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Analysis and Comparison of Detection and Quantification Methods for Influenza Virus

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Abstract. Influenza virus is a major pathogen of acute respiratory infectious diseases and poses a serious global public health threat. Rapid, accurate detection and quantification are essential for clinical diagnosis, epidemic control, and evaluation of therapeutic efficacy. This article compares four commonly used methods for influenza virus detection and quantification: hemagglutination assay (HA assay), 50% tissue culture infectious dose (TCID50) assay, plaque-forming unit (PFU) assay, and reverse transcription quantitative PCR (RT-qPCR), in terms of principle, quantitative capacity, sensitivity, detection time, and applicable scenarios. HA assay semi-quantifies the virus through hemagglutinin binding to red blood cells, it is simple and remains a classical method for WHO surveillance. TCID50 and PFU assays rely on cell culture and quantify infectious viral particles, they are core virological tools but require long turnaround times. RT-qPCR monitors amplification in real time and, with a standard curve, absolute quantification of viral RNA, offering the highest sensitivity and specificity as the gold standard for clinical molecular diagnosis. These methods represent three quantification levels: antigen-based (HA assay), infectivity-based (TCID50/PFU), and nucleic acid-based absolute quantification (RT-qPCR). They suit different scenarios, including virological research, vaccine development, and clinical diagnosis, providing a systematic reference for method selection.

Keywords: influenza virus, viral quantification, hemagglutination assay, TCID50, plaque assay, RT-qPCR

1. Introduction

Influenza viruses belong to the family Orthomyxoviridae and are single-stranded negative-sense RNA viruses. Among them, influenza A and influenza B viruses are the major pathogens responsible for seasonal influenza epidemics in humans. Influenza viruses are characterized by high transmissibility, rapid spread, and universal susceptibility among humans. Worldwide, there are approximately 3–5 million cases of severe influenza and 290,000–650,000 deaths each year [1]. Rapid and accurate detection of influenza viruses is not only essential for early diagnosis and antiviral treatment of individual patients but also has important implications for the development of public health prevention and control strategies.

Viral load is a key indicator for assessing disease severity, predicting transmission risk, and monitoring the efficacy of antiviral treatment, therefore, its precise quantification is essential.

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Currently, the main methods for influenza virus detection and quantification include hemagglutination (HA) assay [2], median tissue culture infectious dose (TCID50) assay [3], plaque assay [4], and real-time quantitative reverse transcription PCR (RT-qPCR) [5]. These methods differ markedly in detection principle, quantitative capacity, sensitivity, and applicable scenarios.

In this study, the principles and characteristics of these four methods were compared, and the methods were classified into three quantitative levels: antigen-based quantification, infectivity-based quantification, and absolute nucleic acid quantification, to provide a reference for the selection of appropriate methods for different research purposes.

2. Methodology

2.1. Main experimental materials and instruments

The main materials and instruments used in this study are listed in Table 1.

These included PBS (1×), DMEM (1×), penicillin-streptomycin, fetal bovine serum, trypsin-EDTA, 4% chicken red blood cells, cell culture plates, and cell culture flasks. The instruments included a low-speed benchtop centrifuge, a three-well thermostatic water bath, an automated cell fluorescence analyzer (Countstar Mira FL), and an inverted fluorescence microscope (Nikon TS100).

Table 1. Names, model/catalog numbers, and serial/lot numbers of materials and instruments

Material/instrument	Model/Catalog No.	Serial/Lot No.
Low-speed benchtop centrifuge	TD25-WS	/
Three-well thermostatic water bath	DK-8D	240103909
Automated cell fluorescence analyzer Countstar Mira	Countstar Mira FL	RY124C2019
Imported inverted fluorescence microscope	Nikon TS100	KY2023134
PBS (1×) cell culture medium	414038076	P1603060
DMEM cell culture medium (1×)	10-013-CVRC	10013181
Penicillin-Streptomycin Solution	BC-CE-007	BC20260602
Fetal Bovine Serum (Superfine)	BC-SE-FBS01	BC20240606
Trypsin-EDTA, 2.5% STERILE	CCo12.1	C0326N3
4% chicken red blood cells	SBJ-RBC-C003-100mL	20260820
12-well cell culture plate	11210	21625074BC
75 mL cell culture flask	13212A	/
96-well V-bottom cell culture plate	11538	14226631E
96-well cell culture plate	11510	11425077E
96-well deep-well plate (square well, U-bottom)	110424002	20260105

