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Short communication

Rescue of eGFP-expressing small ruminant morbillivirus for identifying susceptibilities of eight mammalian cell lines to its infection

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ABSTRACT

Small ruminant morbillivirus (SRMV), formerly called peste-des-petits-ruminants virus (PPRV), is classified into the genus *Morbillivirus* in the family *Paramyxoviridae*. If genetically modified using reverse genetics, the SRMV would be a useful vector to express foreign proteins *in vitro* and *in vivo*. In this study, a recombinant SRMV was rescued by reverse genetics for efficiently expressing an enhanced green fluorescent protein (eGFP) *in vitro*. Based on green fluorescence-tracked characteristics of the recombinant SRMV, eight mammalian cell lines (BHK-21, F81, MDBK, RK13, MDCK, PK15, Vero and GT) were selected for identifying their susceptibilities to SRMV infection. The result showed that all cell lines could be infected with the recombinant SRMV but at different efficiencies. The Vero and PK15 cell lines showed the highest and lowest susceptibilities to its infection, respectively, if merely comparing the proportions of green fluorescence-emitting cells among eight cell monolayers.

Peste des petits ruminants (PPR) is an acute or subacute, highly contagious and economically important viral disease of small ruminants, characterized by high fever, oculonasal discharges, pneumonia, stomatitis and inflammation of gastrointestinal tract (Balamurugan et al., 2010; Gur and Albayrak, 2010; Khan et al., 2008). The etiological agent of PPR is peste-des-petits-ruminants virus (PPRV), which however has been renamed small ruminant morbillivirus (SRMV), according to the latest virus taxonomy of the International Committee on Taxonomy of Viruses (Liu et al., 2018). SRMV is classified into the genus *Morbillivirus* in the family *Paramyxoviridae*, and its genome is a single strand of RNA with negative polarity (Mahapatra et al., 2006), which encodes six structural (N, P, M, F, H and L) and two nonstructural (V and C) proteins in the order of 3'-N-P (V/C)-M-F-H-L-5'. The first PPR vaccine strain, Nigeria 75/1, was developed by serial passages of SRMV in cell culture. It has been mostly used for producing live vaccines, conferring immune protection for at least 1 (Rashid et al., 2010) and probably 3 years in sheep and goats.

Although effective systems of reverse genetics were established long ago for other morbilliviruses (Baron and Barrett, 1997; Gassen et al., 2000; Radecke et al., 1995), a platform to rescue SRMV was unavailable until reported by two independent groups a few years ago (Hu et al., 2012; Muniraju et al., 2015). More recently, we developed another reverse genetics system for recovery of recombinant SRMV Nigeria 75/1 strain to express a foreign protein (Liu et al., 2019). In the present study, a recombinant SRMV was successfully rescued for expressing an enhanced green fluorescent protein (eGFP) *in vitro* using the established platform of reverse genetics. This recombinant virus (rSRMV-eGFP) was serially passaged for RT-PCR analysis to confirm its recovery and for comparison of its growth kinetics with that of its parental virus. The passage-7 rSRMV-eGFP was used to infect eight different mammalian cell lines for identifying their susceptibilities to SRMV infection. The above-mentioned studies were performed as follows.

A genome sequence of SRMV Nigeria 75/1 strain (Genbank accession No.: HQ197753) was used for designing a full-length rSRMV-eGFP cDNA clone (Fig. 1A). An open reading frame (ORF) of eGFP (Genbank accession No.: KY295913) was flanked by the Kozak sequence (Kozak, 1987) at its 5' end to improve eGFP expression possibly. The modified eGFP ORF with *Not* I and *Pme* I sites was regulated both by an M gene start (GS) sequence and by a P gene end (GE) sequence. In the upstream region of cDNA clone, three guanine residues were added to the 3' end of T7 promoter sequence to enhance transcription efficiency (Martin et al., 1988). In the downstream region of cDNA clone, a hepatitis delta virus ribozyme (HDV-Rbz) sequence was introduced between the 3' end of cDNA clone and the 5' end of T7 terminator sequence. The 5'- and 3'-

