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Bubaline alphaherpesvirus 1 induces a latent/reactivable infection in goats

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ABSTRACT

Latent infection is a common mechanism used by several alphaherpesviruses to persist in their host but it is not clear whether this mechanism is also triggered in heterologous infections. Cross-species infections have been documented repeatedly for alphaherpesviruses of ruminants, a group of closely related viruses. Herewith we report latent infection with bubaline alphaherpesvirus 1 (BuHV-1) in experimentally infected goats and subsequent virus reactivation after treatment with dexamethasone (DMS) at 10 months after infection. After DMS treatment, the virus was isolated in one such animal in the nasal swabs from day 3 to 9 post treatment and in the ocular swabs at day 6. The goat was euthanized 48 days after DMS treatment and viral DNA was detected by PCR in the trigeminal ganglia and in two cervical ganglia. Additionally, BuHV-1 DNA was detected by PCR in the trigeminal ganglia of the other 3 goats.

1. Introduction

Ruminant alphaherpesviruses are a group of seven closely related viruses. Cross-species infection studies have been performed in domestic and wild ruminants and serological investigations have shown cross reactivity among the viruses [1,2]. This may easily confound the serological diagnosis used for Infectious Bovine Rhinotracheitis (IBR) caused by bovine alphaherpesvirus 1 (BoHV-1) [1–4]. The potential role of ruminant species other than cattle as BoHV-1 reservoirs has been investigated thoroughly [5–11].

Several alphaherpesviruses are able to establish latent infections in their host [12–18] but it is not clear whether and to what extent this mechanism of virus persistence is also triggered in heterologous infections.

Bubaline alphaherpesvirus 1 (BuHV-1) generally induces subclinical infection in water buffalo (*Bubalus bubalis*). Recently BuHV-1 has been detected in an aborted fetus and in nasal secretion of buffaloes with respiratory signs [19,20]. In a recent study, we demonstrated that BuHV-1 is able to infect goats via intranasal route. The goats were inoculated intra-nasally with BuHV-1 and monitored clinically, virologically and serologically for 42 days. Two out 4 infected animals had a slight transient increase in temperature and the virus was shed for up to 12 days, and with titers as high as 10^5-$10^{5.5}$ $TCID_{50}$/50 µl. The goats shed the virus by the nasal route, but not by the ocular, genital or rectal routes. BuHV-1 was also detected in white blood cells of two animals in the first week post-infection [21]. The goats were maintained in the isolation unit for ten months after the end of the experimental infection study. Over the entire period, the goats were subjected to virological investigation every 15 days to monitor possible shedding of BuHV-1. However, virological testing did not reveal spontaneous re-activation of BuHV-1 in any of the animals. Gathering information on this aspect could be interesting to understand the possible role of goats in the epidemiology of BuHV-1. Herewith we report the experimental re-activation of BuHV-1 in the experimentally infected goats.

2. Materials and methods

The experiment was carried out in the Isolation Unit of the Department of Veterinary Medicine of the University of Bari following the National Guide for Care and Use of Experimental Animals. The experiment was approved and authorized by the committee responsible for animal welfare (OPBA) of the University of Bari and by the Ministry of Health (aut. n. 852/2015-R1).

2.1. Experimental design

This experiment is the extension of a previous experiment in which four adult goats had been infected intranasally with BuHV-1 [21]. Ten months after the experimental infection, three goats were euthanized following the authorized protocol (Ministry of Health, aut. n. 852/2015-R1), whilst one animal was used for experimental re-activation of BuHV-1. From the euthanized animals, trigeminal, cervical and thoracic

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ganglia were collected for detection of BuHV-1 DNA by PCR and serum samples were collected for evaluation of antibodies specific for BuHV-1.

The remaining goat was treated with dexamethasone sodium phosphate (DMS) (Dexadreson, Intervet Italia Srl) using a standardized protocol used to reactivate caprine alphaherpesvirus (CpHV-1) infection in goats [12]. Briefly, the goat was treated for 6 days with DMS receiving daily a total of 4.40 mg/kg body weight (bw), of which 2.75 mg/kg bw by intravenous administration (i/v), 0.55 mg/kg by intramuscular administration (i/m) in the morning and 1.10 mg/kg bw by i/m administration in the late afternoon. To avoid secondary bacterial infections, the goat was treated with antibiotics (Enrofloxacin, Baytril, Bayer AG, Leverkusen, RFT) for 10 days.

