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Isolation of feline viral rhinotracheitis agent and its antigenic properties

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ABSTRACT

Introduction. Viral respiratory diseases pose a serious problem for the health of cats all over the world. *Feline herpesvirus* type 1 (FHV-1) is the causative agent of a highly contagious infectious disease that induces upper respiratory tract infections, as well as conjunctiva and cornea ulcers. *Feline herpesvirus* 1 is considered the main etiological agent of respiratory infections in cats, and, according to experts, about 50–70% of respiratory infection cases are associated with this pathogen.

Objective. Isolation of feline viral rhinotracheitis agent from pathological material of diseased animals in cell culture; its identification and study of antigenic properties.

Materials and methods. To isolate the virus *in vitro*, a trypsinized primary feline embryo kidney cell culture was used, which was subsequently adapted to continuous feline kidney cell line CRFK. The causative agent of feline viral rhinotracheitis was detected by reverse transcription polymerase chain reaction and real-time polymerase chain reaction using specific primers complementary to the amplified complementary DNA fragments. The virus neutralizing antibodies were detected in the sera of immunized rabbits by virus neutralization test using continuous feline kidney cell CRFK monolayer.

Results. It was found that feline viral rhinotracheitis agent is detected in the conjunctival, nasal, and oropharyngeal swabs. It is replicated with the pronounced cytopathic effect in the primary and subcultured feline embryo kidney cells and in continuous feline kidney cell culture CRFK. The infectious activity titre 72 hours post inoculation was (6.50 ± 0.25) and (7.40 ± 0.22) lg TCID₅₀/mL, respectively.

Conclusion. *Feline herpesvirus* 1 isolate obtained as a result of the study and designated as Lavr strain, can be used for the manufacture of diagnostic products and means of specific prevention of feline viral rhinotracheitis in cats.

Keywords: feline viral rhinotracheitis, feline calicivirus infection, virus isolation, cell culture

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Выделение вируса инфекционного ринотрахеита кошек и его антигенные свойства

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РЕЗЮМЕ

Введение. Вирусные респираторные заболевания являются серьезной проблемой для здоровья кошек во всем мире. Герпесвирус кошек 1-го типа (*Feline herpesvirus* type 1, FHV-1) – возбудитель высококонтагиозного заболевания, вызывающий инфекции верхних дыхательных путей, а также язвы конъюнктивы и роговицы. Герпесвирус 1-го типа считается основным этиологическим агентом респираторных инфекций у кошек, и, по оценкам специалистов, около 50–70% случаев респираторных инфекций связаны с данным возбудителем.

Цель исследования. Выделение вируса инфекционного ринотрахеита кошек из патматериала от больных животных в культуре клеток, идентификация и изучение его антигенных свойств.

Материалы и методы. Для выделения вируса *in vitro* использовали первично трипсинизированную культуру клеток почки эмбриона котенка, затем адаптировали его к перевиваемой культуре клеток почки кошки CRFK. Детекцию возбудителя инфекционного ринотрахеита кошек осуществляли с помощью полимеразной цепной реакции с обратной транскрипцией и полимеразной цепной реакции в режиме реального времени с применением специфичных праймеров, комплементарных участкам амплифицируемых фрагментов комплементарной ДНК. Вируснейтрализующие антитела в сыворотке крови иммунизированных кроликов определяли в реакции нейтрализации с использованием монослойной перевиваемой клеточной линии почки кошки CRFK.

Результаты. Установлено, что вирус инфекционного ринотрахеита кошек обнаруживается в конъюнктивальных, назальных, ротоглоточных смывах,

реплицируется с проявлением выраженного цитопатического действия в первичной и субкультуре клеток почек эмбриона котенка и перевиваемой культуре клеток почки кошки CRFK, титр инфекционной активности через 72 ч после его инокуляции составлял $(6,50 \pm 0,25)$ и $(7,40 \pm 0,22)$ Ig TCD₅₀/см³ соответственно.