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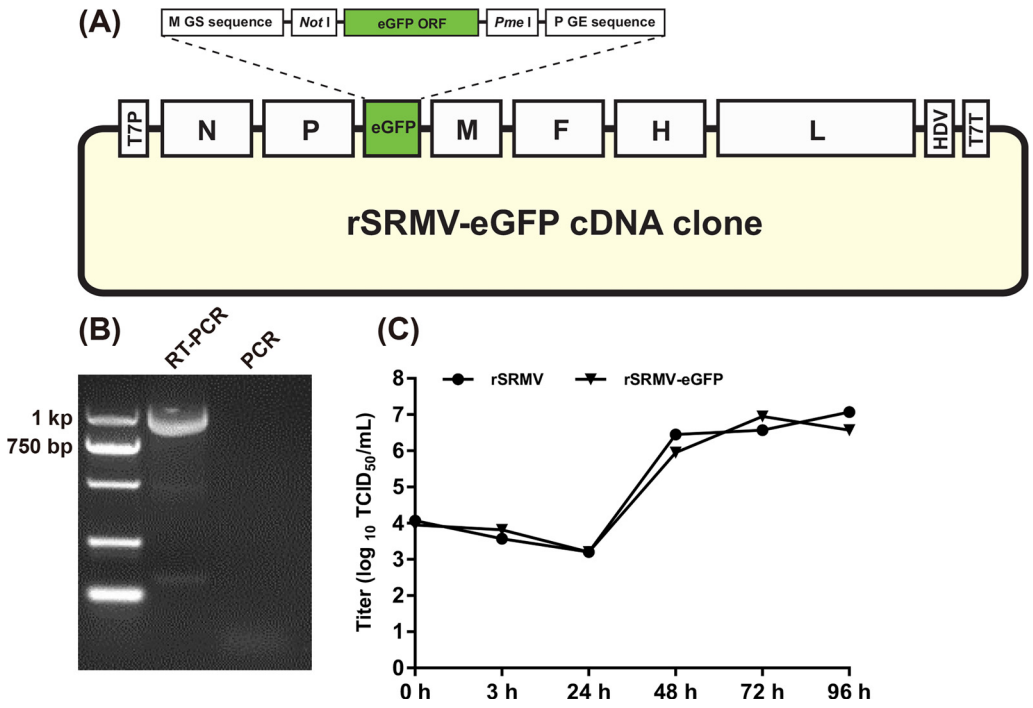

Fig. 1. Schematic representation of rSRMV-eGFP cDNA clone and characterization of rSRMV-eGFP. The 5'- and 3'-end-modifying rSRMV-eGFP cDNA clone was chemically synthesized and subcloned into the pBR322 plasmid (A). Total RNA was extracted from culture supernatant of the passage-5 rSRMV-eGFP for RT-PCR analysis. An expected 991-bp band of amplicon size was observed only on the lane RT-PCR, and as a control, PCR analysis showed no cDNA clone contamination affecting RT-PCR detection (B). The growth curve of passage-7 rSRMV-eGFP was generally similar to that of passage-7 rSRMV during the 96-h viral replication in VDS cells (C). GS: gene start; GE: gene end; ORF: open reading frame; eGFP: enhanced green fluorescent protein; T7P: T7 promoter with three extra G; HDV: hepatitis delta virus ribozyme; T7T: T7 terminator.

end-modifying rSRMV-eGFP cDNA clone was chemically synthesized, subsequently subcloned into the pBR322 plasmid, and finally purified using a PureLink™ HiPure Plasmid Maxiprep Kit (Thermo Fischer, Carlsbad, USA) according to the manufacturer's instruction. Other three recombinant plasmids, pCAGGS-N, pCAGGS-P and pCAGGS-L, had been constructed previously (Liu et al., 2019) to serve as helper plasmids for rescue of rSRMV-eGFP in this study.

