	0/4
Note: Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.				

Table 4: Inactivation of HRV86 by Sodium hypochlorite.

Disinfectants inactivation

To investigate the resistance of the HRV viruses against various disinfectants, 100 µl of stock solutions containing 100 × $TCID_{50}$ HRV virus titers were treated with sodium hypochlorite, glutaraldehyde, peracetic acid, Virkon-S, or 75% ethanol at room temperature.

Inactivation by sodium hypochlorite

Sodium hypochlorite is a common disinfectant. To determine the effects of sodium hypochlorite on the inactivation of HRV, aliquots of HRV stock were treated with 0.1 g/L, 0.2 g/L, 0.25 g/L, or 0.5 g/L of sodium hypochlorite for 2, 10, 20, or 30 min, respectively. Exposure of HRV to 0.1 g/L sodium hypochlorite beyond 10 min completely inactivated HRV (Table 4). Over a concentration of 0.2 g/L, sodium hypochlorite sufficiently inactivated HRV by 2 min (Table 4). These findings suggest that sodium hypochlorite at 0.2 g/L is suitable for use against the most etiologic agents of common cold. Based on these results, treatment of contaminated surfaces with sodium hypochlorite may reduce infection of contaminated surfaces and thereby reduce the risk of laboratory transmission during outbreaks.

Virkon inactivation

Virkon-S was next used at 1 g/L, 5 g/L, 10 g/L, 15 g/L, 20 g/L for 5, 10, 20 and 30 min to assess its effects on HRV infectivity. At 1 g/L, Virkon-S led to partial inactivation of HRV, as CPE appeared in only 1 of 4 wells after 30 min (Table 5). Similar results were observed when used at 5 g/L for 5 min (Table 5).

Treat time	Virkon-S				
	1 g/L	5 g/L	10 g/L	15 g/L	20 g/L
05 min	3/4	2/4	0/4	0/4	0/4
10 min	3/4	0/4	0/4	0/4	0/4
20 min	2/4	0/4	0/4	0/4	0/4
30 min	1/4	0/4	0/4	0/4	0/4
Note. Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.					

Table 5: Inactivation of HRV86 by Virkon-S.

Treat time	Peracetic acid			
	3 g/L	1.5 g/L	0.75 g/L	0.375 g/L
02 min	0/4	1/4	3/4	4/4
10 min	0/4	1/4	2/4	4/4
20 min	0/4	1/4	1/4	1/4
30 min	0/4	0/4	0/4	0/4
Note. Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.				

Table 6: Inactivation of HRV86 by Peracetic acid.

However, prolong treatment time for over 10 min, HRV infectivity on cells was complete lost (Table 5). Similar results were also observed when used at 10 g/L or more and exposed for 5 min or longer.

Glutaraldehyde inactivation

Glutaraldehyde is popularly used in hospitals, which is able to effectively eliminate many types of bacteria, fungi, and bacillus. To further understand the effects of this disinfectant on HRV infectivity, 100 × $TCID_{50}$ of HRV86 was treated with 10 g/L, 20 g/L, 50 g/L, or 100 g/L glutaraldehyde for 5, 10, 20, or 30 min, respectively. In all experiments, no residual infectivity was observed until the third passage. These results indicated that effective viral inactivation was achieved, and that glutaraldehyde is an effective disinfectant to inactivate HRV.

Peracetc acid inactivation

According to the US Center of Disease Control (CDC) "Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008" [9], peracetic acid (PAA) is described as a very effective virucide and sporocide. To understand the extent in which PAA inactivates HRV, aliquots of HRV viral stocks were treated with 3 g/L, 1.5 g/L, 0.75 g/L and 0.375 g/L diluted PPA and exposed for 2, 10, 20, or 30 min. These results showed that 100 × $TCID_{50}$ of the infectivity of HRV was completely loss when treated with 3 g/L diluted PAA for 2 min or longer (Table 6). However, it took 30 min for 1.5 g/L, 0.75 g/L and 0.375 g/L diluted PAA to completely inactivate HRV (Table 6). Thus, these results showed that 3g/L diluted PAA could effectively disinfectant HRV.