Заключение. Полученный в результате исследований изолят герпесвируса кошек 1-го типа, обозначенный как штамм «Лавр», может использоваться для изготовления диагностических препаратов и средств специфической профилактики инфекционного ринотрахеита у кошек.

Ключевые слова: вирусный ринотрахеит кошек, калицивирусная инфекция кошек, вирусывыделение, культура клеток

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INTRODUCTION

The causative agent of feline viral rhinotracheitis is a DNA-containing virus belonging to the family *Orthoherpesviridae*, the species *Varicellovirus felidalpha 1* (*Feline herpesvirus type 1*, FHV-1, *Felid alphaherpesvirus-1*). It is the cause of approximately 50–70% of all viral infections of the upper respiratory tract in domestic cats [1, 2, 3, 4, 5, 6]. The clinical signs of FHV-1-caused infection are usually manifested by ocular and respiratory lesions in cats. The nature and severity of the disease may vary depending on the clinical case. The main common signs of feline viral rhinotracheitis include conjunctivitis, stromal keratitis, corneal sequestrum and keratoconjunctivitis sicca, ocular and nasal serous discharge, sneezing, coughing and anorexia [7, 8, 9]. The virus replicates in epithelial cells of the conjunctiva and upper respiratory tract, affecting local neurons and establishing latency in the trigeminal, pterygopalatine, and craniospinal ganglia [10, 11]. Although virus replication is usually limited to the upper respiratory tract and conjunctiva, viremia has been detected during the acute phase of infection [12, 13]. Additionally, a case of non-suppurative meningoencephalitis has been described in cats, suggesting that FHV-1 may be capable of causing more invasive disease [14]. After the infection, the recovered cats become the virus carriers and periodically experience relapses, especially after stress [10]. It was previously reported that in cats, stress caused by lactation can result in the reactivation of latent FHV-1 infection, which further leads to the virus shedding and possible transmission of the infection. Cats of any age, sex, and breed are susceptible to the infection, but severe disease clinical signs are more common in kittens aged 2 to 6 months, and their mortality can reach 50% [9, 15].

In many studies, the prevalence of FHV-1 has been reported in both clinically healthy and diseased cats. The frequency of FHV-1 detection varied depending on the breeding site, country, and clinical condition of the cats [15, 16, 17]. Research by some scientists have shown that more than 90% of cats are seropositive to FHV-1, at least 80% remain latently infected, and 45% shed the virus throughout their lives [4].

All FHV-1 strains belong to the same serogroup, although minor genetic changes have been recorded for some strains [3, 6, 15, 18]. Gaskell R. and Willoughby K. reported

the genetic and antigenic similarity between FHV-1 and canine herpesvirus type 1 (CHV-1), as well as phocid herpesvirus type 1 (PhHV-1), as well as cross-protection between FHV-1 and PhHV-1 [19].

Cats with upper respiratory tract disorders are often co-infected with both FHV-1 and feline calicivirus (FCV), which creates great difficulties in clinical diagnosis as well as in isolating FHV-1 alone from biological material in cell culture, since most samples collected from cats are co-infected [20, 21, 22, 23]. Many researchers have reported the detection of co-infection using molecular methods, and the isolation of both viruses in cell culture has typically been used for the full characterization of the pathogen [24].

Since the mid-1970s, vaccination has been used to control the disease caused by FHV-1. Currently, both attenuated and inactivated vaccines are used in veterinary practice. Although they can reduce the severity of feline infectious rhinotracheitis, they do not prevent infection. Despite the widespread use of vaccines against feline viral rhinotracheitis and calicivirus in catteries, infection with these viruses still occurs frequently, especially when animals are housed in groups. One possible reason for this is that even cats vaccinated against these infections can become FHV-1 carriers after infection and can infect susceptible animals through contact. Another reason may be that kittens born to persistently infected mothers can become infected and experience herpesvirus infection in a subclinical or mild form before vaccination, when the level of maternally derived antibodies declines at 3–9 weeks of age [25, 26, 27].