2.2. Hemagglutination (HA) assay

The hemagglutination assay was performed according to the WHO GISRS manual [1]. In a 96-well V-bottom microplate, 50 μL of PBS was added to each well. The 4% chicken red blood cell stock was diluted 1:8 with PBS (1 volume stock + 7 volumes PBS) to prepare a 0.5% working suspension. An equal volume of the 0.5% chicken red blood cell suspension was added to each well, and the plate was left at room temperature. The hemagglutination pattern was then observed. The reciprocal

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of the highest dilution that caused complete hemagglutination (red blood cells evenly covering the bottom of the well) was recorded as the hemagglutination titer (HAU). Each sample was tested in triplicate.

2.3. Median tissue culture infectious dose (TCID50) assay

The TCID50 assay was performed according to the Reed–Muench method. The influenza virus suspension was serially diluted 10-fold in serum-free DMEM and inoculated onto MDCK cell monolayers at 80-90% confluence in 96-well plates. Eight replicate wells were used for each dilution, with 100 μL of virus suspension inoculated per well. The plates were incubated at 37 °C for 72 h. After incubation, cytopathic effect (CPE) was observed under an inverted microscope. The number of CPE-positive wells at each dilution was recorded, and the TCID50/mL was calculated using the Reed-Muench formula.

2.4. Plaque assay (PFU)

For the plaque assay, the virus suspension was serially diluted 10-fold and inoculated onto confluent MDCK cell monolayers in 12-well plates. The plates were incubated at 37 °C for 1 h for virus adsorption, with gentle rocking every 0.25 h. After aspiration of the inoculum, each well was overlaid with DMEM containing 0.6% agarose and 1 μg/mL TPCK-trypsin. After the overlay solidified, the plates were inverted and incubated at 37 °C for 72 h. Plaques were visualized by crystal violet staining. Wells containing 20-100 plaques were counted, and the virus titer was calculated as PFU/mL = number of plaques × dilution factor / inoculum volume.

2.5. Real-time quantitative reverse transcription PCR (RT-qPCR)

Viral RNA was extracted using the TaKaRa MiniBEST Universal RNA Extraction Kit (Takara, Cat. #9767) according to the manufacturer's instructions. Briefly, cultured cells (1×10^5 – 1×10^7) were lysed in 350–600 μL of Buffer RL supplemented with 50× DTT Solution, and the lysate was passed through a gDNA Eraser Spin Column to remove genomic DNA. An equal volume of 70% ethanol was added to the flow-through, and the mixture was loaded onto an RNA Spin Column. After washing with Buffer RWA and Buffer RWB, RNA was eluted with 50 μL of RNase-free water. And the extracted RNA was reverse transcribed into cDNA. Using the cDNA as template, qPCR amplification was performed with specific primers. Plasmid DNA containing the target sequence or in vitro-transcribed RNA was serially diluted 10-fold and amplified in parallel with the test samples to generate a standard curve. A linear regression was performed with Ct values as the ordinate and the logarithm of the initial copy number as the abscissa. The Ct values of the samples were substituted into the standard curve equation to calculate the absolute copy number of viral RNA (copies/μL). Each sample was tested in triplicate.

2.6. Preparation of influenza virus

The influenza virus strain A/WSN/1933 (H1N1) was used in this study. The virus seed was prepared in our laboratory and was at the first passage (P1). Briefly, the frozen virus seed was thawed on ice and inoculated onto MDCK cell monolayers at 80-90% confluence in cell culture flasks. The flasks were incubated at 37 °C with 5% CO2 for 1 h to allow virus adsorption, with gentle rocking every 15 min to ensure even distribution. After adsorption, the unadsorbed virus suspension was removed and replaced with serum-free DMEM maintenance medium containing 1 μg/mL TPCK-trypsin. The