75% Alcohol inactivation

Because 75% alcohol is one of the most common disinfectant used in hospitals, households, schools, and other public areas, we next investigated the virucidal activities of 75% alcohol in HRV from 45 s to 30 min. As shown in Table 7, 75% alcohol did not inactivate HRV after treating for 45 s. There was some inhibition of HRV infectivity by 75% alcohol after exposure for 1 or 2 min (Table 7). However, complete inactivation of HRV was observed when treated for 5 min or longer.

Discussion

Heat treatment is one of widely used viral inactivation methods against viruses [10]. Heat-inactivated virus exhibits denatured viral proteins and dissociated viral particles into noninfectious viral subunits [11]. Many viruses, such as avian influenza A (H7N9), HCV, and EBOV have been shown to be sensitive to heat treatment [12-14]. In this study, an analysis was conducted on the inactivation of HRV at various environmental temperatures. Our results showed that HRV are very sensitive to temperature. HRV survival at RT were dependent on virus titers and exposure time. HRV infectivity reduced gradually upon longer exposure time and at lower viral titers (Table 1). Of note, the stability of HRV was severely impaired at 37°C (Table 1). It is possible that HRV virus may have a different structure when incubated at 37 °C and at room temperature just like Dengue virus [15]. Our results suggest that survival of HRV are very fragile in the environment. In this current study, exposure for 5 min at 60°C was sufficient to eliminate the infectivity of HRV86 in all 4 viral titers (Table 2). Therefore, disinfection and inactivation of daily necessities in the cold

season for 5 min at 60°C could eliminate HRV infectivity and effectively prevent colds caused by rhinovirus.

UV light irradiation is another commonly used physical method for viral inactivation. UV irradiation prevents viral replication and inactivates virus through formation of pyrimidine dimers in the viral genome [12,14,16-18]. This study demonstrated that the efficiency of UV irradiation to completely inactivate HRV was dependent on exposure time and virus titer (Table 3). Higher exposure time to UV irradiation was required to inactivate HRV at higher titers (Table 3). All 4 HRV titers were completely inactivated upon UV irradiation for 45 min. Therefore, UV irradiation is a highly effective method for inactivating HRV.

Treat time	CPE
45 s	4/4
01 min	3/4
02 min	2/4
05 min	0/4
10 min	0/4
20 min	0/4
Note: Results were showed that positive well numbers of CPE in each group (4 wells). For example, 1/4 meant that CPE was only observed in one of the 4 wells. 1 × $TCID_{50}$ meant that 100 µl virus stock solution contained 1 × $TCID_{50}$ virus in this study.	

Table 7: Inactivation of HRV86 by 75% alcohol.

The efficacy of several other commonly used disinfectants was also evaluated. Because PPA can inactivate virus through oxidizing S-H groups and N-H of viral proteins of the envelope and the capsid [19], this agent has been used worldwide during food processing and animal breeding, as well as in the beverage industry [9]. In this study, the virucidal activity of PAA against HRV occurred when using concentrations as low as 3 g/L for 2 min, demonstrating that HRV is more sensitive to PPA than Polio virus (0.2% PAA solution an exposure time of 5 min) [19]. However, it took about 30 min for 0.75 g/L diluted PPA or higher to inactivate HRV.

Sodium hypochlorite is one of the most widely used disinfectants. These agents has been successfully used during many infectious disease outbreaks including SARS, EBOV, and influenza H1N1 to decontaminate hospital instruments, infectious materials, and personal protective equipment [20-22] for health care professionals. In the current West African EVD outbreak, a 0.5% chlorine solution has been recommended to disinfect surfaces contaminated with EBOV, and is now recommended for disinfection by the World Health Organization [23]. Our results showed that HRV, compared with other known viruses, was more sensitive to sodium hypochlorite. We found that 0.2 g/L sodium hypochlorite could effectively inactivate HRV within 2 min of exposure. Therefore, sodium hypochlorite is effective in decontaminating surfaces in hospitals, as well as for using among health care professional for proper hand hygiene.

Glutaraldehyde is a chemical cross-linking reagent that is mostly used for tissue fixation for electron microscopy. Although its mechanism of action remain unclear, successful inactivation of many viruses with including hepatitis B virus, human immunodeficiency virus (HIV), hepatitis C virus, and SARS, coronavirus have been reported using glutaraldehyde. [14,16,24,25]. In this study, we demonstrated that 20 min of exposure to glutaraldehyde (10 g/L) could inactivate HRV infectivity on cells. Thus, 25 g/L glutaraldehyde could be used to effectively inactivate HRV.