Due to the high prevalence of feline viral rhinotracheitis in the feline population, continuous epizootological monitoring is necessary. Research aimed at isolating the virus and identifying it for further study will provide a theoretical basis for the development of new tools for specific prevention and control of herpesvirus infection in cats.

MATERIALS AND METHODS

Biological samples were collected from cats with suspected herpesvirus infection (24 nasal, conjunctival and oropharyngeal swabs) and delivered from veterinary clinics in Vladimir, Moscow, Vologda, Rybinsk, Nizhny Novgorod,

Table 1
Design of oligonucleotide primers for detection of DNA of feline viral rhinotracheitis agent using qPCR

Oligo-nucleotide	Oligonucleotide sequence 5'–3'	Amplified fragment range	Amplified fragment size
FHV-F1-66684	AGATTGCCGCCACCATCCTTC	66684...67221	538 bp
FHV-R1-67199	GATCTCCATTTGGTCGAGAGC		

Table 2
Thermocycling conditions for detection of DNA of feline viral rhinotracheitis agent using qPCR

Step	Sub-step	Temperature, °C	Time	Number of cycles
Pre-denaturation	–	98	3 min	1
qPCR	denaturation	95	15 sec	40
	primer annealing	60	30 sec	
	elongation*	72	30 sec	

* signal detection is carried out during the elongation step on the Green channel. Thermocycling was run for about 1.5 hours.

as well as from private cat owners in the period from 2020 to 2021. To confirm the FHV infection, the biological samples from the diseased cats were tested using reverse transcription polymerase chain reaction (RT-PCR) and real-time polymerase chain reaction (qPCR) with commercial kits "CALICIVIR" and "RINOVIR" manufactured by the Central Research Institute of Epidemiology of Rospotrebnadzor, Russia.

PCR procedure. To detect the DNA of the causative agent of feline viral rhinotracheitis, nucleic acid was isolated from biological samples using RIBO-sorb kit (Central Research Institute of Epidemiology of Rospotrebnadzor, Russia) in accordance with the manufacturer's instructions. RT-PCR and qPCR were performed using specific primers complementary to the sites of amplified complementary DNA fragments. The amplification results were recorded using TaqMan technology (with a hybridization-fluorescence probe) based on the quantification cycle (Cq) value. The oligonucleotide primers shown in Table 1 were used in the work. The thermocycling conditions are shown in Table 2.

Preparation of the viral suspension for cell culture infection. Samples of oral and nasal mucous membranes, as well as conjunctiva were collected with sterile cotton swabs, which were placed in sterile phosphate-buffered saline (PBS). They were then centrifuged at 3,000 rpm for 10 minutes, passed through filters with a pore diameter of 0.22 µm, and stored at -80 °C until use. The samples thus obtained were used to infect cell cultures.

FHV-1 isolation. For *in vitro* virus isolation, trypsinized and subcultured kitten embryo kidney cells were used. These were grown in Dulbecco's Modified Eagle Medium, DMEM (BioLoT LLC, Russia) supplemented with 10% fetal bovine serum (HyClone Laboratories LLC, USA), 100 U/mL penicillin, and 100 µg/mL streptomycin. The cells were grown under standard cultivation conditions: at (37.0 ± 0.5) °C in 5% CO₂ atmosphere and at 90% air humidity. The viral suspensions prepared from biological samples qPCR-positive for FHV-1 were inoculated in 500 µL

to the cell culture monolayer in T25 flasks (TPP, Switzerland) and incubated as indicated above (standard conditions). After the completion of 60 min adsorption period, DMEM with 1% fetal bovine serum was added. The infected cell culture was cultivated for 5 days. In total, at least 5 "blind" passages were performed before the virus cytopathic effect (CPE) was detected in the cell culture. When no CPE was observed during 5 passages, the sample was considered negative. If CPE appeared, the cells were frozen and thawed three times to isolate the virus. During the subsequent manipulations, the virus was adapted and cultured in the continuous feline kidney cell line CRFK (Crandell-Rees Feline Kidney), which was grown in culture DMEM supplemented with 10% fetal bovine serum (HyClone Laboratories LLC, USA), 100 U/mL penicillin and 100 µg/mL streptomycin and incubated as specified above (standard conditions).