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cells were further cultured at 37 °C with 5% CO2. Cytopathic effect (CPE) was monitored daily under an inverted microscope. When more than 80% of cells showed obvious CPE (such as cell rounding, detachment, fusion, or fragmentation), the culture supernatant was harvested. The harvested virus was centrifuged at 3,000 × g for 15 min at 4 °C, and the supernatant was collected to remove cell debris and large particulate impurities. The collected supernatant was then sterile-filtered through a 0.22 μm filter, aliquoted according to single-experiment volumes, labeled with the virus strain name, passage number, and preparation date, and stored at -80 °C until use. All subsequent hemagglutination assay (HA assay), median tissue culture infectious dose (TCID50), RT-qPCR and plaque assay (PFU) were performed using the same batch of virus working stock to minimize inter-batch variation in virus titer determination.

3. Results

3.1. Hemagglutination (HA) assay

The hemagglutination assay is one of the most classical methods for influenza virus detection (Figure 1), first established by Hirst in 1941. Its basic principle is that the surface HA protein of influenza viruses can specifically bind to sialic acid receptors on red blood cells, causing red blood cell agglutination. By observing the presence and pattern of hemagglutination, the assay can detect hemagglutinating activity in a sample and estimate its relative level [6].

In practice, the hemagglutination assay employs a serial dilution method: the test virus suspension is subjected to two-fold serial dilution in a 96-well V-bottom plate, an equal volume of the chicken red blood cell suspension is added to each well, and the plate is left at room temperature for 30 min. The hemagglutination pattern in each well is then observed. The hemagglutination titer is expressed as the reciprocal of the highest dilution at which complete hemagglutination occurs. For example, if the virus suspension still causes complete hemagglutination at a 1:256 dilution, the hemagglutination titer is 256, corresponding to a reciprocal dilution endpoint of 256.

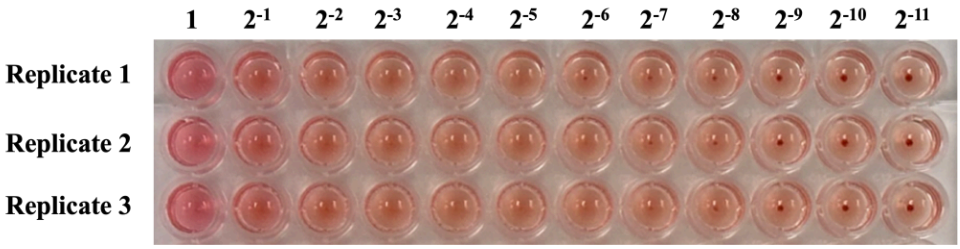


Figure 1. Results of the hemagglutination assay

"+" indicates complete hemagglutination (red blood cells evenly covering the bottom of the well, forming a thin layer), "-" indicates no hemagglutination (red blood cells settled at the center of the well bottom, forming a dot). The results showed that the hemagglutination titer of the virus strain was 256 HAU.

3.2. 50% tissue culture infectious dose (TCID50) assay

The 50% tissue culture infectious dose (TCID50) assay is a classical method for quantifying viral infectivity (Figure 2). TCID50 is defined as the amount or dilution of virus estimated to produce infection in 50% of inoculated cell cultures under specified experimental conditions [7]. This

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method was proposed by Reed and Muench in 1938 and remains one of the core quantitative tools in virological research.

The experimental procedure is as follows: the virus sample is serially diluted 10-fold (typically from $10^{-}1$ to $10^{-}10$). For each dilution, 100 μL is inoculated onto monolayers of host cells (such as MDCK cells) cultured in 96-well plates, with 8 replicate wells per dilution. After incubation at 37 °C for 3 days, the cells are examined under a microscope for cytopathic effect (CPE) to determine whether each well is infected, confirmation can also be performed by hemagglutination assay or immunostaining. The number of CPE-positive wells at each dilution is recorded, and the TCID50 value is calculated using the Reed-Muench method.