The rSRMV-eGFP cDNA clone, pCAGGS-N, pCAGGS-P and pCAGGS-L were simultaneously transfected into BSR-T7/5 cells (Buchholz et al., 1999) for recovery of recombinant virus. Briefly, the BSR-T7/5 cells were seeded into a 6-well plate 1 d before co-transfection. Twenty-four hours later, a cell monolayer at 70–90% confluency was co-transfected with the rSRMV-eGFP cDNA clone (2.5 μg/well), pCAGGS-N (1.5 μg/well), pCAGGS-P (1.0 μg/well) and pCAGGS-L (1.0 μg/well) using Lipofectamine 2000 (Thermo Fischer, Carlsbad, USA), according to the manufacturer's instruction. Transfected cells were cultured at 37 °C with 5% CO_2 in DMEM supplemented with 10% FBS but without G418. On day 3 post co-transfection, the 6-well plate was observed by a fluorescence microscope, showing that a small number of cells had begun to emit green fluorescence (Fig. 2a). The proportion of fluorescence-emitting cells was growing over time: aggregated cells exhibited such a phenotype on day 5 post co-transfection (Fig. 2c), indicating expression of the eGFP and, more importantly, implying recovery of the rSRMV-eGFP. Another cell monolayer, which as a control was co-transfected with the cDNA clone, pCAGGS-N and pCAGGS-P but without the pCAGGS-L, did not show any green "dots" at all (data not shown).

The BSR-T7/5 cell line is non-permissive to SRMV infection, and thus can only be used for the co-transfection to rescue recombinant virus, rather than for its blind passage. Vero-Dog-SLAM (VDS) cell line can express the dog signaling lymphocyte activation molecule (SLAM, a major cellular receptor for morbilliviruses), and therefore is widely used for isolation and propagation of SRMV. The VDS cell line was also used for serial blind passages of the rSRMV-eGFP in this study.

The BSR-T7/5 cell monolayer was harvested on day 6 post co-transfection, and subjected to one freeze-and-thaw cycle for collecting supernatant. The supernatant was used to infect VDS cells for 3–5 h, and then replaced with growth medium with G418 for further incubation at 37 °C. When either green VDS cells especially or a typical cytopathic effect (CPE) (*e.g.*, syncytium formation) appeared during blind passage, it would basically confirm the successful recovery of recombinant virus. The number of green VDS cells was growing during the first passage (Fig. 2e), but without syncytium formation (Fig. 2f). The typical syncytium formation appeared during the second passage (Fig. 2g and h), and moreover, was always maintained in following passages (Fig. 2i and j). The phenotype of cell-to-cell fusion was even exacerbated to reveal a hyper-fusogenic CPE over time during a single passage (Fig. 2k and l).

The culture supernatant of rSRMV-eGFP at passage 5 was harvested for extraction of viral RNA by a High Pure Viral Nucleic Acid Kit (Roche Diagnostics GmbH, Mannheim, Germany). The extracted RNA was used as template for RT-PCR analysis using the PrimeScript™ One Step RT-PCR Kit (Takara, Dalian, China). The forward primer (5'-AGCCATTCT TGCCAAGCAGCCGTAA-3') targeted the downstream region of P ORF, and the reverse primer (5'-TATCAAAATCGTAGATCTCGGTCAT-3') targeted the upstream region of M ORF. The RT-PCR reaction underwent 50 °C for 30 min, 94 °C for 2 min and then 30 cycles at 94 °C (30 s), 55 °C (30 s) and 72 °C (70 s) in a thermocycler. To eliminate a false-positive result caused by residual contamination of the rSRMV-eGFP cDNA clone, the extracted RNA was simultaneously subjected to PCR analysis as a control using the same primer pairs. The PCR reaction contained 2 × PrimeSTAR Max Premix (Takara, Dalian, China) and underwent 35 cycles at 98 °C (10 s), 55 °C (10 s) and 72 °C (10 s).

RT-PCR and PCR products were detected by agarose gel electrophoresis, showing an expected band of amplicon size (991 bp) only on the RT-PCR lane (Fig. 1B, lane RT-PCR). As a control, PCR analysis did not indicate potential contamination of the cDNA clone affecting the RT-PCR detection (Fig. 1B, lane PCR). In addition, the Sanger sequencing showed that the passage-5-based RT-PCR product was totally identical to the 991-bp sequence, confirmed recovery of the rSRMV-eGFP from its cDNA clone.