During the 20-day observation period, clinical examination was performed daily. Starting two days before the DMS treatment and then for 18 days post treatment (dpt), nasal and ocular swabs were collected using sterile dry rayon tip swabs (Aptacas.r.l., Italy) for virological investigations. Heparinized blood samples were also collected. Blood samples were taken the day before DMS treatment and 18 dpt to evaluate the antibody titer to BuHV-1 by a neutralization assay. Forty-eight dpt, the goat was euthanized and the ganglia were collected for detection by PCR of BuHV-1 DNA.

2.2. Virus isolation and titration

Swabs were transported to the laboratory within 1 h of collection at 4 °C temperature, dipped in 1.5 ml of Dulbecco Minimal Essential Medium (D-MEM) and centrifuged at 5000 × *g* for 5 min. The supernatant was treated with a 10% mixture of antibiotics (5000 IU/ml penicillin, 2500 µg/ml streptomycin, and 10 µg/ml amphotericin B) for 30 min at room temperature, serially diluted 10-fold, and inoculated in quadruplicate onto Madin Darby Bovine Kidney (MDBK) cells in 96-well microtiter plates. The plates were read after 5 days of incubation at 37 °C in a humidified atmosphere with 5% CO_2, based on the presence of cytophatic effect (CPE). The viral titers were calculated using Reed-Muench method and expressed as Tissue culture infective dose 50% $TCID_{50}$ /50 µl [22].

White blood cells (buffy coats) were obtained from heparinized blood samples using the standard density-gradient separation procedure (Lympholyte, CEDARLANE laboratories Ltd., Burlington, NC, USA) and washed twice with RPMI medium before the use. Buffy coats were used for virus isolation by cells freeze-thawing and used for titration.

2.3. DNA extraction and PCR

Viral DNA was extracted from swabs, buffy coats and ganglia using the commercial QIAamp tissue kit (Qiagen GmbH, Hilden, Germany), according to the instructions of the manufacturer. In order to confirm the viral identity, viral DNA was also extracted from MDBK cells inoculated with nasal and ocular samples and showing CPE.

The PCR was carried out using a pair of primers (BuHV1F 5′- GGC GGTGCAGGTGTAGTC-3′; BuHV1R 5′-CTCGCGCACGTCCGTCCTCAC GCT-3′) designed to amplify a sequence of 360 bp of the gene coding for the glycoprotein C (gene UL44) of BuHV-1 (accession nr. KF679678) (unpublished data). The PCR was carried out in a total volume of 50 µl containing 5 µl of DNA, 2 µl of 10 × PCR buffer, 1.5 mM $MgCl_2$, 1.25 mM of each dinucleotide triphosphate, 200 µM of each primer, 1.5 U of Takara LA Taq (Takara Bio, Inc.), and 5 µl of DMSO. The thermal profile consisted of 94 °C for 2 min followed by 40 cycles at 94 °C for 1 min (denaturation), 55 °C for 1 min (annealing), and 68 °C for 1 min (extension), followed by a final extension at 68 °C for 10 min. Ten µl of the PCR products was analyzed by electrophoresis through a 1.5% agarose gel after staining with GelRed™(Biotium, Hayward, CA, USA) and visualized using a UV trans-illuminator equipped with software data acquisition and image processing (Gel Doc, Biorad, Segrate Milan, Italy). The expected amplicon size was 360 bp in length.

2.4. Sequencing

The obtained PCR products were sequenced directly by Eurofins Genomics laboratories (Vimodrone, Milano, Italy). Sequence editing and analysis was carried out using a commercially available software package (Geneious version 9.1.8, Biomatters Ltd, Auckland, New Zealand).