Virkon-S has been previously reported to be effective for inactivating avian influenza A (H7N9) virus [12], enterovirus, and decreasing transmission of foot and mouth diseases [26,27]. When exposed to 10 g/L Virkon-S for 10 min, titer of feline herpesvirus was significantly reduced [28]. We showed that Virkon-S could completely inactivate HRV infection, consistent with the results of previous studies which used 5 g/L Virkon-S for 10 min or 10 g/L Virkon-S for 5 min or longer. Virkon-S was previously found to be effective against virus, however, the concentrations tested in this study were far higher than those previously employed [29,30]. Combined with previous studies, we think that Virkon-S could have effects against a broad-spectrum of viruses.

Although the Food and Drug Administration suggests ethanol concentrations should range from 60% to 95% [31], a previous study showed virucidal activity of ethanol concentrations which ranged between 58% to 75% [32]. In this study, 75% ethanol was used, as it was the concentration most commonly used in daily life, and at hospitals and laboratories. The results showed that 75% ethanol at 5 minutes effectively inactivated HRV. Additionally, we speculate that paper towel or cotton containing 75% ethanol was less effective in inactivating HRV.

Overall, our results provide effective methods for inactivating HRV, which could be implemented to control a HRV outbreak, reduced risk of transmission of HRV, decontaminate infected environments, or as preventative measures to improve public hygiene.

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References

1. Iikura M, Hojo M, Koketsu R, Watanabe S, Sato A, et al. (2015) The importance of bacterial and viral infections associated with adult asthma exacerbations in clinical practice. PloS one 10: e0123584.