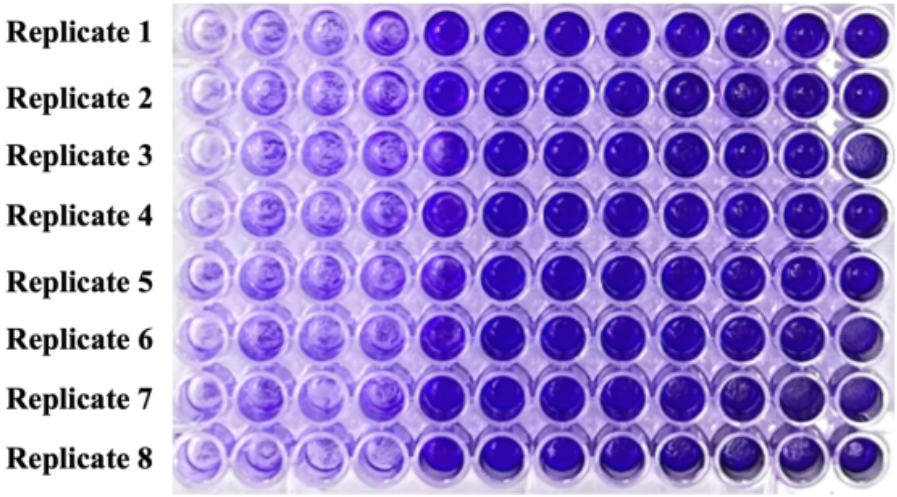


Figure 2. Results of the median tissue culture infectious dose (TCID50) assay

The virus strain was serially diluted 10-fold and inoculated onto MDCK cell monolayers. Eight replicate wells were set for each dilution, and the TCID50 was calculated using the Reed–Muench method. The result showed that the TCID50 was 3.16 × 104/mL.

3.3. Plaque-forming unit assay (PFU)

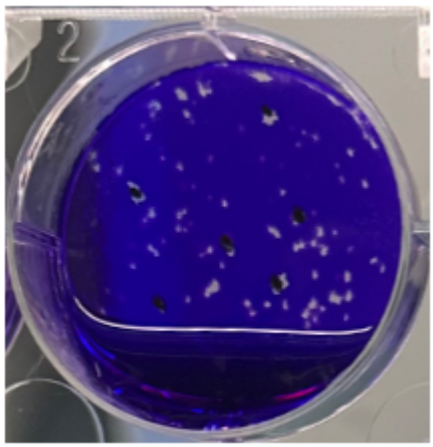


Figure 3. Plaque assay

The plaque-forming unit assay is another classical method for quantifying viral infectivity titer (Figure 3). Its basic principle is similar to that of TCID50 but achieves higher quantitative precision

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[7]: the test virus sample is appropriately diluted and inoculated onto monolayers of adherent host cells, and then overlaid with agarose or methylcellulose medium to restrict viral spread within the cell layer. After replication in the initially infected cells, the released progeny viruses can only infect neighboring cells. At an appropriate virus dilution (typically 20-100 plaques per well), it can be assumed that one plaque originates from one infectious unit. By counting the number of plaques and multiplying by the dilution factor, the virus titer in the sample can be calculated (PFU/mL).

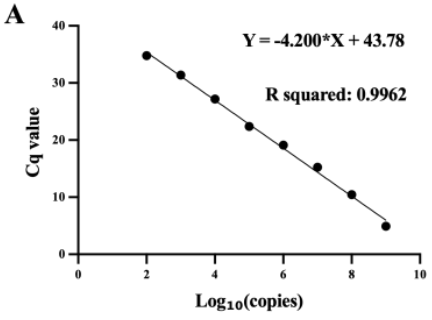
The influenza virus strain was serially diluted 10-fold and used to infect MDCK cell monolayers. The cells were overlaid with agarose medium and cultured for 72 h, and plaques were visualized by crystal violet staining. The plaque titer was determined to be 26,000 PFU/mL.

3.4. Real-time quantitative RT-PCR (RT-qPCR)

Real-time quantitative RT-PCR is currently the gold standard for influenza virus nucleic acid detection [8] and represents one of the most mature technologies in the field of molecular diagnostics. Its basic principle consists of two steps: first, viral RNA is reverse transcribed into complementary DNA (cDNA) by reverse transcriptase, second, a fluorescently labeled hydrolysis probe (such as a TaqMan probe) or a DNA-binding dye (such as SYBR Green) is added during PCR amplification. The fluorescence signal intensity in each cycle is monitored in real time by a fluorescence detection system, and the cycle threshold (Ct) value is recorded.Ct values are negatively correlated with the logarithm of the initial template copy number: the higher the viral RNA content in the sample, the fewer cycles are required to reach the set fluorescence threshold, and the lower the Ct value.