2.5. Serological analysis

Serial two-fold dilutions of serum samples from 1:2 to 1:256, were mixed in 96-well microtiter plates with 100 $TCID_{50}$ (50 µl) of the BuHV-1 strain b6, isolated in Australia [23] kindly supplied by prof. E. Thiry, University of Liege. The plates were held for 90 min at room temperature before adding 20,000 MDBK cells to each well. Reading was done after three days of incubation at 37 °C in presence of 5% CO_2. The titer of each serum was expressed as reciprocal of the highest dilution of the serum that neutralized the virus.

3. Results

3.1. Clinical examination

During and after DMS treatment, the goat did not show general or local clinical signs of disease such as fever, anorexia or depression.

3.2. Virological results

3.2.1. Goats euthanized

BuHV-1 DNA was detected by PCR only in the trigeminal ganglia of 3/3 euthanized goats whilst BuHV-1 DNA was not detected in thoracic and cervical ganglia.

3.2.2. Goat treated with DMS

The goat shed the virus from the nasal route from 3 to 9 dpt, with a peak of viral excretion ($10^{2.50}$ $TCID_{50}$/50 µl) at 6 to 8 dpt (Fig. 1). The virus was isolated from the ocular swabs only once, on day 6 post-treatment. By PCR, BuHV-1 DNA was detected in nasal swabs on 8 occasions between 3 and 10 dpt and in the ocular swabs twice at days 4 and 6 post-treatment. The buffy coat samples were consistently negative for BuHV-1 by both virus isolation and PCR. The viruses isolated in cell cultures were characterized as BuHV-1 by PCR. Moreover, BuHV-1 DNA was detected by PCR in the trigeminal ganglia and in two cervical ganglia whilst the virus was not detected in the remaining cervical and thoracic ganglia.

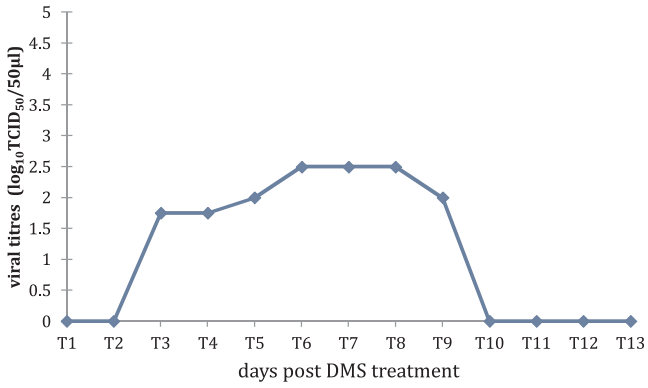

Fig. 1. Viral titers of BuHV-1 on MDBK cells from nasal swabs of the goat treated with DMS.

3.3. Sequencing

Sequence analysis of the PCR amplicons confirmed the specificity of the PCR amplification. We analyzed the gC gene sequences obtained from nasal and ocular swabs, from trigeminal and cervical ganglia of the DMS-treated goat and of the euthanized goats and from MDBK cells infected with the challenge virus [15] using BlastN [24]. The gC gene sequences displayed 100% nt identity to each other and to the BuHV-1 strain b6 (GenBank accession number KU936049) used in this study [25].

3.4. Neutralization test

The euthanized goats had BuHV-1 antibody titers ranging from 2 (one goat) to 4 (two goats). The DMS-treated goat had antibodies to BuHV-1. The initial neutralization titer (before DMS treatment) was 2 and it increased to 8 at 18 dpt.

4. Discussion

The results obtained from our experiments demonstrate that BuHV-1 is able to induce latent infection in an heterologous host, i.e. goats, and that the virus is reactivable following pharmacological treatment. Heterologous alphaherpesvirus infections among various ruminant species are not uncommon. BuHV-1, BoHV-5 and BoHV-1, CpHV-1, rangiferine alphaherpesvirus 1 and cervid alphaherpesvirus 1 (CerHV-1) are antigenically and genetically related viruses and they are all able to cross the species barrier [1,26]. However there are few studies on latency and reactivation of these viruses in heterologous host under either natural or experimental conditions. This information acquires high relevance in cattle as this phenomeon might confound the prohylaxis plans for BoHV-1, wherever enacted. Calves are susceptible to CpHV-1 and CerHV-2 infections [26]. CpHV-1 is able to induce antibodies cross-reacting with BoHV-1 and may induce latent, not reactivable, infections in calves [6]. CerHV-2 induces mild disease eliciting a serologic response but the virus is apparently not able to induce latent infection in calves [27,28]. On the contrary, calves do not appear suceptible to infection with CerHV-1 and elk alphaherpesvirus (ElkHV-1) [27,29–31]. Interestingly, calves are also susceptible to infection with BuHV-1 under experimental conditions [32]. Calves infected intranasally with BuHV-1 discontinuously shed the virus for 18 days after infection. Virus shedding and IgG_1 mucosal antibodies were detected in a sentinel calf housed in close contact with BuHV-1-infected buffaloes. Viral DNA was detected in the trigeminal ganglia of calves suggesting infection and latency of BuHV-1 in cattle [32].

