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Impact of storage temperature and ultraviolet irradiation on rotavirus survival on food matrices

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ABSTRACT

This study investigated the survival of human rotavirus (HRV) on fresh beef, chicken, and lettuce stored at various temperatures, as well as the effect of UV-C exposure on HRV viability on these food surfaces. At 20 °C, the survival rate of three HRV strains (WA, 89-12C2, and DS-1) on beef, chicken, and lettuce decreased within 3 days, with the most significant reduction observed on beef. When stored at 4 °C, a significant reduction in HRV viability was observed by day 7, with the greatest decrease observed on beef, followed by chicken and lettuce. Conversely, storage at −20 °C for up to 28 days did not significantly reduce HRV viability on any of the food surfaces. Exposure to UV-C irradiation at a dosage of 100 mJ/cm^2 reduced the viral titers on beef and chicken surfaces by approximately 1 $\log_{10}$ PFU/mL, while those on the surfaces of lettuce were more than 4 $\log_{10}$ PFU/mL. These findings indicate that HRV strains exhibit strong viability on beef, chicken, and lettuce surfaces, enduring extended periods at low temperatures, but display varying susceptibility to UV-C irradiation. Due to the persistence of HRV on contaminated food, implementing effective measures to prevent food contamination is crucial. The findings of this study contribute to the development of a robust sanitation strategy utilizing UV-C to mitigate foodborne HRV transmission.

1. Introduction

Foodborne diseases, commonly referred to as food poisoning, typically occur when individuals consume food contaminated with bacteria, toxins, pathogens, viruses, chemicals, or other harmful substances. According to the WHO, an estimated 600 million—almost 1 in 10 people in the world—fall ill each year due to contaminated food, leading to 420,000 deaths annually. In low- and middle-income countries, unsafe food results in economic losses of approximately US$110 billion annually, encompassing both productivity and medical expenses. Notably, children under the age of five bear a disproportionate burden, accounting for 40 % of foodborne disease cases and 125,000 deaths every year (WHO, 2022). The Republic of Korea reported a total of 311 incidents of foodborne disease outbreaks in 2022, an increase from the 245 incidents reported in the previous year. Although there have been fluctuations, the overall trend in outbreaks has been upward over the years. Notably, in 2020, the onset of the COVID-19 pandemic, coupled with lockdowns and restrictions on dining out, led to a significant decline in foodborne disease outbreaks compared to previous years (Statista, 2024). In the European Union, viral agents were identified as the cause of 11.9 % of foodborne outbreaks, making them the second most common etiologic agents (EFSA, 2011). Viruses remain a major contributor to foodborne diseases, causing numerous outbreaks worldwide.

Human intestinal viruses, including hepatitis A virus, norovirus, and rotavirus (RV), are significant contributors to global cases of food-borne viral gastroenteritis (Lemon et al., 2018; Scallan et al., 2015). Human RV (HRV) is particularly concerning due to its role in causing severe gastrointestinal infections, posing a significant threat to public health worldwide. Between 2013 and 2017, HRV was responsible for approximately 258 million cases of diarrhea in children under the age of five, resulting in an estimated 122,000–215,000 deaths worldwide (Bulto et al., 2017; Tate et al., 2016; Troeger et al., 2018). HRV, a member of the *Reoviridae* family, is a nonenveloped virus with a double-stranded, segmented RNA genome (Angel et al., 2007). To date, 36 G-types and 51 P-types have been identified through human and animal surveillance

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worldwide (RCWG, 2021). Globally, most infections are caused by one of six genotypes: G1P[8], G2P[4], G3P[8], G4P[8], G9P[8], and G12P[8] (Dóró et al., 2014). Group A HRV, which accounts for approximately 90 % of HRV gastroenteritis cases, has a global distribution (Crawford et al., 2017). The three primary genotype constellations are WA-like (G1P[8]), DS-1-like (G2P[4]), and AU-1-like (G3P[9]) (Donato & Bines, 2021), with WA- and DS-1-like genotypes being the most prevalent in humans (Sadiq et al., 2019). The HRV strain 89-12 (G1P[8]), isolated from the feces of a 14-month-old child with a spontaneous HRV infection in 1988, is a prime candidate for the development of rotavirus vaccines aimed at preventing rotavirus disease (Colau & De Vos, 2009).

HRV contamination in food products is a significant public health concern. Pereira et al. (2018) reported the presence of RV in fresh and processed products from Argentina and Uruguay. HRV has also identified in beef and chicken from various commercial sources in Uluguayana, Brazil (Soares et al., 2022). Additionally, RV has been detected in ready-to-eat packaged green leafy vegetables, lettuce, and oysters (Quiroz-Santiago et al., 2014; Mattison et al., 2010; Felix-Valenzuela et al., 2012). Improper handling and storage of food products can further increase the risk of HRV spread within households (Hessel et al., 2019). Enteric virus is commonly transmitted via the fecal-oral route, which includes contaminated food and water as well as contact with contaminated surfaces (Sánchez & Bosch, 2016). HRV can persist on various types of foods and under different environmental conditions for days or months (Butot et al., 2008; Gagné et al., 2022). The adherence of HRV to fresh produce surfaces is influenced by several factors, including chemical composition, surface roughness, and hydrophobicity (Kukavica-Ibrulj et al., 2004; Lu et al., 2015; Deboosere et al., 2012; Vega et al., 2008). Beef and chicken are among the most consumed meats globally, and lettuce is a major source of foodborne viruses (CDC, 2021; OECD/FAO, 2021).

