Detection of Caprine Herpesvirus 1 in Sacral Ganglia of Latently Infected Goats by PCR

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A study of the latency of caprine herpesvirus 1 (CpHV.1) was carried out with four latently infected goats. Three goats were treated with dexamethasone and euthanized after 4 and 6 days. PCR and virus isolation allowed us to detect CpHV.1 only in the third and fourth sacral ganglia of the two animals euthanized 6 days after the start of treatment.

Caprine herpesvirus 1 (CpHV.1) is associated with two different syndromes in goats, depending on the age of the animals at the time of infection. In 1- or 2-week-old kids, CpHV.1 causes an often lethal generalized disease affecting mainly the digestive tract (5, 11). In adult goats, however, the infection is mild or subclinical, with affected animals showing very mild signs of respiratory distress, vaginitis, balanoposthitis, or, occasionally, abortion (3, 6, 12).

CpHV.1 establishes latent infections, but, unlike other herpesviruses, its reactivation is extremely difficult to demonstrate under both natural and experimental conditions and very rarely has been reported (1, 4, 8). In natural infections, CpHV.1 reactivates during estrus, but only in animals with low neutralizing antibody titers (8). In previous studies, CpHV.1 was experimentally reactivated in adult goats by administration of a high dose of dexamethasone (DMS) for several days (1, 7).

It is interesting to note that, following reactivation or experimental infection, even when the virus was intranasally inoculated, CpHV.1 was shed from the vagina for longer periods of time than from the nose (1, 9). The results of those studies led us to hypothesize that CpHV.1 recognizes the genital tract as the main target tissue. Nevertheless, neither the pathogenesis of the cpHV.1 infection nor the latency sites of the virus in the goat tissues are presently known.

In this paper, we report the results of a study of the latency sites of CpHV.1 in goats by using the PCR assay. Four 3-year-old female goats latently infected with CpHV.1 were used. All animals had neutralizing antibody titers of 1:8 for CpHV.1.

Goat 1 was euthanized, and her ganglia were immediately collected and frozen. Goats 2, 3, and 4 were treated with DMS, as described in a previous paper (1). They were euthanized, respectively, at 4 (goat 2) and 6 (goats 3 and 4) days following the DMS treatment, and their ganglia were immediately collected. Thoracic, lumbar, sacral, and trigeminal ganglia were investigated by both PCR assay and virus isolation trials.

The PCR was carried out according to the protocol described in a previous paper (10). The viral DNA was extracted from 25 mg of each ganglion by using the QIAamp tissue kit (Qiagen GmbH, Hilden, Germany) and amplified by using a pair of primers corresponding to the sequences 759 to 779 and 1172 to 1154 of the gene encoding glycoprotein C (2). This pair of primers gave an amplification product with a size of 414 bp.

To attempt virus isolation, the ganglia were homogenized and inoculated into Madin-Darby bovine kidney (MDBK) cells. The strains isolated were identified by both virus neutralization tests and PCR.

Both the PCR and the virus isolation attempts were constantly negative when carried out with the ganglia of goats 1 (not treated with DMS) and 2 (treated for 4 days with DMS). The PCR results for goats 3 and 4 were positive, generating a 414-bp amplification product, but only when performed with the third and the fourth sacral ganglia of both animals. The virological investigations carried out with the same animals led to the isolation of CpHV.1, but only from the third and the fourth sacral ganglia.

The results obtained in the present study provide further evidence that CpHV.1 has a selective tropism for the genital tract of the goat, since the latency sites of the virus were discovered only in the corresponding sacral ganglia.

PCR was able to detect CpHV.1 genomic DNA only in the sacral ganglia of those animals (goats 3 and 4) who had been administered DMS for at least 6 days before euthanasia. In other words, CpHV.1 was detected only after the beginning of reactivation, probably as a consequence of the very low number of neurons infected by the virus.

The identification of the latency sites of the virus gives additional information about the pathogenesis of CpHV.1 infection and leads us to believe that the genital tract of the goat might be the most important route for the spread of the disease.

The results of this study are also interesting from the standpoint of comparative pathology: the genital tropism of CpHV.1, the lesions (the necrosis and ulcers) on the vulva, and the latency in the sacral ganglia are typical features also observed in the herpesvirus type 2 infection of humans. Our results suggest that CpHV.1 infection might represent a useful animal model of genital herpesvirus infection.

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