Determination of FHV-1 infectivity titre. The infectivity titre was determined using CRFK cells cultivated for 24 hours in 96-well plates at a density of 1.5 × 10⁵ cells/mL and with 100 µL/well. FHV-1 dilutions were added and left for 60 minutes, then the supernatant was removed and replaced with 200 µL of DMEM containing 1% fetal bovine serum. The cells were monitored every 12 hours for 5 days and 50% tissue culture infectious dose (TCID₅₀) was calculated using Reed – Muench method.

Determination of the effect of multiplicity of infection on FHV-1 infectious activity. To study the effect of multiplicity of infection (MOI) on FHV-1 infectious activity, infectious doses of 0.0001; 0.001; 0.01, and 0.1 TCID₅₀/cell were used *in vitro*. The virus was inoculated to the continuous feline kidney cell line CRFK at the doses of 0.1; 0.01; 0.001 and 0.0001 MOI, respectively. The cell culture was harvested in 12, 24, 36, 48, and 72 hours after inoculation. The TCID₅₀ value was determined using the Reed – Muench method.

Animals. To evaluate the antigenic properties of the recovered FHV-1 isolate, 45-day-old rabbits weighing 1.5–3.0 kg (*n* = 4) were used. The rabbits were housed in the animal keeping facilities of the Federal Centre for Animal Health (Vladimir).

All procedures involving animals complied with the ethical standards adopted by the European Convention ETS No. 123, and were approved by the Bioethics Commission of Federal Centre for Animal Health.

Virus neutralization test procedure (VNT). Neutralizing FHV-1 antibodies were determined using VNT on the monolayer of continuous feline kidney cell line CRFK. The antibody titre was determined by preparing serial dilutions of the serum sample from the immunized rabbits, which was further added to the standard amount of the virus. Brief description of the test procedure: 50 µL of the diluted rabbit serum and 50 µL of the infectious culture medium containing 100 TCID₅₀ of the selected FHV-1 strain were mixed and incubated for 2 hours at (37.0 ± 0.5) °C and 5% CO₂. Upon the incubation completion, the virus and antibody mixture was inoculated onto CRFK cell cultures in 96-well CellBIND-treated flat-bottom microplates. Each rabbit serum dilution was tested using four wells per dilution. The cultures were incubated for 5 days at (37.0 ± 0.5) °C and 5% CO₂. The reaction was visually recorded using the Olympus CKX53 inverted microscope (Olympus Corporation, Japan). The titre of the virus neutralizing antibodies was determined as the reciprocal of the highest dilution that prevented infection of the cells.

Statistical analysis of the results. The received data was processed using statistical methods in Microsoft Excel. Mean group titres and standard deviation were determined.

The virus-neutralizing antibody titre in the rabbit serum was calculated using Kärber's formula and expressed as logarithms to the base 2 ($\log_2$).

RESULTS AND DISCUSSION

Biological samples collected from the diseased cats for the virus isolation were tested using RT-PCR and horizontal gel electrophoresis to detect the presence of the FHV-1 genome and using qPCR to quantify the FHV-1 DNA.

Detection of FHV-1 results was carried out by recording the values of the quantification cycles of the tested sample DNA (Fig. 1, Table 3).

Using PCR tests, the FHV-1 genome was detected in all 24 (100%) samples; the number of copies of the DNA of feline viral rhinotracheitis agent was approximately the same across all samples. A pairwise comparative analysis demonstrated that the nucleotide sequences of the 24 identified FHV-1 isolates were 99–100% homologous.