To reduce the occurrence of viral foodborne infections from contaminated food products, it is crucial to understand the environmental and agricultural conditions that influence the stability of foodborne viruses and their potential to cause infection. The number of steps involved in processing food from harvest to table increases the risk of cross-contamination from dirty hands or surfaces. The risk of foodborne illness rises when surfaces that contact prepared foods are not properly cleaned. Consequently, maintaining a clean environment during food processing is essential. Minimizing HRV contamination in food processing areas can help reduce HRV-associated illnesses, thereby enhancing preventive measures against such infections. To achieve this objective, effective methods for the controlled removal of HRV from contaminated surfaces are necessary. Non-contact disinfection methods, particularly UV-C irradiation (200–280 nm), have been proposed for their potential to deactivate various viruses (Biasin et al., 2021). Numerous studies have examined the effects of UV-C radiation on viruses (Nishisaka-Nonaka et al., 2018; Araud et al., 2020; Park & Ha, 2015). One primary virucidal mechanism of UV-C is the inactivation of the virus through the production of photoproducts, caused by the absorption of UV-C photons by nucleic acids and/or capsid proteins (Qiao & Wigginton, 2016; Wigginton & Kohn, 2012). The USFDA and the USDA have determined that UV-C radiation at 253.7 nm is a safe treatment for use in food processing and have authorized its use as an alternative method to reduce pathogens and other microorganisms (USFDA, 2000). Additionally, the USFDA has issued Code 21CFR179.41, which approves the use of UV-C light in the production, processing, and handling of food.

Given the increasing prominence of food safety and disease control issues related to HRV, and the limited feasibility studies on HRV contamination of food substrates, comprehensive studies are crucial. The present study aimed to address this gap by evaluating the survival patterns of three different HRV strains (WA, DS-1, and 89-12C2) on beef, chicken, and lettuce surfaces under various storage temperatures. Additionally, the study aimed to evaluate the efficacy of UV-C irradiation in inactivating HRV-contaminated food substrates.

2. Materials and methods

2.1. Preparation of rotavirus stock

MA-104 cells (ATCC number: CRL-2378.1) were cultured in 75-cm^2 culture flasks containing Dulbecco's Modified Eagle Medium (DMEM; Gibco, Maryland, USA) supplemented with 10 % fetal bovine serum (FBS; Gibco) and 1 % antibiotic-antifungal solution (AA, Gibco). The cells were incubated in a humidified 5 % CO_2 incubator at 37 °C. The HRV WA strain (ATCC VR-2018), the HRV DS-1 strain (ATCC VR-2550), and the HRV 89-12C2 strain (ATCC VR-2272) were obtained from the American Type Culture Collection. Monolayer MA104 cells were cultured to 90 % confluence (approximately 3–4 days) and then used for viral infection. Virus samples were preactivated by incubating with 10 μg/mL trypsin from porcine pancreas (SIGMA) at 37 °C for 30 min. Subsequently, 100 μL of the virus suspension was added to cells in 1X DMEM (without FBS) containing 1 % AA and 5 μg/mL trypsin from porcine pancreas (SIGMA). The virus was allowed to adsorb to the cells for approximately 2 h. Following virus adsorption, the cells were incubated at 37 °C and 5 % CO_2 in a humid incubator for 3 days. The development of a cytopathic effect (CPE) was monitored daily. When CPE exceeded 100 %, the infected cells were subjected to three consecutive freeze–thaw cycles to release the virus. Cell debris was eliminated by centrifuging the samples at 4,000 rpm for 10 min at 4 °C. The supernatant containing the virus was collected and stored at −80 °C until further use. Viral titers (log_{10} PFU/mL) were calculated using the plaque assay (O'Mahony et al., 2000).

2.2. Experimental design

Before starting the experiment, beef sirloin, chicken breasts, and fresh lettuce were purchased from a local market in Anseong, South Korea. Lettuce was washed with flowing tap water and then rinsed at least three times with distilled water. For chicken breast and beef sirloin, the rinsing step was omitted to prevent microbial growth. The beef and chicken were cut into 0.5 × 0.5 × 0.5 cm^3 cubes, while the lettuce was sliced into 1 × 1 cm^2 pieces using a sterilized knife (Lee et al., 2022). To eliminate any residual pathogens, all samples were dried in a laminar flow hood by exposing them to UV light for 10 min on each side.

The original stock concentrations of virus particles were 7.02, 7.14, and 7.24 log_{10} PFU/mL for the WA, 89-12C2, and DS-1 strains, respectively. The surface of the food samples was inoculated with 50 μL of viral solution from the virus stock. The samples were incubated for 30 min inside a biosafety cabinet to allow the viral suspension to dry. The cubes were then transferred to 5 mL tubes and stored separately at 20, 4, and −20 °C. Storage times were 72 h for 20 °C (0 [immediately after inoculation], 8, 24, 48, 72 h), 1 week for 4 °C (0, 1, 2, 3, 5, 7 days), and 28 days for −20 °C (0, 1, 7, 14, 21, 28 days), respectively.

To recover the virus from the stored samples, 2 mL of 1X DMEM (0 % FBS, 1 % AA) was added to each sample, which was then vortexed for 15 s. The samples were further purified to remove contaminants using a 0.45 μm filter (BioFACT). The pooled samples were subjected to ten-fold serial dilutions using a 1X DMEM solution lacking FBS and containing 1 % AA. The viral infection titers were subsequently analyzed using the plaque assay method.

2.3. Plaque assay

Plaque detection was performed as described by Bidawid et al. (2003). MA104 cells were cultured in 12-well plates (2 × 10^5 cells per well) and incubated at 37 °C with 5 % CO_2 until they reached 100 % confluence (2–3 days). A virus recovery solution was prepared, and ten-fold serial dilutions were made to obtain five virus concentrations. Then, 500 uL of the virus solution was added drop by drop into each well, and the plates were incubated at 37 °C for 2 h for the WA and 89-12C2 strains, and for 4 h for the DS strain. Following incubation, 1 mL of a

2X agarose mixture (2X agarose combined with 2X DMEM containing 0 % FBS, 1 % AA, and 10 μg/mL trypsin from porcine pancreas) was added to each well and allowed to solidify at room temperature. The plates were then incubated for 3 to 4 days at 37 °C with 5 % CO_2. Subsequently, the agarose layer was treated with 1 mL of 10 % formalin for 4 h, then meticulously removed using tap water. Following a 10-min incubation period, each well was stained with a 0.1 % crystal violet solution and subsequently analyzed for the presence of plaques. Viral titers and plaque counts were expressed as Plaque Forming Units (PFU) per milliliter (mL). The plaque counts from a minimum of three replicates for each dilution were averaged.