2. Kotaniemi-Syrjänen A, Vainionpää R, Reijonen TM, Waris M, Korhonen K, et al. (2003) Rhinovirus-induced wheezing in infancy--the first sign of childhood asthma? The J Allergy Clin Immunol 111: 66-71.
3. Kusel MM, de Klerk NH, Kebadze T, Vohma V, Holt PG, et al. (2007) Early-life respiratory viral infections, atopic sensitization, and risk of subsequent development of persistent asthma. J Allergy Clin Immunol 119: 1105-1110.
4. Jackson DJ, Gangnon RE, Evans MD, Roberg KA, Anderson EL, et al. (2008) Wheezing rhinovirus illnesses in early life predict asthma development in high-risk children. Am J Respir Crit Care Med 178: 667-672.
5. Wu P, Dupont WD, Griffin MR, Carroll KN, Mitchel EF, et al. (2008) Evidence of a causal role of winter virus infection during infancy in early childhood asthma. Am J Respir Crit Care Med 178: 1123-1129.
6. Regnier SA, Huels J (2013) Association between respiratory syncytial virus hospitalizations in infants and respiratory sequelae: systematic review and meta-analysis. Pediatr Infect Dis J 32: 820-826.
7. Song QQ, Lu MZ, Song J, Chi MM, Sheng LJ, et al. (2015) Coxsackievirus B3 2A protease promotes encephalomyocarditis virus replication. Virus Res 208: 22-29.
8. de Oliveira TM, Rehfeld IS, Coelho Guedes MI, Ferreira JM, Kroon EG, et al. (2011) Susceptibility of Vaccinia virus to chemical disinfectants. Am J Trop Med Hyg 85: 152-157.
9. (2017) Healthcare Infection Control Practices Advisory Committee (HICPAC). CDC.
10. Burnouf T, Radosevich M (2000) Reducing the risk of infection from plasma products: specific preventative strategies. Blood Rev 14: 94-110.
11. Schlegel A, Immelmann A, Kempf C (2001) Virus inactivation of plasma-derived proteins by pasteurization in the presence of guanidine hydrochloride. Transfusion 41: 382-389.
12. Zou S, Guo J, Gao R, Dong L, Zhou J, et al. (2013) Inactivation of the novel avian influenza A (H7N9) virus under physical conditions or chemical agents treatment. Virol J 10: 289.
13. Remington KM, Trejo SR, Buczynski G, Li H, Osheroff WP, et al. (2004) Inactivation of West Nile virus, vaccinia virus and viral surrogates for relevant and emergent viral pathogens in plasma-derived products. Vox Sang 87: 10-18.
14. Song H, Li J, Shi S, Yan L, Zhuang H, et al. (2010) Thermal stability and inactivation of hepatitis C virus grown in cell culture. Virology J 7: 40.
15. Zhang X, Sheng J, Plevka P, Kuhn RJ, Diamond MS, et al. (2013) Dengue structure differs at the temperatures of its human and mosquito hosts. Proc Natl Acad Sci USA 110: 6795-6799.
16. Perdiz D, Grof P, Mezzina M, Nikaido O, Moustacchi E, et al. (2000) Distribution and repair of bipyrimidine photoproducts in solar UV-irradiated mammalian cells. Possible role of Dewar photoproducts in solar mutagenesis. J Biol Chem 275: 26732-26742.
17. Azar Daryany MK, Hosseini SM, Raie M, Fakharie J, Zareh A (2009) Study on continuous (254 nm) and pulsed UV (266 and 355 nm) lights on BVD virus inactivation and its effects on biological properties of fetal bovine serum. J Photochem Photobiol B 94: 120-124.
18. Smirnov YA, Kapitulets SP, Amitina NN, Ginevskaya VA, Kaverin NV (1991) Effect of UV-irradiation on rotavirus. Acta Virol 35: 1-6.
19. Wutzler P, Sauerbrei A (2000) Virucidal efficacy of a combination of 0.2% peracetic acid and 80% (v/v) ethanol (PAA-ethanol) as a potential hand disinfectant. J Hosp Infect 46: 304-308.
20. Lai MY, Cheng PK, Lim WW (2005) Survival of severe acute respiratory syndrome coronavirus. Clinical infectious diseases. Clin Infect Dis 41: e67-71.
21. Cook BW, Cutts TA, Nikiforuk AM, Poliquin PG, Court DA, et al. (2015) Evaluating environmental persistence and disinfection of the Ebola virus Makona variant. Viruses 7: 1975-1986.
22. Abe M, Kaneko K, Ueda A, Otsuka H, Shiosaki K, et al. (2007) Effects of several virucidal agents on inactivation of influenza, Newcastle disease, and avian infectious bronchitis viruses in the allantoic fluid of chicken eggs. Jpn J Infect Dis 60: 342-346.
23. WHO (2014) Interim Infection Prevention and Control Guidance for Care of Patients with Suspected or Confirmed Filovirus Haemorrhagic Fever in Health-Care Settings, with Focus on Ebola.
24. Payan C, Cottin J, Lemarie C, Ramont C (2001) Inactivation of hepatitis B virus in plasma by hospital in-use chemical disinfectants assessed by a modified HepG2 cell culture. J hosp Infect 47: 282-287.
25. Roberts PL, Dunkerley C (2003) Effect of manufacturing process parameters on virus inactivation by solvent-detergent treatment in a high-purity factor IX concentrate. Vox Sang 84: 170-175.
26. Ekstrom JO, Tolf C, Edman KA, Lindberg AM (2007) Physicochemical properties of the Ljungan virus prototype virion in different environments: inactivated by heat but resistant to acidic pH, detergents and non-physiological environments such as Virkon-containing solutions. Microbiol Immunol 51: 841-850.
27. Chan YF, Abu Bakar S (2005) Virucidal activity of Virkon S on human enterovirus. Med J Malaysia 60: 246-248.
28. Eleraky N, Potgieter LN, Kennedy MA (2002) Virucidal efficacy of four new disinfectants. J Am Anim Hosp Assoc 38: 231-234.
29. Clark EM, Wright H, Lennon KA, Craik VA, Clark JR, et al. (2012) Inactivation of recombinant bacteriophage lambda by use of chemical agents and UV radiation. Appl Environ Microbiol 78: 3033-3036.
30. Halfhide DE, Gannon BW, Hayes CM, Roe JM (2008) Wide variation in effectiveness of laboratory disinfectants against bacteriophages. Lett Appl Microbiol 47: 608-612.
31. FDA, HHS (2016) Safety and Effectiveness of Consumer Antiseptics; Topical Antimicrobial Drug Products for Over-the-Counter Human Use. Final rule. Fed Regist 81: 61106.
32. Shimizu-Onda Y, Akasaka T, Yagyu F, Komine-Aizawa S, Tohya Y, et al. (2013) The virucidal effect against murine norovirus and feline calicivirus as surrogates for human norovirus by ethanol-based sanitizers. J Infect Chemother 19: 779-781.