Growth kinetics of the rSRMV-eGFP was compared with that of its parental virus *in vitro*. This parental virus was a recombinant SRMV Nigeria 75/1 strain (rSRMV) without any modification in its genome, which was rescued previously using reverse genetics at our laboratory. Briefly, VDS cells were plated into two T-25 flasks (4×10^6 cells/flask) for incubation at 37 °C up to 2 h. Both passage-7 viruses were inoculated into individual flasks (MOI = 0.01) for incubation at 37 °C. At

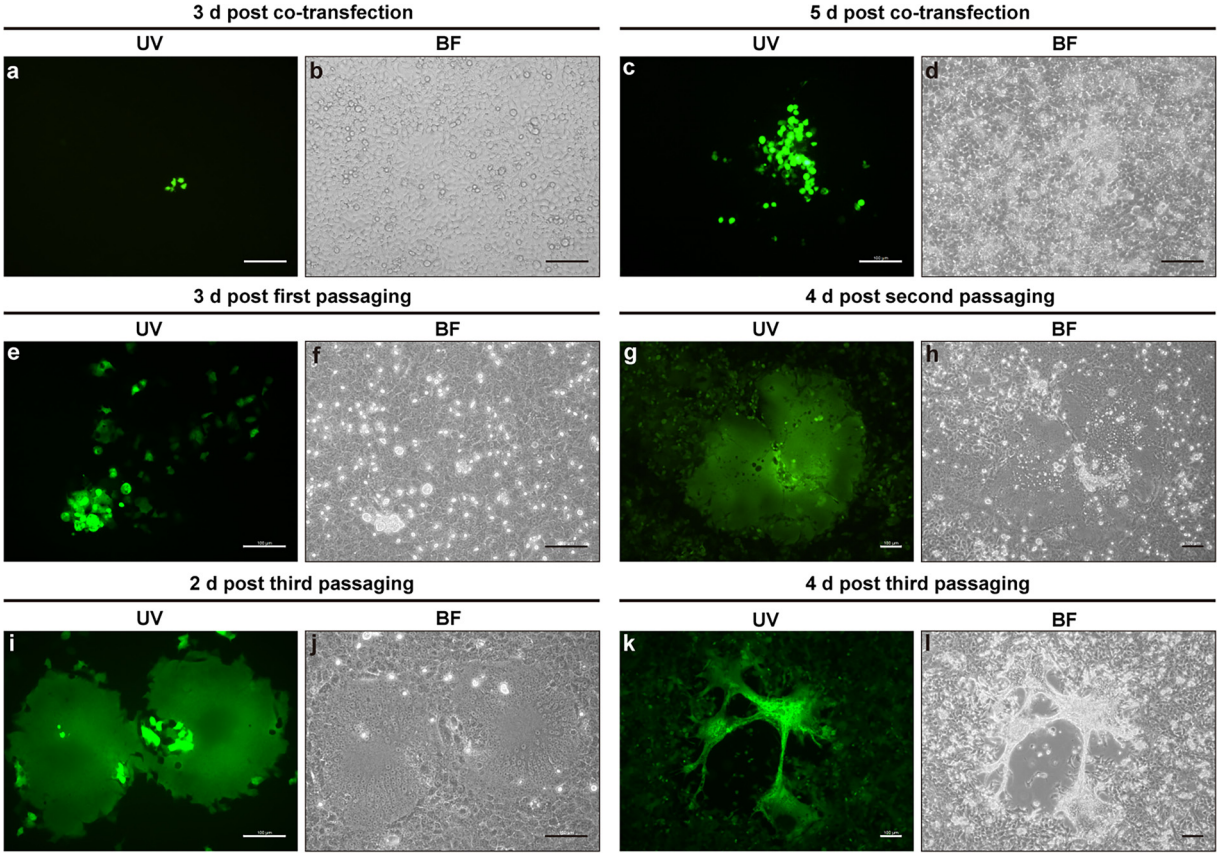

Fig. 2. Rescue and blind passage of rSRMV-eGFP. BSR-T7/5 cells were co-transfected with four recombinant plasmids. A few cells had begun to emit green fluorescence on day 3 post co-transfection (a), and more cells exhibited such a phenotype on day 5 post co-transfection (c). The rescued rSRMV-eGFP was subjected to serial blind passages in VDS cells. The number of green VDS cells was growing during the first passage (e) but without syncytium formation (f). The typical syncytium formation appeared during the second passage (g and h), was always maintained in following passages (i and j), and was even exacerbated to reveal a hyper-fusogenic phenotype over time (k and l). UV: ultraviolet; BF: bright field; Bar = 100 μm.