2.4. UV inactivation

A DS-701-1 UV sterilizer (SUNG JIN), equipped with a G6T5 germicidal lamp (SANKYO DENKI) that emits UV-C radiation (254 nm wavelength), was utilized in this study. A UV meter was employed to quantify the intensity of the UV-C radiation. The experimental procedure followed the radiation intensity parameters set by Li et al. (2009). The food surfaces were inoculated with 50 μL virus solution from the virus stock, which had titers of 6.55, 7.07, and 6.63 $\log_{10}$ PFU/mL for the WA, 89-12C2, and DS-1 strains, respectively. The samples were incubated for 30 min inside a biosafety cabinet to allow the virus solutions on the samples to dry. Subsequently, the samples were subjected to UV radiation with doses ranging from 10 to 100 mJ/cm^2 in 10 mJ/cm^2 increments, using a UV-C lamp with an intensity of 0.21 mW/cm^2. To determine the effective UV-C dosage for microbial deactivation, the exposure time was calculated based on the UV-C lamp's intensity, as off-the-shelf UV-C lamps may vary in performance. The standard unit for measuring UV-C intensity is milliwatts per square centimeter (mW/cm^2). The required duration of UV-C exposure is directly related to the amount of UV-C radiation received and can be calculated using the following formula (Baldelli et al., 2022): Exposure time (s) = UV dose (mJ/cm^2)/ Intensity (mW/cm^2) [in this study, the intensity was 0.21 mW/cm^2].

For virus recovery from UV-C treated samples, each sample was added to 2 mL of 1X DMEM (0 % FBS, 1 % AA) and vortexed for 15 s. The mixture was then filtered through a 0.45 μm filter to remove contaminants. Subsequently, the samples were diluted ten-fold with 1X DMEM (0 % FBS, 1 % AA), and the effect of UV-C light on HRV survival was assessed using a plaque assay. The quantity of infectious viral plaques in the samples was counted and compared to that in the corresponding non-irradiated virus samples (control). Each experiment included three negative controls (samples not exposed to the virus but subjected to UV treatment) and three positive controls (samples inoculated with the virus but not exposed to UV treatment).

2.5. Statistical analysis

Each experiment was performed in triplicate, and the data are expressed as the mean ± standard deviation (SD). Graphs were constructed using GraphPad Prism 9.0 software for Windows (GraphPad Software, Inc., La Jolla, CA, USA). Statistical significance was determined using a one-way analysis of variance (ANOVA) followed by Duncan's Post-hoc test, performed with SPSS statistics v26.0 (Statistical Package for Social Science, SPSS Inc., Chicago, IL, USA). Statistical significance was set at $P < 0.05$.

3. Result

3.1. Survival of HRV WA strains on food surfaces

The survival of HRV WA strains on different food surfaces at different temperatures (20, 4, and −20 °C) and over different storage times is depicted in Figs. 1–3. The initial titer of the HRV WA strain stock was 7.08 ± 0.24 $\log_{10}$ PFU/mL, and the initial recovery titers (at 0 h) from beef, chicken, and lettuce were 5.49 ± 0.05, 5.45 ± 0.13, and 5.26 ± 0.28 $\log_{10}$ PFU/mL, respectively.

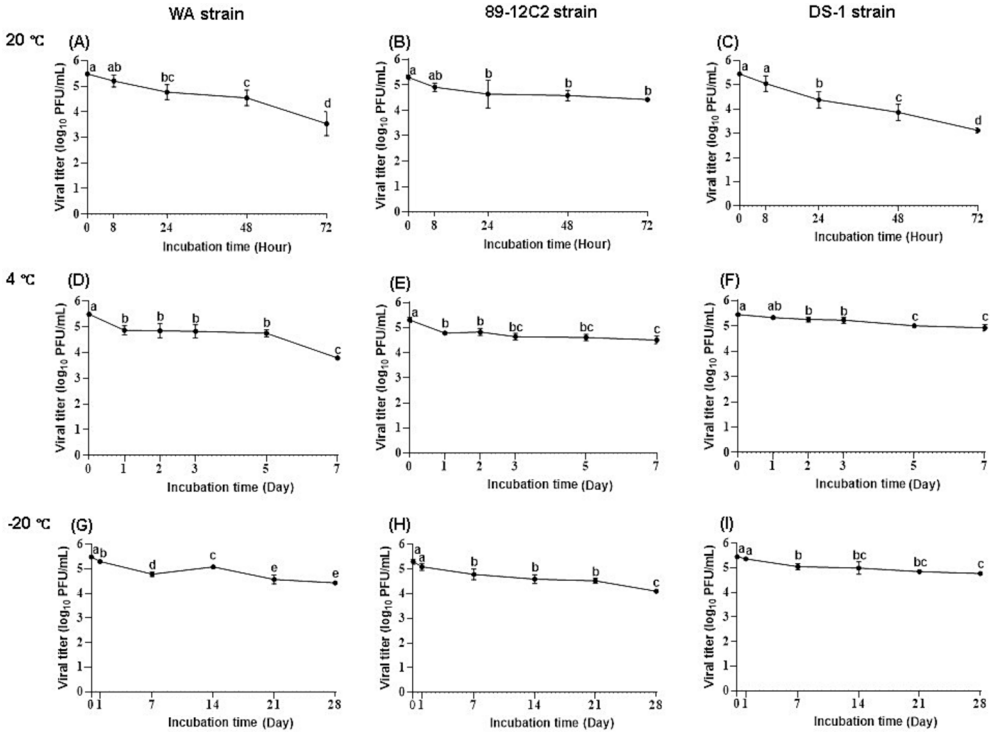

Fig. 1. Survival of three human rotavirus (HRV) strains on beef at 20, 4, and −20 °C. The panels are arranged such that each row represents a different temperature (20, 4, and −20 °C), and each column represents a different HRV strain (WA, 89-12C2, and DS-1). Panels (A), (D), and (G) represent the WA strain; panels (B), (E), and (H) represent the 89-12C2 strain; and panels (C), (F), and (I) represent the DS-1 strain. Each data point indicates the mean ± standard deviation (SD). Different letters indicate significant differences ($P < 0.05$) between the survival rates at different temperatures.