In turn, it has also been observed that BoHV-1 can infect ruminants other than cattle, suggesting that this virus possesses an extended host spectrum in ruminants. Red deer and reindeer are susceptible to BoHV-1 but BoHV-1-specific antibody levels are low and virus shedding is limited. Also, latency and reactivation of the virus was not fully demonstrated [26,27,29,30] even if viral DNA was found in lymphoid tissue [33]. Epidemiological studies indicate that the seroprevalence rates for BoHV-1 in different deer species are low [34,35]. BoHV-1 infection has also been reported in sheep with fatal pneumonia or abortion and the virus is able to establish latent infection in the trigeminal ganglia [36,37]. Buffaloes are susceptible to both natural [38] and experimental infection with BoHV-1 [8]. BoHV-1 DNA has been detected in the trigeminal ganglia of experimentally infected buffaloes [8] but virus reactivation was not observed. BoHV-1 gE-deleted vaccine administered intranasally in buffalo calves induces antibodies able to cross-protect buffaloes preventing clinical signs and decreasing virus shedding after intranasal challenge with BuHV-1 [39]. Overall, the literature suggests that heterologous infections of alphaherpesviruses among ruminants may vary in their pathological/biological patterns and outcomes, chiefly in the ability of the virus to induce latent infection and reactivate.

We previously demonstrated that goats are also susceptible to experimental infection with BuHV-1. After experimental intranasal infection, none of the goats showed either local or general clinical signs [21]. In this study BuHV-1 was shown to induce latent infection in goats. One year after experimental infection with BuHV-1, viral DNA was detected in the trigeminal ganglia of the infected animals. Also, the virus was reactivated and shed by the nasal and ocular routes after DMS treatment of one such animals. Goats are also higly susceptible to natural and experimental infection with BoHV-1, showing respiratory signs, latency and reactivation/shedding of the virus [5,6,40]. Intranasal immunization of goats with a BoHV-1 gE^- deleted vaccine elicits cross-protective immunity against intravaginal challenge with CpHV-1 [41], indicating that the gE-deleted virus is able to replicate and induce active immunization. In this perspective, goats are a unique model, as they seem fully susceptible to infection with heterologous herpesviruses of large ruminants and they are also able to shed the viruses at high titers after reactivation, unlike what was observed in other domestic and wild ruminants [26]. This suggests that goats may represent a possible source of both BuHV-1 and BoHV-1 for large ruminants.

A major limitation of our study was the fact that trascriptomic profiling was not used to unveil the expression patterns of BuHV-1 in latent infections in goats or in its natural host. This information is not available for BuHV-1 whilst the latency mechanisms of BoHV-1 are being dissected by several studies [42]. However, we obtained firm evidence for reactivation of BuHV-1 in the caprine host by monitoring the clinical, virological and serological patterns.

Overall, our findings support a scenario in which related alphaherpesviruses may circulate in ruminant, thus potentially enacting novel epidemiological pathways. Identification of potential reservoirs of bovine alphaherpesviruses, i.e. animal species able to sustain circulation of potentially economically relevant livestock pathogens, should be considered when planning effective IBR eradication programs in European bovine populations. Also, considering that it is technically difficult to discriminate between BoHV-1 and BuHV-1-induced antibodies, the eradication plans should keep in consideration the possibility of contact of cattle with other ruminant species, harboring BoHV-1 or BoHV-1-related alphaherpesviruses.

Declarations of interest

None.

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