0, 3, 24, 48, 72 and 96 h post infection, 100 μL supernatants from infected cells were collected immediately for viral titration by 50% tissue culture infective dose ($TCID_{50}$) assay, respectively. The viral titer for each sample was calculated by the Spearman-Kärber equation (Finney, 1952). The growth curve of passage-7 rSRMV-eGFP was compared with that of the passage-7 rSRMV (Fig. 2C): the former was generally similar to the latter during the 96-h viral replication in VDS cells.

Some eGFP-expressing viruses have been proven to be convenient tools either for tracing pathways of virus infection *in vivo* (Ackermann et al., 2010; Guerra-Varela et al., 2018; Ludlow et al., 2012), or for identifying cellular susceptibilities to virus infection *in vitro* (Jiang et al., 2017; Suphatrakul et al., 2018; Yoneda et al., 2006). In this study, the rSRMV-eGFP was used for identifying susceptibilities of eight mammalian cell lines (BHK-21, F81, MDBK, RK13, MDCK, PK15, Vero and GT) (Table 1) to its infection. Briefly, eight cell lines and the VDS cells (as a control) were seeded into a 12-well plate. All cell monolayers at 70% confluency were infected (MOI = 0.1) with the rSRMV-eGFP, and subsequently cultured at 37 °C with 5% CO_2 in DMEM with 5% FBS. Forty-eight hours later, all rSRMV-eGFP-infected cell monolayers were observed under the fluorescence microscope.

The result showed that all eight cell monolayers more or less emitted green fluorescence, but none of them exhibited SRMV infection-like syncytium formation (Fig. 3). In the control group, numerous VDS cells not only emitted bright green fluorescence, but also had formed typical syncytia at 48 h post infection (Fig. 3, VDS and VDS BF). Therefore, compared with the highly susceptible VDS cell line, the others revealed low sensitivities to SRMV infection. Of them, the Vero (Fig. 3, Vero) and PK15 (Fig. 3, PK15) cell lines showed the highest and lowest susceptibilities, respectively, if merely comparing the proportions of green fluorescence-emitting cells among eight cell monolayers.

The Vero cell line was derived from kidney epithelial cells of an African green monkey. We simultaneously tested other two monkey-

Table 1
Eight mammalian cell lines used for identifying their susceptibilities to rSRMV-eGFP infection.

Characteristics	Cell lines								
	BHK-21	F81	MDBK	MDCK	PK15	RK13	Vero	GT**	VDS*
Organism	Hamster	Cat	Cow	Dog	Pig	Rabbit	Monkey	Goat	Monkey
Tissue	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney	Kidney	Testis	Kidney
Morphology	Fibroblast	Epithelial-like	Epithelial	Fibroblast-like	Epithelial	Epithelial	Epithelial	Epithelial	Epithelial
Culture properties	Adherent	Adherent	Adherent	Adherent	Adherent	Adherent	Adherent	Adherent	Adherent

* As a control, the VDS cell line is highly susceptible to rSRMV-eGFP infection.
** The GT (goat testis) is an immortalized cell line developed at our laboratory.