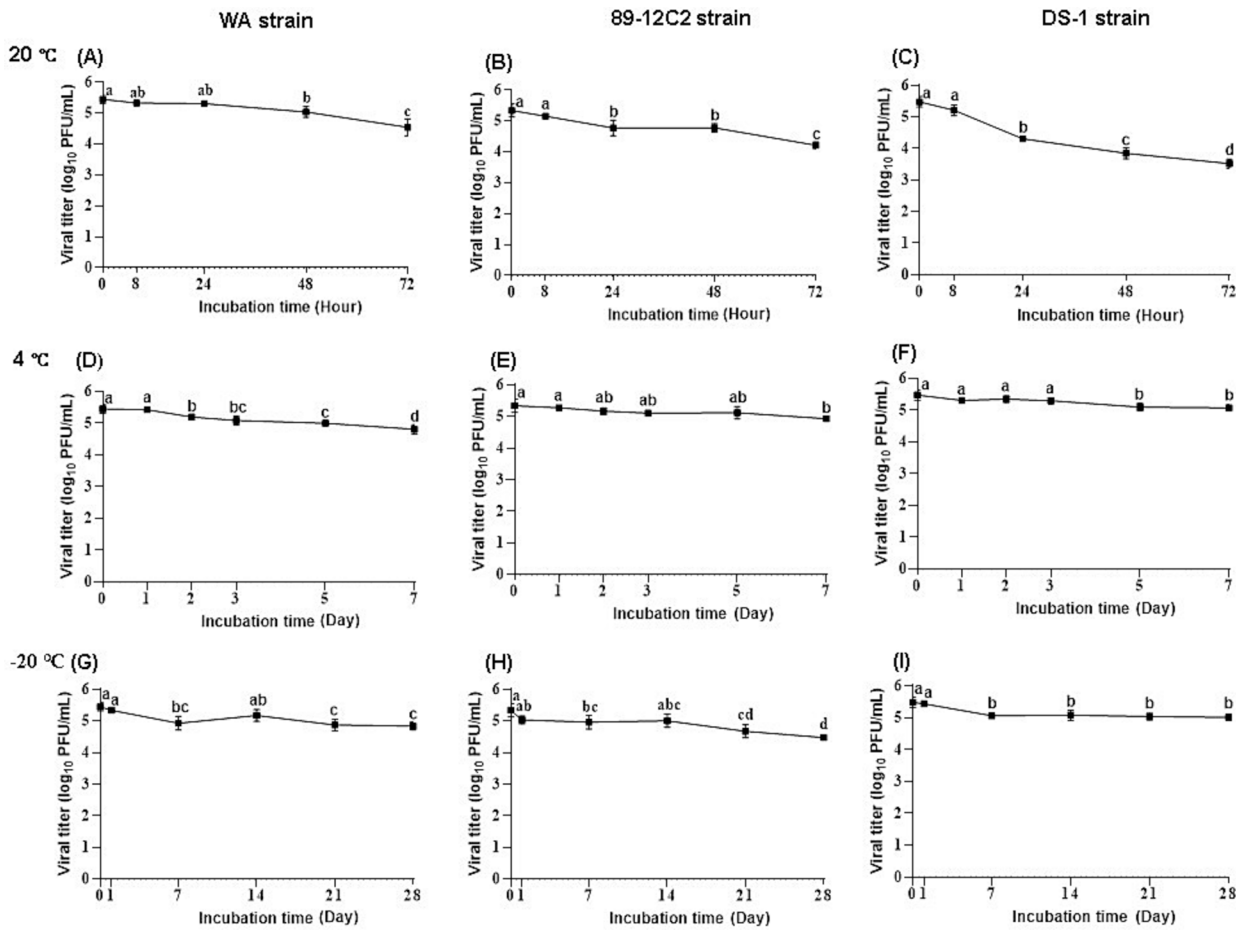

Fig. 2. Survival of three HRV strains on chicken at 20, 4, and −20 °C. The panels are arranged such that each row represents a different temperature (20, 4, and −20 °C), and each column represents a different HRV strain (WA, 89-12C2, and DS-1). Panels (A), (D), and (G) represent the WA strain; panels (B), (E), and (H) represent the 89-12C2 strain; and panels (C), (F), and (I) represent the DS-1 strain. Each data point indicates the mean ± SD. Different letters indicate significant differences ($P < 0.05$) between the survival rates at different temperatures.

After 72 h at 20 °C, the virus titers on the surfaces of beef, chicken, and lettuce decreased to 3.54 ± 0.47 (1.95 $\log_{10}$ reduction), 4.54 ± 0.28 (0.90 $\log_{10}$ reduction), and 3.70 ± 0.13 $\log_{10}$ PFU/mL (1.55 $\log_{10}$ reduction), respectively (Figs. 1A, 2A, and 3A).

After storage at 4 °C for 7 days, virus titers on the surfaces of beef, chicken, and lettuce decreased to 3.79 ± 0.05, 4.81 ± 0.14, and 4.63 ± 0.20 $\log_{10}$ PFU/mL, respectively (Figs. 1D, 2D, and 3D). The reductions in virus titer on the surfaces of beef, chicken, and lettuce were 1.70, 0.64, and 0.64 $\log_{10}$ PFU/mL, respectively.

After storage at −20 °C for 28 days, the WA strain titers on the three food surfaces were reduced by 1.06 (4.43 ± 0.04 $\log_{10}$ recovery on beef), 0.61 (4.84 ± 0.12 $\log_{10}$ recovery on chicken), and 0.54 $\log_{10}$ PFU/mL (4.72 ± 0.30 $\log_{10}$ recovery on lettuce) (Figs. 1G, 2G, and 3G).

These results indicate that the HRV WA strain can survive under various storage temperature conditions (20, 4, and −20 °C).

3.2. Survival of HRV 89-12C2 strain on food surfaces

Figs. 1–3 illustrate the survival ability of the HRV 89-12C2 strain on beef, chicken, and lettuce surfaces at different temperatures (20, 4, and −20 °C) and over different storage periods. The initial titer of the 89-12C2 strain stock was 7.42 ± 0.12 $\log_{10}$ PFU/mL, and the initial recovery titers from the beef, chicken, and lettuce surfaces were 5.30 ± 0.10, 5.34 ± 0.21, and 5.49 ± 0.17 $\log_{10}$ PFU/mL, respectively.