During the PCR, it was found that all 24 samples contained FHV-1 nucleic acid, of which 19 samples (Nos. 1–3, 5, 6, 8, 10–14, 16–23) demonstrated FCV and FHV-1 co-infection; in samples Nos. 4, 7, 9, 15 and 24, there was only FHV-1 genome; these samples were used in further work.

As can be seen from Table 3, the lowest accumulation of FHV-1 DNA was in samples Nos. 1–3, 5, 6, 8, 10–14, 16–23; the highest – in sample No. 4.

The viral suspensions prepared from samples Nos. 4, 7, 9, 15 and 24 (isolates V-07/21, M-06/20, NO-10/21, M-10/20 and MO-06/20, respectively) were inoculated in 500 μ L to the formed monolayer of the primary trypsinized kitten embryo kidney cell culture in T25 flasks.

After several passages in the primary trypsinized and subcultured kitten embryo kidney cell culture, cytopathic FHV-1 isolates (V-07/21, M-06/20, NO-10/21) were isolated, which were accumulated after 3 passages in the infectious titres of (6.50 ± 0.25) , (3.91 ± 0.14) and (2.75 ± 0.43) $\lg$ TCID₅₀/mL respectively. Infectious titre of FHV-1 isolates M-10/20 and MO-06/20 in kitten embryo kidney cell culture was at the level of (1.66 ± 0.38) $\lg$ TCID₅₀/mL during 2 passages and no CPE was observed by the passage 3. Therefore, these materials were not used for further work.

Since the nucleotide sequences of the cytopathic FHV-1 isolates are 99–100% homologous (according to the pairwise comparative analysis results), isolate V-07/21, which exhibited the highest infectious titre in primary trypsinized and subcultured kitten embryo kidney cells, was selected for further research. FHV-1 isolate V-07/21 was adapted and cultivated in the continuous feline kidney cells CRFK. The first signs of typical CPE were observed 24 hours after inoculation on the monolayer and they were manifested as cell swelling and rounding, while the integrity of the monolayer was preserved. At 48–72 hours after monolayer inoculation, cytogamy was observed, resulting in the formation of individual foci of large rounded cells; these eventually detached completely and remained in suspension in 96 hours.

In Figures 2A and 2B, it can be seen that the isolated V-07/21 FHV-1 isolate induced pronounced CPE in CRFK continuous cell line 72 hours after infection. The virus, after 3 passages, accumulated at an infectious activity titre of (7.33 ± 0.14) $\lg$ TCID₅₀/mL.

The results of studying the effect of multiplicity of infection on the infectious activity of FHV-1 isolate V-07/21 in CRFK cells showed that the lower the inoculum dose, the slower is the virus replication. The highest FHV-1

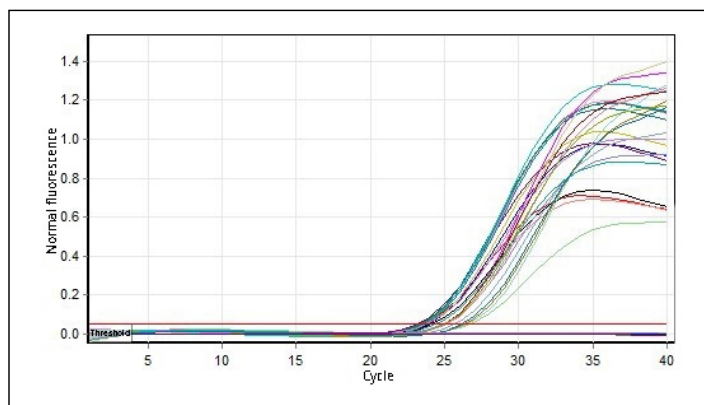


Fig. 1. Sigmoid curves of fluorescence signal accumulation during qPCR for the detection of FHV-1 DNA

