

In situ hybridization : a method to confirm the etiological diagnosis of parvovirus enteritis in dogs and cats?

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Objektyp: **Article**

Zeitschrift: **Schweizer Archiv für Tierheilkunde SAT : die Fachzeitschrift für Tierärztinnen und Tierärzte = Archives Suisses de Médecine Vétérinaire ASMV : la revue professionnelle des vétérinaires**

Band (Jahr): **132 (1990)**

Heft 8

PDF erstellt am: **17.05.2024**

Persistenter Link: <https://doi.org/10.5169/seals-593714>

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IN SITU HYBRIDIZATION: A METHOD TO CONFIRM THE ETIOLOGICAL DIAGNOSIS OF PARVOVIRUS ENTERITIS IN DOGS AND CATS?

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In situ hybridization was used to localize parvovirus in section from formalin fixed, paraffin embedded intestinal and lymphoid tissue from dogs with atrophic enteritis and cats with feline panleukopenia. The probe consisted of the pBR322 plasmid containing the entire VP1- and VP2-coding sequences of canine parvovirus. It was labeled by nicktranslation with biotinylated dUTP.

For the in situ hybridization the section was deparaffinized, endogenous peroxidase was inactivated by incubating with H_2O_2 and the tissue was digested by proteinase K. The probe was denatured on the slide at 98°C for 10 minutes and hybridization was performed over night at 42°C. The hybridization product was localized by incubating the slide with an avidin-biotinylated horseradish peroxidase complex

and the peroxidase activity was detected by amino-ethyl-carbazol and H_2O_2 as substrates.

The morphologic diagnosis of an atrophic enteritis correlated well with the presence of parvovirus in dogs. However, parvovirus could only be detected in 5 out of 9 cats with marked atrophic enteropathy. In this study we were able to confirm the etiologic diagnosis in several cases, but could not rule out a parvovirus infection, where the in situ hybridization yielded negative results. Whether this discrepancy between morphologic findings and results of the in situ hybridization reflected a) a lack of sensitivity or b) some cats had already eliminated the virus by the time of necropsy or c) the atrophic enteritis was caused by another agent, such as FeLV, still needed to be investigated.

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PATHOLOGY OF SWINE – A PORTRAIT OF ECONOMIC LOSS IN PIG PRODUCTION IN SWITZERLAND

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Economic loss in pig production due to runtting or premature death is one of the most important economic factors in Swiss agriculture. In 1971 the costs were estimated at about SFr. 100 million. Half of this loss was due to the spontaneous death of younger pigs, the other half was due to bad feed conversion caused primarily by lung affections or unfavourable husbandry conditions. In 1988, 1447 pigs were necropsied at our institute. A statistic evaluation of the findings is presented with an attempt to show a relationship between the diagnoses and the economic losses in Switzerland.

Necropsy results

75% of the pigs were younger than 2 months. An explanation for this age distribution is that deaths caused by infectious diseases predominate in pigs of this age and farmers thus show more concern for the aetiology. Runtting is infrequent.

Intestinal infections with *E. coli* were of greatest importance. One peak occurs in the first two weeks after birth and is caused by *E. coli*

strain O 149. The next peak is found in the second month after birth, i. e. after weaning. Here, the strains O 139 and O 141 dominate. In the next three months, intestinal infections with *E. coli* decrease and occur only after moving animals and the assembly of new groups.

The second major cause of death in younger pigs is septicemia. In the first month more than 60% are infections with *E. coli*, *Streptococcus* and *Erysipelothrix*. In the following months, *Haemophilus parasuis* (Glässer's Disease) often occurs. Affections of joints, skin or other diseases are relatively seldom, namely in only 13% of all the pigs under 2 months.

In the animals over 2 months, the respiratory tract was primarily affected; predominantly infections with *Mycoplasma hyopneumoniae*, *Pasteurella multocida*, *Actinobacillus pleuropneumoniae* and *Bordetella bronchiseptica*. The maximum incidence was in the 7 month-old pigs, where 100% had pneumonic infections. Respiratory diseases in SPF-pigs were rare.

Pigs with respiratory problems usually only tended to runt.