After 72 h at 20 °C, the titer of the 89-12C2 strain decreased to 4.43 ± 0.02 $\log_{10}$ PFU/mL on beef, 4.21 ± 0.10 $\log_{10}$ PFU/mL on chicken, and 4.19 ± 0.06 $\log_{10}$ PFU/mL on lettuce. The virus titers were reduced by 0.87, 1.13, and 1.30 $\log_{10}$ PFU/mL on beef, chicken, and lettuce, respectively (Figs. 1B, 2B, and 3B).

After storage for 7 days at 4 °C, the recovery titers of the 89-12C2 strain on beef, chicken, and lettuce were 4.51 ± 0.15, 4.93 ± 0.03, and 4.99 ± 0.16 $\log_{10}$ PFU/mL, respectively. The virus titers on the beef, chicken, and lettuce surfaces were reduced by 0.79, 0.41, and 0.50 $\log_{10}$ PFU/mL, respectively (Figs. 1E, 2E, and 3E).

After storage at −20 °C for 28 days, the titers of the 89-12C2 strain were 4.10 ± 0.05, 4.47 ± 0.09, and 4.81 ± 0.11 $\log_{10}$ PFU/mL on the beef, chicken, and lettuce surfaces, respectively. The virus titers were reduced by 1.20, 0.87, and 0.68 $\log_{10}$ PFU/mL on the beef, chicken, and lettuce surfaces, respectively (Figs. 1H, 2H, and 3H).

In summary, the HRV 89-12C2 strain exhibited variable viability across different temperature conditions (20, 4, and −20 °C).

3.3. Survival of HRV DS-1 on food surfaces

The survival dynamics of the HRV DS-1 strain on the beef, chicken, and lettuce surfaces at different temperatures and across different storage times are illustrated in Figs. 1–3. The initial titer of the DS-1 strain stock was 7.24 ± 0.21 $\log_{10}$ PFU/mL, and the initial recovery titers from beef, chicken, and lettuce were 5.45 ± 0.08, 5.48 ± 0.17, and 5.37 ± 0.05 $\log_{10}$ PFU/mL, respectively.

After 72 h at 20 °C, the viral titers on beef decreased to 3.13 ± 0.11 $\log_{10}$ PFU/mL, while those on chicken and lettuce decreased to 3.52 ± 0.14 and 4.67 ± 0.77 $\log_{10}$ PFU/mL, respectively. The reductions in the viral titers on the beef, chicken, and lettuce surfaces were 2.32, 1.96, and

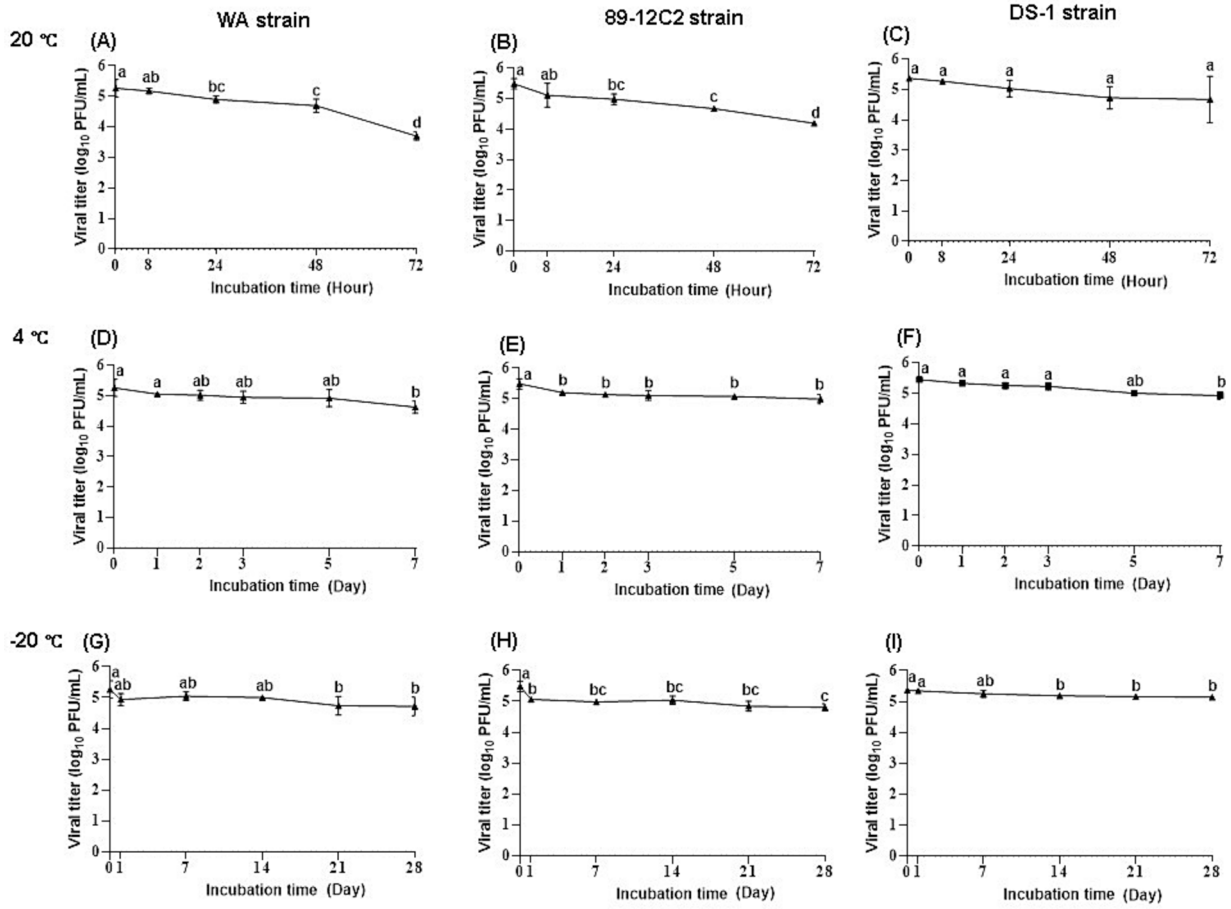

Fig. 3. Survival of three HRV strains on lettuce at 20, 4, and −20 °C. The panels are arranged such that each row represents a different temperature (20, 4, and −20 °C), and each column represents a different HRV strain (WA, 89-12C2, and DS-1). Panels (A), (D), and (G) represent the WA strain; panels (B), (E), and (H) represent the 89-12C2 strain; and panels (C), (F), and (I) represent the DS-1 strain. Each data point indicates the mean ± SD. Different letters indicate significant differences ($P < 0.05$) between the survival rates at different temperatures.

