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EQUINE INFECTIOUS ANAEMIA VIRUS-INDUCED PULMONARY INTERSTITIAL DAMAGE: WHAT ABOUT AEROSOL TRANSMISSION?

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Introduction: Equine infectious anaemia (EIA) is considered a blood-borne disease primarily transmitted iatrogenically or by haematophagous insects and the virus infects cells of the monocyte/macrophage lineage. Alternative routes of transmission have not been explored, although there is evidence that EIA was spread via aerosolized particles during the 2006 EIA virus (EIAV) outbreak in Ireland.

Materials and Methods: Haematoxylin and eosin-stained sections of lung from 77 EIAV seropositive Romanian horses were scored based on lymphocyte infiltration, (peri) bronchiolar inflammation and thickness of the alveolar septa. Immunolabelling for p26 EIAV capsid protein expression and smooth muscle actin was performed.

Results: 52% of the EIAV-positive horses displayed a mild inflammation around the bronchi; 22%, moderate inflammation with inflammatory cells inside the wall and epithelial bronchiolar hyperplasia; and 6.5%, moderate to severe inflammation, with destruction of the bronchiolar epithelium and accumulation of smooth muscle cells within the pulmonary parenchyma. Interestingly, EIAV p26 was expressed in the cytoplasm of cells compatible by morphology and localization with alveolar and bronchiolar epithelial cells.

Conclusions: The observed lesions were compatible with the interstitial diffuse pneumonia observed during other lentiviral infections. The presence of EIAV capsid in lung epithelial cells suggests that EIAV might be responsible for the bronchointerstitial damage observed.

ACTIVATION OF THE EXTRINSIC PATHWAY OF APOPTOSIS DURING PRRS

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Introduction: Porcine reproductive and respiratory syndrome virus (PRRSV) is able to evade the host immune response. One of the strategies followed by PRRSV could be the induction of apoptosis. In this study we assess the expression of cleaved caspase (CCasp) -8, -9 and -3, TUNEL and PRRSV in tonsil and mediastinal lymph node of infected piglets.

Materials and Methods: Twenty-eight SPF piglets were distributed into groups of four, inoculated with PRRSV field isolate 2982 and killed sequentially at different time points. Control animals were mock-inoculated and killed at the end of the study. Samples of tonsil and mediastinal lymph node were collected at necropsy examination and fixed to perform immunohistochemical studies.

Results: Enhanced expression of CCasp8 was observed earlier in tonsils than in mediastinal lymph nodes. CCasp3 was increased at the end of the experiment. No significant changes were found for CCasp9 and TUNEL. Lymphocytes and macrophages mainly expressed CCasp8, CCasp9 and CCasp3. PRRSV expression showed two peaks, at 3 and 14 days post inoculation, in tonsil with only one peak at the beginning of the experiment in mediastinal lymph node.

Conclusions: The activation of the extrinsic apoptotic pathway by strain 2982 highlights that the apoptosis of crucial effector cells, such as lymphocytes or macrophages, might be a strategy used by PRRSV to evade the host immune response.

THE NOVEL HUMAN CORONAVIRUS EMC CAUSES DISEASE IN MACAQUES BUT NOT IN FERRETS

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Introduction: In 2012 a novel human coronavirus (HCoV-EMC) causing lower respiratory tract infection was discovered in human patients. The clinical course of the disease was similar to that seen in severe acute respiratory syndrome (SARS). However, the virulence and pathogenesis of the virus is not yet known and no animal models for human disease have been established.

Materials and Methods: Cynomolgus macaques and ferrets were inoculated with HCoV-EMC. At 1 or 4 days after inoculation the animals were killed and pathological, immunohistochemical, in-situ hybridization, virological and serological analyses were performed on samples from those animals. To determine the presence of the receptor for the HCoV-EMC dipeptidyl peptidase 4 (DDP4) in different cell types of the respiratory tissue, immunohistochemical analysis was used.

Results: HCoV-EMC caused mild respiratory disease in macaques, with non-ciliated bronchiolar epithelial cells as important target cells. Ferrets were not productively infected.

Conclusions: Our results confirm HCoV-EMC as the cause of respiratory disease in a non-human primate model. This suggests that the cynomolgus macaque is a suitable experimental animal species to model this disease in man.

LESIONS IN PERIPHERAL ORGANS OF RUMINANTS INFECTED WITH SCHMALLENBERG VIRUS

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Introduction: Schmallenberg virus (SBV), an orthobunyavirus of the family Bunyaviridae, causes epidemic abortion and birth of malformed or stillborn animals in ruminants. The main pathomorphological findings are skeletal malformations and abnormalities of the central nervous system (CNS), including hydrocephalus, cerebellar hypoplasia and micromyelia.

Materials and Methods: To further elucidate the changes in the organs of SBV-infected animals, paraffin wax-embedded material of ruminants originating from Northern Germany was investigated by haematoxylin and eosin staining, SBV immunohistochemistry and in-situ hybridization. Thirty calves, 13 lambs and one goat kid, as well as age-matched controls, were included in the study.

Results: Preliminary results comprised a moderate to severe muscular hypoplasia with fatty replacement, mild to moderate hepatocellular degeneration with interstitial fibrosis and biliary hyperplasia and mild lymphoid depletion of thymus and spleen in some SBV-infected animals. Single SBV antigen-positive cells were detected in one adrenal gland. However, this needs to be substantiated by further investigations. The majority of the animals showed no SBV-positive cells in the viscera.

Conclusions: Predominantly muscular and hepatic changes were found in SBV-infected and aborted ruminants, which were presumably caused by CNS lesions or represent residual lesions of systemic spread, respectively.