In terms of quantification strategy, RT-qPCR employs an external standard absolute quantification method: RNA standards of known concentration (usually in vitro-transcribed RNA or plasmid DNA containing the target sequence) are serially diluted 10-fold (10^1-108 copies/μL) and amplified in parallel with the test samples to establish a standard curve of Ct value versus initial copy number (Figure 4A). The Ct values of the samples are then substituted into the standard curve equation to calculate their absolute viral copy numbers (Figure 4B).

A



B

Group	Cq	Log_{10}(copies)	Copies	Mean copies
Replicate 1	18.36070158	6.05221391	1127752.788	1201873.199
Replicate 2	18.2214259	6.085374786	1217235.993	
Replicate 3	18.15753074	6.100587919	1260630.816	

Figure 4. Results of real-time quantitative RT-PCR

(A) Standard curve for absolute quantification of the influenza virus M gene. The M gene PCR product of the virus strain was serially diluted 10-fold, and the Ct values of each dilution standard were plotted against the logarithm of the initial template copy number to generate a standard curve. (B) Quantitative detection of viral M gene expression levels after MDCK cells were infected with influenza virus. The result was 1.2 × 108 copies/mL.

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3.5. Comprehensive comparison of the four methods

The comprehensive comparison of the four methods is summarized in Table 2. The four methods can be classified into three levels in terms of quantification [9, 10]:

The first level is antigen-based quantification: the hemagglutination assay measures the hemagglutinating activity titer of the virus through serial dilution, reflecting the relative amount of viral particles with hemagglutinating activity. It cannot distinguish between live and dead virus and is therefore a semi-quantitative method, suitable for preliminary identification of virus strains and as the standard reference in the WHO influenza surveillance network.

The second level is infectivity-based quantification: both TCID50 and the plaque assay are based on cell culture systems and detect infectious virus, thus distinguishing live virus from inactivated virus and best reflecting transmission risk. Both are absolute quantification methods, but the plaque assay achieves higher quantitative precision through direct plaque counting. These methods are suitable for vaccine potency testing, neutralization antibody detection, and basic virological research.

The third level is absolute nucleic acid quantification: RT-qPCR detects viral RNA copy number and does not distinguish whether the virus is infectious, but it has the highest sensitivity and specificity and is the gold standard for clinical molecular diagnosis. It is suitable for early diagnosis, low-load detection, and therapeutic monitoring.

Table 2. Comparison of the four methods

Method	Detection target	Quantification level	Quantitative capacity	Sensitivity	Detection time	Applicable scenarios
Hemagglutination assay(HA)	Viral hemagglutinating activity	Antigen-based quantification	Relatively weak	Relatively Low	0.5 hours	Preliminary virus strain identification, WHO influenza surveillance reference method
50% tissue culture infectious dose(TCID50)	Viral infectivity	Infectivity-based quantification	Relatively strong	Moderate	3 days	Vaccine potency testing, neutralizing antibody detection, basic virological research (no distinct plaques required)
Plaque-forming unit assay(PFU)	Viral infectivity	Infectivity-based quantification	Strong	Moderate	3 days	Vaccine potency testing, neutralizing antibody detection, basic virological research
Real-time quantitative RT-PCR(RT-qPCR)	Viral RNA copy number	Absolute nucleic acid quantification	Strong	Relatively high	2-4 hours	Not directly comparable.

4. Discussion

The four methods differ in their quantitative principles, accuracy, and applicability. The HA assay provides a simple and low-cost approach to evaluating viral hemagglutinating activity, making it suitable for standardized surveillance, including in resource-limited regions. However, its results are only semi-quantitative. The hemagglutination titer reflects the amount of viral particles with

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hemagglutinating activity but cannot distinguish infectious virus from inactivated virus or accurately reflect the infectious titer. Furthermore, the results are influenced by the density and spatial conformation of hemagglutinin on the surface of viral particles, and the relationship between hemagglutination activity and the total number of viral particles is not strictly linear.