Table 3

Results of testing the samples for the detection of the genome of feline viral rhinotracheitis agent

No.	Isolates	Quantification cycle (Cq) value	No.	Isolates	Quantification cycle (Cq) value
1	K-03/20	23.63	13	V-07/20	25.09
2	KV-03/20	23.76	14	M-04/21	26.67
3	KV-05/20	23.90	15	M-10/20	26.52
4	V-07/21	23.16	16	V-11/21	25.69
5	K-04/21	23.64	17	VL-09/20	24.97
6	VL-05/21	23.62	18	M-07/20	23.81
7	M-06/20	23.36	19	M-05/21	25.12
8	NN-09/21	24.14	20	Rnn-12/21	24.94
9	NO-10/21	23.45	21	VR-06/21	26.31
10	R-11/20	24.90	22	V-02/20	25.00
11	MO-08/21	24.17	23	I-04/21	24.42
12	R-06/21	23.64	24	MO-06/20	26.15

V-07/21 infectivity titre was reported 72 hours after inoculation at a dose of 0.01 MOI, reaching (7.40 ± 0.22) $\lg$ TCID₅₀/mL (Fig. 3).

To study the antigenic properties of FHV-1 isolate V-07/21, four rabbits were intramuscularly immunized with the inactivated viral material at a dose of 1.0 mL twice at a 21-day interval. The antibody titre against the antigen of feline viral rhinotracheitis agent in rabbit sera was determined using VNT before administration and at 21 and 35 days after immunization. Before the inactivated material administration, the rabbits were seronegative to FHV-1.

The results of the study of the antigenic activity of FHV-1 isolate V-07/21 in rabbits are shown in Figure 4. On day 21, the virus-neutralizing antibody titre against FHV-1 in the immunized rabbits after double administration of the inactivated virus-containing material averaged (4.91 ± 0.15) $\log_2$ SN₅₀ for the group; on day 35, the antibody titre in the sera reached a statistically significant value of (6.87 ± 0.59) $\log_2$ SN₅₀ ($n = 3$, $p < 0.05$), which indicates a high antigenic activity of the virus.

The recovered FHV-1 isolate V-07/21 was designated as Lavr strain, which was deposited as a production and challenge strain in the All-Russia State Collection of Exotic FMD Virus Types and Other Animal Pathogens of the Federal Centre for Animal Health under accession No. 457-dep/23-7-GKSHM Federal Centre for Animal Health. A patent was obtained for the strain: Patent No. 2806603 C1 "Lavr strain of