0.70 $\log_{10}$ PFU/mL, respectively (Figs. 1C, 2C, and 3C).

After storage at 4 °C for 7 days, the DS-1 strain titers on the surfaces of beef, chicken, and lettuce decreased to 4.93 ± 0.13, 5.07 ± 0.10, and 5.01 ± 0.27 $\log_{10}$ PFU/mL, respectively. The virus titers on the beef, chicken, and lettuce surfaces were reduced by 0.52, 0.40, and 0.35 $\log_{10}$ PFU/mL, respectively (Figs. 1F, 2F, and 3F).

After storage at −20 °C for 28 days, the DS-1 strain titers on the beef, chicken, and lettuce surfaces decreased to 4.77 ± 0.05, 5.01 ± 0.08, and 5.14 ± 0.07 $\log_{10}$ PFU/mL, respectively. The virus titers on the beef, chicken, and lettuce surfaces were reduced by 0.68, 0.47, and 0.23 $\log_{10}$ PFU/mL, respectively (Figs. 1I, 2I, and 3I).

In summary, the survival pattern of the HRV DS-1 strain varied across temperatures (20, 4, and −20 °C).

3.4. UV-C inactivation

The capacity to inactivate HRV strains using UV-C radiation was assessed through a series of experiments. The initial titers of the HRV WA, HRV 89-12C2, and HRV DS-1 strain stocks were 6.55 ± 0.81, 7.07 ± 0.25, and 6.63 ± 0.13 $\log_{10}$ PFU/mL, respectively. The HRV strains were recovered from beef, chicken, and lettuce, respectively. The initial recovery titers of the WA strain from beef, chicken, and lettuce were 4.85 ± 0.29, 5.08 ± 0.40, and 4.75 ± 0.44 $\log_{10}$ PFU/mL, respectively. The initial recovery titers of the 89-12C2 strains from beef, chicken, and lettuce were 4.72 ± 0.37, 5.17 ± 0.33, and 4.72 ± 0.37 $\log_{10}$ PFU/mL, respectively. The initial recovery titers of the DS-1 strains from beef, chicken, and lettuce were 5.03 ± 0.27, 5.11 ± 0.10, and 4.66 ± 0.29 $\log_{10}$ PFU/mL, respectively.

The viability of these HRV strains across the tested UV-C energy spectrum is illustrated in Fig. 4. The results demonstrated that UV-C irradiation significantly ($P < 0.05$) reduced the infectivity of the HRV strains in a dose-dependent manner. As the UV-C dose increased, both in intensity and duration, the survival rate of HRV decreased. Notably, dose of 100 mJ/cm^2 UV-C irradiation reduced the infectivity of the HRV strains by more than 4 $\log_{10}$ PFU/mL on lettuce surfaces (Fig. 4).

Overall, UV-C dose increases were less effective in inactivating HRV on beef and chicken, with greatest sensitivity observed on lettuce surfaces (Fig. 4). This variation in the effectiveness of UV-C radiation is likely due to differences in the surface characteristics of the food items. The three HRV stains—WA, 89-12C2, and DS-1—exhibited similar inactivation patterns on chicken and beef. However, on lettuce, the three HRV strains were more inactivated compared to the beef and chicken.

4. Discussion

To evaluate the efficacy of potential control methods, the agri-food and food-processing sectors require a viral surrogate that exhibits prolonged persistence under realistic production conditions and throughout the food's shelf life. Consequently, it is of utmost importance to assess the viability of different lab-cultivable viral surrogates in nearly identical matrices, using consistent protocols and under identical circumstances. The present study investigated the survival efficiency of HRV three stains (WA, 89-12C2, and DS-1) on beef, chicken, and lettuce surfaces stored at different temperatures. Leblanc et al. (2019) reported

