10th IEIDC Abstracts-Neurological

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Re-emerging West Nile virus in horses from South Eastern France, 2015

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West Nile virus (WNV) is a mosquito-borne flavivirus cycling in the wild avifauna. This virus has been shown to more frequently cause equine outbreaks of West Nile neuro-invasive disease in Europe for the last few years. Even if France had already registered ancient WNV circulation episodes in equids from 1962 to 2006, France had not faced West Nile outbreaks for nearly a decade. Two suspect cases with signs of meningoencephalitis (anxiety, impaired consciousness, decubitus) since August 11th and 17th were referred to the RESPE network and confirmed to be infected by West Nile virus on August 28th through the use of serological diagnostic tools (competition ELISA, IgM capture ELISA, flavivirus Luminex assay, Virus Neutralization Test) (Beck et al, 2015). So far, 30 equids with neurological symptoms have been found infected by West Nile virus, as well as 3 asymptomatic and 1 febrile horses, in three departments surrounding the Camargue area in South-Eastern France (Figure 1); the number of confirmed cases has been quite stable over the last 4 weeks (August 24th-September 20th), with an average of 7 cases a week (5-9). Clinical signs and evolution have been registered, and marked weariness and ataxia appear as the most striking features of this West Nile epizootics, reported in 14 and 17 cases out of 27 respectively (51.9% and 63.0% rates), while hyperthermia (over 38.5°C) is inconstant at neurological signs onset (7/27, 25.9%). West Nile infection has been lethal in 5 equids (lethality of 16.1%, CI95 [3.2-29.0%]). Several samples (EDTA blood, cerebrospinal fluid, urine) from confirmed West Nile cases have been investigated by West Nile real-time RT-PCR and have been found negative so far. Other samples are being processed in order to amplify and characterize the West Nile strain circulating in the Camargue area. No human case and only rare bird mortalities have been concurrently reported. A complete descriptive analysis of the French 2015 epizootics involving clinical, epidemiological and virological (whenever possible) analysis will be presented.


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Evaluation of immunity and clinical disease following infection of horses with Equine herpesvirus-1 and mutants of differing neuropathogenic potential

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Equine herpesvirus type 1 (EHV-1) is the cause of a devastating myeloencephalopathy (EHM) in horses worldwide, but our
understanding of its pathogenesis beyond the essential role of viremia is rudimentary. To address questions regarding viral and host factors for the pathogenesis of EHM, the focus of this study was to compare viruses with differing neuropathogenic potential. Four experimental groups of horses were established and included uninected controls, wild type infected horses (Ab4), polymerase mutant infected horses (Ab4 N752) and gD mutant infected horses (EHV-1 gD4). Animals were assessed for 21 days post infection (p.i.). Blood and nasal swabs were assessed for viremia and nasal shedding by real time PCR. Nasal secretions and cerebrospinal fluid (CSF) were collected for cytokine measurement and serum neutralization (SN) titers were determined at regular intervals. All horses were euthanized 10 weeks p.i. and tissues were collected for histology. Ab4 N752 infected horses showed the most severe respiratory disease followed by Ab4 infected horses. EHV-1 gD4 infected horses showed only mild respiratory disease. Three Ab4 infected horses developed signs of EHM but no horses in the other groups showed neurological signs. Furthermore, only Ab4 infected horses presented a classical bi-phasic fever. Ab4 N752 infected horses showed only primary fevers and EHV-1 gD4 infected horses showed only late secondary fevers. Nasal shedding and viremia differed significantly between infection groups, while EHV-1 SN increased in all groups p.i. to comparable levels. IFN-gamma was reduced and IL-10 production was completely inhibited in the CSF after Ab4 infection, but no significant changes were observed in any of the other groups. In contrast to CSF, IFN-gamma was significantly increased in nasal secretions of Ab4 infected horses. IFN-alpha was not detectable in nasal secretions before infection, but was significantly induced following infection in all infection groups. Finally, horses in the Ab4 infected group showed significant signs of inflammation and degeneration in the spinal cord, brain and testis upon histological examination, while inflammation was much milder or absent in horses of the other infection groups. In conclusion, we detect differences in respiratory and neurological disease as well as nasal shedding and viremia of horses infected with Ab4 and EHV-1 mutant viruses of differing neuropathogenic potential. These differences appear to be associated with a differing ability of WT and mutant viruses to infect immune privileged sites and modulate host responses and induction of interferons and IL-10.

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Are any microbes involved in the riddle of acquired equine polyneuropathy?

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Acquired equine polyneuropathy (AEP) is an emerging disease of horses in northern Europe since the 1990’s [1,2]. Today it is the most prevalent peripheral nerve disease in equids in Scandinavia. Affected horses show knuckling of the fetlock joints due to dysfunction of metatarsophalangeal extensor muscles, mostly in the pelvic limbs. In severe cases, paraplegia leads to euthanasia, with loss of about one third of the cases. Surviving cases normally recover after a long convalescence. The pathologic features are unique to the syndrome, with a large fibre neuropathy of peripheral nerves and pathology of the myelin-building Schwann cells. The disease is still an enigma despite scientists’ attention and many efforts to find its etiology. This presentation aims to discuss factors of importance to build a hypothesis for the etiology for AEP. Speaking for an infectious or toxico-infectious etiology is the typical course of the disease with several genetically unrelated cases appearing in one premises during a limited time period, and there are signs of spatio-temporal clusterings of outbreaks [3]. Often the outbreaks start at winter time, when horses are stabled in close contact. Also, some inflammatory changes are apparent in the nerves. On the other hand, if affected horses are moved, they do not transmit disease to new animals, which means that direct and indirect contact is not enough to transmit disease. Also, the horses do not display neither fever nor changes in their leukogram or inflammatory markers in blood. They are often seronegative for EHV-1, Borrelia and granulocytic anaplasma. A similar inflammatory nerve disease in humans is Guillain-Barré syndrome (GBS), also with unknown etiology. However, there is evidence that GBS in humans may be an immune-mediated sequel within 6 weeks after a gastrointestinal or respiratory infection like Campylobacter jejuni, Epstein-Barr virus, Mycoplasma pneumoniae and cytomegalovirus [4]. Sometimes vaccinations are also suggested to cause GBS. Which subclinical or clinical infections in a horse might be candidates for an autoimmune reaction to nerves? The main theory for AEP today is that wrapped forage has a part in the riddle. Could a forage-borne microbe or microbial toxin be the culprit? In conclusion, we need to work with open minds on this riddle at this point, and merge knowledge from various areas.

Acknowledgments

Thanks to Kaspar Matiasek, Ludwig-Maximilians University of Munich, for neuropathological analyses.

References


172 Bunyamwera virus, an emerging pathogen of veterinary importance in Argentina

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In Argentina the viruses with veterinary importance belong mainly to the genus Alphavirus (Eastern Equine Encephalitis virus, Western Equine Encephalitis virus) and Flavivirus (West Nile virus); the first showed high impact until the late 80’s while the second became important after the twenty-first century. Currently the Bunyamwera virus (BUNV) included within the Orthobunyavirus genus has emerged in our country as a