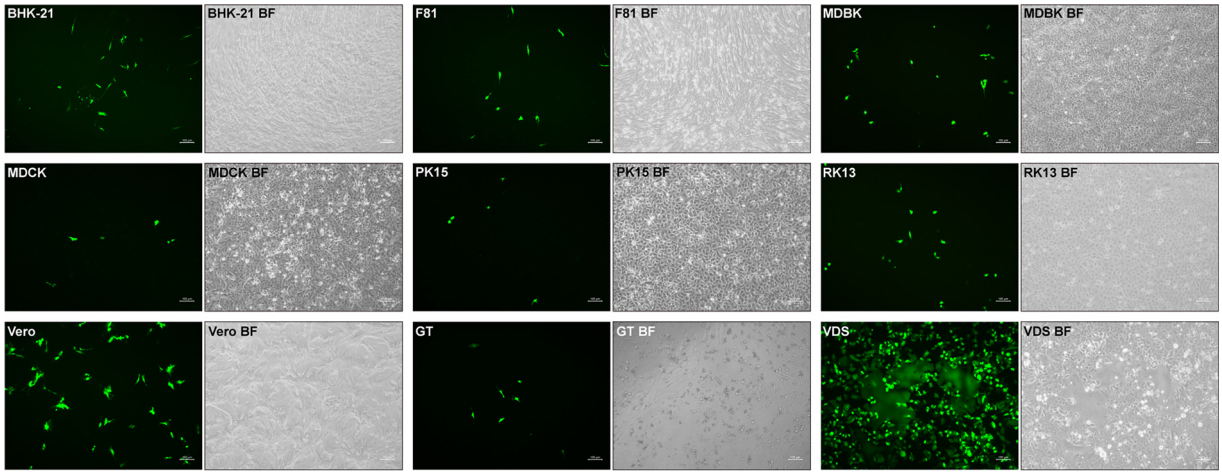

Fig. 3. Susceptibilities of eight mammalian cell lines to rSRMV-eGFP infection. Eight cell lines and the VDS cells (as a control) were infected (MOI = 0.1) with the rSRMV-eGFP for 48 h. All eight cell monolayers more or less emitted green fluorescence, but none of them exhibited SRMV-infection-like syncytia formation. Of them, the Vero and PK15 cell lines showed the highest and lowest susceptibilities, respectively, if merely comparing the proportions of green fluorescence-emitting cells among eight cell monolayers. In the control group, numerous VDS cells not only emitted bright green fluorescence, but also had formed typical syncytia. BF: bright field; Bar = 100 μm.

origin cell lines (CV1 and MARC-145) for determining cellular susceptibilities to SRMV infection, both showing significantly higher sensitivities than other seven mammal-origin cell lines in this study (data not shown). In addition to the eight mammalian cells, both embryonated SPF eggs and *Sf*9 insect cells were also inoculated with the rSRMV-eGFP. The former exhibited weak susceptibility to viral infection in its allantoic fluid, where an extremely small number of green "dots" appeared; the latter cultured at 27 °C never showed any rSRMV-eGFP infection-like phenotypes (data not shown).

The principal cellular receptor for morbilliviruses is SLAM, which is selectively expressed in immunocytes (Sato et al., 2012; Tatsuo et al., 2000, 2001). Although lymphoid tissues are major replication sites of morbilliviruses, it is also observed that they can infect and replicate in epithelial cells of other organs. Nectin-4 has been recently identified as the other morbillivirus receptor which is expressed in epithelial cells (Birch et al., 2013; Noyce et al., 2011, 2013). Cell lines, if modified to express SLAM or Nectin-4, would be highly permissive for efficient isolation and propagation of SRMV (Adombi et al., 2011; Fakri et al., 2016). As to entry pathways of morbilliviruses into cells, virus-to-cell fusion is a major route to release viral nucleic acids from infected into neighboring uninfected cells. However, it was recently reported that morbilliviruses could enter cells either by endocytosis (Goncalves-Carneiro et al., 2017; Yang et al., 2018) or by macropinocytosis (Delpeut et al., 2017). It remained unclear in this study which pathway was dominant for entry of rSRMV-eGFP into the eight cell lines.

In conclusion, the rSRMV-eGFP was successfully generated in this study using the platform of SRMV reverse genetics. A distinctive feature of the rSRMV-eGFP was the virus tracking *in vitro* which facilitated the identification of cellular susceptibility to SRMV infection. Eight different mammalian cell lines could be infected with the recombinant SRMV but at different efficiencies. The Vero and PK15 cell lines showed the highest and lowest susceptibilities, respectively, if merely comparing the proportions of green fluorescence-emitting cells among eight cell monolayers. The characteristics of virus tracking would contribute to sensitive pathological analysis on routes of virus spread *in vivo*.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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