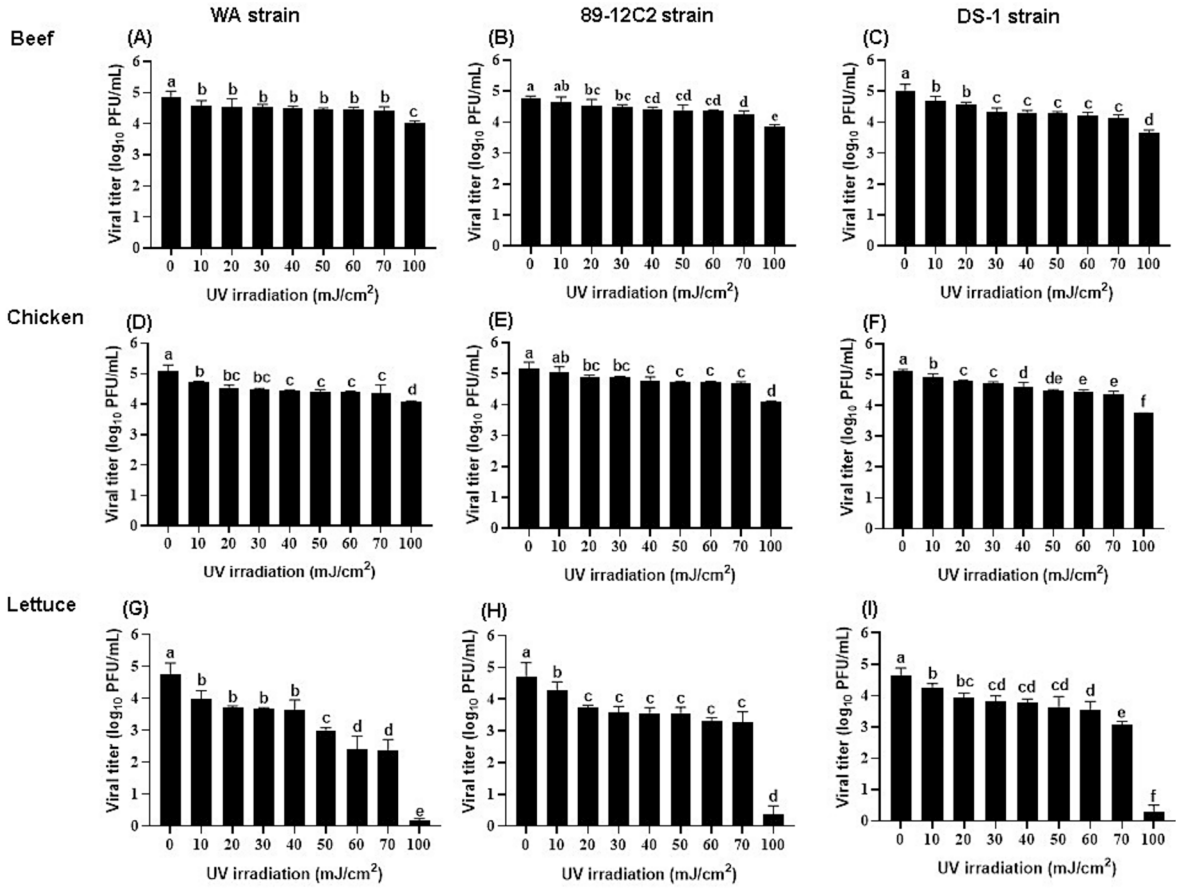

Fig. 4. Inactivation of three HRV strains through UV irradiation on different food surfaces. The panels are arranged such that each row represents a different food type (beef, chicken, and lettuce), and each column represents a different HRV strain (WA, 89-12C2, and DS-1). Panels (A), (D), and (G) represent the WA strain; panels (B), (E), and (H) represent the 89-12C2 strain; and panels (C), (F), and (I) represent the DS-1 strain. Each data point indicates the mean ± SD. Different letters indicate significant differences ($P < 0.05$) between the effects of different UV doses.

that blueberries can still maintain their infectious properties for 21 days when stored at 4 °C and −20 °C conditions; however, the infectious properties of bovine rotavirus decreased by more than 2 $\log_{10}$ when incubated at 21 °C for 7 days. Butot et al. (2008) demonstrated that freezing berries at −20 °C did not significantly inhibit virus replication; the number of RVs decreased by less than 1 $\log_{10}$ after 90 days of storage at −20 °C. Viruses can endure for extended periods under low and freezing temperatures (Dublineau et al., 2011). In contrast, exposure to high temperatures notably reduces the viral load within a few days (Yang & Griffiths, 2013). Furthermore, Paluszak et al. (2012) demonstrated that heat can eliminate viruses within a minute, whereas freezing temperatures allow viruses to survive for several months, albeit with an initial decrease in their numbers within the first day (Shoham et al., 2012). How temperature impacts RNA virus replication, modifies the immune response, and effects virus survival outside the host is not entirely known, while numerous research have investigated the molecular pathways via which temperature impacts bacteria and bacterial illnesses (Bisht et al., 2021; Townsley et al., 2016). The viral infection cycle relies on all these factors, and Bisht and Te Velthuis (2022) reviewed the decoding role of temperature in RNA virus infections. In the cytoplasm or nucleus of the host cell, an RNA-dependent RNA polymerase (RdRp) replicates RNA viruses once they bind to receptors on host cells. Here, the viral RdRp interacts with one or more host components to replicate or transcribe the viral RNA genome according to multiple reviews on positive-sense and negative-sense RNA viruses. Following this, the translation of viral messenger RNAs (mRNAs) and the subsequent assembly of viral proteins (virions) from viral RNA occur. Viral enzyme conformation and activity, protein-RNA and protein–protein interactions, and the folding of RNA secondary structures in the genome are all affected by the temperature at which virion assembly and RNA synthesis occur. A number of factors, including the infection's temperature, influence the innate immune response, viral RNA molecule formation, viral proliferation, and transmission efficiency. Understanding how temperature affects viral RNA synthesis, the immune response, and the stability of RNA viruses in various environments is crucial for estimating the future risk of human infection with spread RNA viruses, as well as for developing antiviral strategies. Hence, our study aimed to examine the impact of temperature on viral load. To simulate the temperature conditions encountered during food storage and processing, experiments were conducted at three distinct temperatures: a low temperature of 4 °C, an ambient temperature of 20 °C, and a freezing temperature of −20 °C. The results demonstrated that the persistence of HRV stains on food substrates is inversely proportional to the storage temperature. Notably, HRV stains could survive for extended periods in refrigerated (4 °C) and frozen (−20 °C) environments, compared to room temperature (20 °C). Additionally, all three HRV strains exhibited robust survivability on the surface of lettuce at temperatures of 20, 4, and −20 °C. Remarkably, they remained viable for over 28 days at temperatures lower than −20 °C. Notably, the DS-1 strain demonstrated greater consistency in viability compared to the other two HRV strains. These findings suggest that HRV could persist for extended periods on refrigerated or frozen food surfaces. In a broader context, the survival and recovery of viruses are influenced by several factors, including temperature, incubation time, relative humidity, the specific viral strain, viral load in the inoculum, the virus recovery method employed, and the type of food and its surface characteristics.

Therefore, it is essential to consider these variables to interpret results accurately and account for potential discrepancies when comparing findings with other studies (D'Souza & Joshi, 2016; Turnage & Gibson, 2017; Vasickova & Kovarcik, 2013).