Among the infectivity-based methods, TCID50 and the plaque assay both detect infectious viral particles, making them more suitable for evaluating viral infectivity than the HA assay. TCID50 is widely used in influenza virus vaccine potency testing, antiviral drug screening, and virus-host interaction studies. However, its relatively long turnaround time (typically 3 days) and dependence on cell culture systems impose requirements for biosafety facilities, operator proficiency, and experimental condition stability. In comparison, the plaque assay enables absolute quantification of infectious viral particles through direct plaque counting, whereas TCID50 relies on statistical estimation using the limiting dilution method and is subject to a certain degree of probabilistic error. The plaque assay is widely used for virus neutralization antibody detection, evaluation of antiviral drug efficacy, and construction of virus growth curves. Some influenza virus strains do not readily form distinct plaques on cultured cells, limiting its general applicability. Therefore, although the plaque assay offers higher quantitative precision, TCID50 is more broadly applicable because it does not require the formation of distinct plaques. In practical research, an empirical conversion relationship between TCID50 and PFU is sometimes used (typically 1 PFU ≈ 0.5–1 TCID50), however, this relationship varies depending on the virus strain and cell line.

RT-qPCR offers the highest sensitivity among these methods by detecting viral RNA copy number, with a reported limit of detection reaching 10^1–10^2 copies/μL and a wide linear dynamic range of 10^2–10^8 copies/mL. Through appropriate primer and probe design, it can accurately distinguish influenza A and B viruses and their subtypes, with good reproducibility. These characteristics make RT-qPCR a core tool for clinical influenza diagnosis, epidemic surveillance, and evaluation of antiviral treatment efficacy. However, the RNA detected by RT-qPCR may originate from infectious intact viral particles, non-infectious defective interfering particles, or free viral RNA fragments. RNA copy number does not have a fixed conversion ratio with TCID50 or PFU. In practical applications, the detection limit of RT-qPCR is generally considered to be approximately 100–1,000 times lower than that of TCID50. Therefore, samples that are PCR-positive but culture-negative may indicate low viral loads or the presence of residual non-viable viral material.

Despite its high sensitivity and specificity, RT-qPCR requires a professional molecular biology laboratory, a real-time fluorescence PCR instrument, and well-trained operators. Its turnaround time is approximately 2–3 hours, and its reagent costs are relatively high, which may limit its widespread application in resource-limited regions.

5. Conclusion

This study systematically compared four classical methods for influenza virus detection and quantification: the hemagglutination (HA) assay, the median tissue culture infectious dose (TCID50) assay, the plaque-forming unit (PFU) assay, and real-time quantitative reverse transcription PCR (RT-qPCR). They were classified into three quantification levels: functional activity, infectivity, and genomic abundance.

The four methods differ in their quantitative targets and application scenarios. The HA assay reflects hemagglutinin functionality and is suitable for standardized influenza surveillance, including as a reference method in the WHO influenza surveillance network. TCID50 and the plaque assay measure infectious virus titers, with the plaque assay providing higher quantitative resolution, they

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are appropriate for vaccine potency testing and virological research. RT-qPCR detects viral RNA copy numbers with the highest sensitivity and specificity and is the gold standard for clinical diagnosis. Therefore, method selection should depend on the specific research objective, and results from different methods should be interpreted according to their biological meanings, because RNA copy number is not equivalent to infectious viral titer.

This study has several limitations. The virus strain and cell lines used in this study were specific to this work, and different virus strains may yield varying results. Consequently, the results should not be directly generalized to all influenza viruses. Future studies should include broader validation across diverse strains to confirm the findings. In addition, the four detection methods discussed in this study are influenced by the sources of experimental materials and experimental procedures. The hemagglutination (HA) assay is influenced by experimental conditions, such as the source of red blood cells. The TCID50 and plaque assays are affected by cell status and interpretation criteria. RT-qPCR relies heavily on standard materials, primers, probes, and standard curves. In further studies, these protocols need to be strictly standardized. Future research could explore standardized conversion systems among different quantification methods, as well as integrated strategies that combine highly sensitive molecular detection methods with infectivity assays.

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