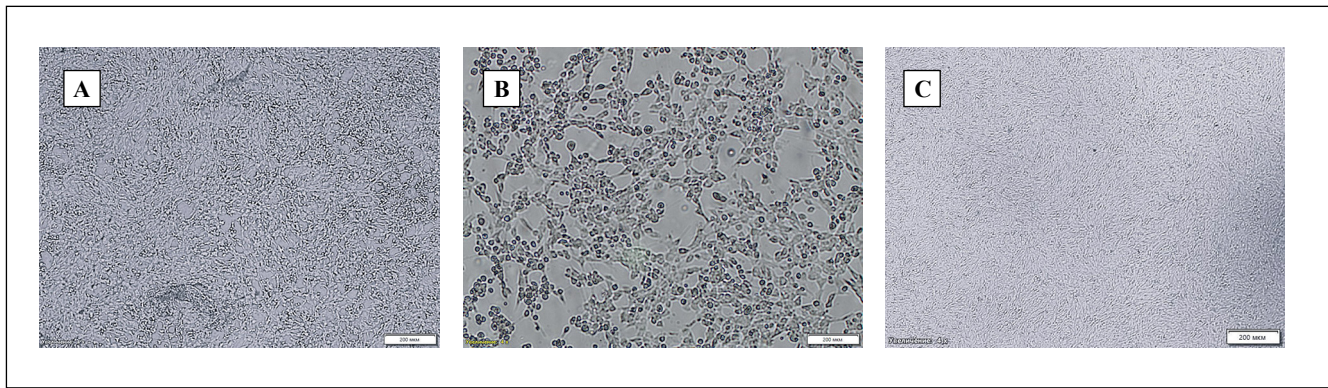


Fig. 2. Cytopathic effect of V-07/21 FHV-1 isolate in CRFK cells 72 hours after infection: A – magnification 4x; B – magnification 10x; C – control (non-infected) CRFK cells (magnification 4x)

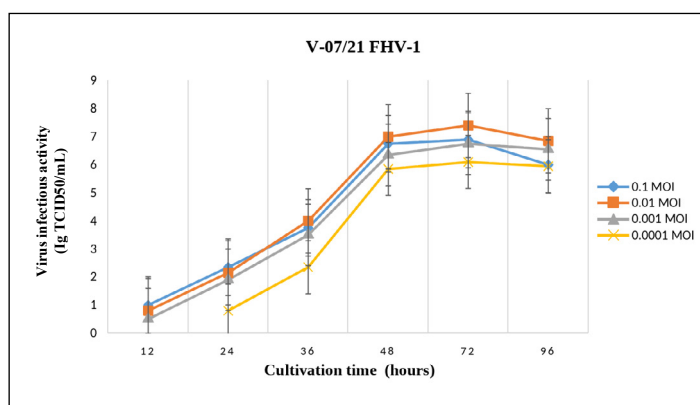


Fig. 3. Replication capacity of FHV-1 isolate V-07/21 at various inoculation doses and cultivation times in CRFK cell culture ($n = 5$, $p < 0.05$)

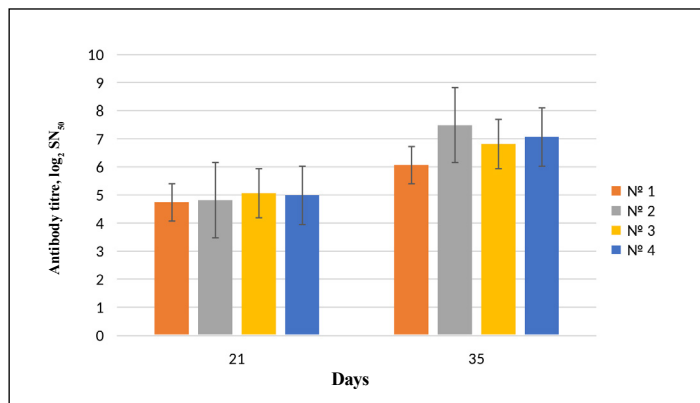


Fig. 4. Dynamics of seroconversion to FHV-1 V-07/21 in rabbits, according to VN test results ($n = 3$, $p < 0.05$)

Alphaherpesvirus 1 virus of infectious feline rhinotracheitis for production of biological products for diagnosis and specific prevention of infectious feline rhinotracheitis¹.

CONCLUSION

Analysis of the obtained study results allows for the conclusion that the feline viral rhinotracheitis agent is detectable in conjunctival, nasal, and oropharyngeal swabs. It has been established that primary and

subcultured kitten embryo kidney cells, as well as CRFK continuous feline kidney cell line, are equally sensitive to *Feline herpesvirus* type 1 (FHV-1), where the virus replicates with the manifestation of a pronounced cytopathic effect (CPE). However, in our opinion, the most promising cell culture for the reproduction of the causative agent of feline viral rhinotracheitis is the continuous feline kidney cell culture CRFK. The use of primary trypsinized and subcultured kitten embryo kidney cultures for subsequent virus cultivation is complicated due to their low practicality and the constant need for organ donors. The obtained study results can be used for diagnostic purposes and in the development of specific means for preventing feline viral rhinotracheitis.

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