This study used three different HRV strains for virus inactivation strategies in food is appropriate due to their prolonged persistence on food surfaces under the evaluated conditions. Furthermore, to ensure that inactivation treatments are effective, more than two different strains must be tested simultaneously. Additionally, understanding the mechanisms underlying the inactivation of various RV strains by different methods is crucial for developing effective food safety protocols. Typically, disinfectants or sanitizers act by destabilizing or degrading viral capsid proteins, including those that bind to host cells (Fuzawa et al., 2019: Araud et al., 2018), or by damaging or degrading the encapsulated viral genome (Brié et al., 2016; Wigginton et al., 2012). The present study investigated the effects of UV-C irradiation on inactivating three HRV strains. UV treatment is cost-effective, does not produce toxic or irritating by-products, and has minimal impact on product quality, making it suitable for application to food products (Pexara & Govaris, 2020). The primary mechanism of virus inactivation by UV involves interactions with viral nucleic acids and proteins, leading to the loss of infectivity. Specifically, UV-C radiation induces damage through the formation of pyrimidine dimers (e.g., thymine dimer) in the nucleic acids (DNA/RNA) of cells or viruses, inhibiting their replication and transcription. Studies have consistently shown that the response of viruses to UV-C radiation depends on their genome size and composition (Pendyala et al., 2020; Rockey et al., 2021; Sagripanti & Lytle, 2020). Photochemical damage may more readily target larger genomes, leading to the rapid inactivation of viruses and other pathogens with larger genomes. The sensitivity of viruses to UV-C exposure is influenced by their genome type—whether they are single-stranded (ss), double-stranded (ds), RNA, or DNA. Viruses with dsDNA genomes tend to be relatively resistant due to the complementary nucleic acid strand's ability to aid in host repair (Rodriguez et al., 2014; Shin et al., 2009). In contrast, dsRNA and dsDNA viruses exhibit lower sensitivity to UV radiation (Augsburger et al., 2021). Interestingly, the presence of a viral envelope can impact a virus's reactivity to certain physical agents, such as heat or shear forces. However, the response to UV-C exposure appears to be less affected by the presence of a viral envelope.

In this study, the concentrations of three HRV strains on beef and chicken surfaces decreased by approximately 1 $\log_{10}$ PFU/mL after exposure to 100 mJ/cm^2 UV-C irradiation. Conversely, the viral load on lettuce surfaces were decreased by over 4 $\log_{10}$ PFU/mL (Fig. 4). Bi et al. (2022) reported that UV irradiation at 13 mJ/cm^2 reduced viral loads by less than 0.1 $\log_{10}$ PFU/mL. Saguti et al. (2022) found no significant difference in inactivation efficiency between UV doses of 600 and 1,000 J/m^2 in 3L of RV SA11-containing tap water, achieving a reduction of 3.3 $\log_{10}$ PFU/mL at 1,000 J/m^2 UV dose. Moreover, in the present study, the physical characteristics of the food surface substantially influenced the effectiveness of UV-C inactivation. The textured or highly absorbent nature of beef and chicken reduced UV-C penetration, resulting in a diminished inactivation effect. Interestingly, lettuce surfaces, which were smooth as mirrors, enhanced the inactivation by UV-C light. A comprehensive understanding of UV-C's impact on HRV, considering all experimental factors, including culture medium, HRV concentration, UV-C irradiance, exposure time, and UV-C absorbance, is essential for replicating these results in other laboratories with different equipment. Furthermore, research indicates that UV radiation from sunlight effectively deactivates the virus (Ratnesar-Shumate et al., 2020). These findings are critical for planning further studies to investigate the impact of UV-A and UV-B on HRV replication. Additionally, Araud et al. (2020) demonstrated that 220 nm UV radiation was more effective than 254 nm UV irradiation for inactivating RV. These findings indicate that UV-C radiation at a wavelength of 254 nm can efficiently deactivate HRV on food surfaces when used at high intensity and for an extended duration.

The results of this study clearly demonstrate HRV's ability to survive on various food surfaces. This study contributes valuable insights for food safety management and provides a scientific foundation for the prevention and control of HRV. To reduce HRV viability on different food surfaces, it is crucial to adopt targeted strategies. These strategies should focus on enhancing disinfection procedures for food ingredients, as well as ensuring prompt heating and cooking. However, further investigations and experimental validation are required to address the limitations in methodology and sample size, ensuring that the findings are generalizable and applicable across a broader range of real-world conditions. Implementing proactive preventive measures and food safety practices is essential to mitigate the health risks associated with HRV's prolonged presence on contaminated food surfaces. This study also demonstrated that UV-C alone with low intensity and duration is insufficient to inactivate large amounts of the virus on food surfaces. However, combining UV-C with other treatments could improve inactivation efficacy. For example, Bi et al. (2022) observed that RV inactivation was enhanced by 63 % when UV irradiation (6 mJ/cm^2) was combined with chloramine (NH_2Cl: PPM $\times$ 60 min). Therefore, combining UV-C with other inactivation methods could enhance overall effectiveness.

5. Conclusion

This study employed plaque assays to investigate the long-term persistence of three HRV strains on beef, chicken, and lettuce, uncovering significant food safety concerns. The findings revealed that the survival of HRV strains varies with temperature and the type of food substrate, with HRV strains maintaining higher infectivity at −20 °C compared to 4 and 20 °C, thereby posing a risk of gastrointestinal diseases. Although UV-C irradiation was generally effective, its impact was inconsistent across different food surfaces. The UV-C dose of 100 mJ/cm^2 proved more effective on lettuce (>4 $\log_{10}$ reduction) than on beef and chicken (>1 $\log_{10}$ reduction). From the perspective of food industries, assessing whether foodborne viruses are inactivated during processing may require selecting different indicator viruses based on the specific type of food. Furthermore, stringent food safety measures are essential to mitigate HRV contamination.

CRediT authorship contribution statement

Yuan Zhang: Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation. **Md. Iqbal Hossain:** Writing – original draft, Visualization, Validation, Methodology, Data curation. **Daseul Yeo:** Writing – review & editing, Investigation, Data curation. **Teng Niu:** Writing – review & editing, Investigation, Data curation. **Seongwon Hwang:** Writing – review & editing, Investigation, Data curation. **Danbi Yoon:** Writing – review & editing, Investigation, Data curation. **Dong Jae Lim:** Writing – review & editing, Investigation, Data curation. **Zhaoqi Wang:** Writing – review & editing, Investigation, Data curation. **Soontag Jung:** Writing – review & editing, Investigation, Data curation. **Hyojin Kwon:** Writing – review & editing, Investigation, Data curation. **Changsun Choi:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

The authors do not have permission to share data.

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