EQUINE

CHOROIDAL VASCULITIS DURING EXPERIMENTAL AND NATURALLY OCCURRING EHV-1 INFECTION IN HORSES. L.S. Goehring[1], G. Soboll Hussey[2], D.P. Lunn[3], L.K. Maxwell[4], E.J. Ehrhart[5]. Faculty or College of Veterinary Medicine at [1]Ludwig-Maximilians University, Munich, Germany., [2]Michigan State University, East Lansing, MI., [3]North Carolina State University, Raleigh, NC., [4]Oklahoma State University, Stillwater, OK., [5]Colorado State University, Fort Collins, CO.

Equid Herpesvirus type 1 (EHV-1) is the cause of respiratory disease, Equid Herpesvirus-associated Myelopathy (EHM), late term abortions and chorioretinopathies in horses. Following infection of the respiratory tract, a cell-associated viremia is often established leading to virus dissemination and endothelial cell (EC) infection of the spinal cord vasculature, the pregnant uterus or the eye. Previously, we showed focal or multifocal shotgun lesions in 50% or more of experimentally infected horses between 5 – 7 weeks post infection (p.i.). Based on this data our hypothesis was that eye lesions are caused by EC infection resulting in vasculitis similar to what occurs for EHM and that the incidence of EHM correlates with the incidence of ocular lesions.

To test this hypothesis, 8 mixed breed horses, aged 18 – 22 years, were infected with EHV-1 (strain OH03), and euthanized on day 14 p.i.. Clinical signs of neurological disease were evaluated between days 1-14 post infection and eyes were collected post euthanasia. In addition, eyes of a yearling horse that developed acute neurologic signs following experimental infection with EHV-1 strain Ab4 and one eye of a middle-aged gelding that developed neurological signs during the 2011 EHV Ogden, UT outbreak were collected. Neurological gait abnormalities of all horses were scored on a scale of 0 (no abnormalities) to 5 (severe abnormalities). In addition, ocular tissues were processed at Colorado State University and histological sections were collected and stained with H&E and immunohistochemistry (IHC) was performed using an anti-EHV-1 antibody. Eye lesions were described and degrees of choroiditis/vasculitis (CV) were scored according to severity and frequency on a scale from 0 (no lesions) to 3 (abundant lesions). A correlation coefficient between eye lesion score and clinical neurological gait abnormalities was determined and differences were considered significant at a p-value < 0.05.

The yearling horse and the Ogden-horse had severe, frequent CV (≥grade 2). In the OH03-study, 6/8 horses were graded ≥1; 4/8 horses were graded ≥2 and 2/8 horses were graded 3. IHC was positive in the yearling and in the Ogden-horse, while horses in the OH03experiment were positive only on occasion. A clinical neurological gait abnormality score during the OH03 infection experiment did not correlate well with eye lesions ($R^2 = 0.65$).

In summary, while the pathogenesis of ocular EHV-1 and EHM might be similar and viral antigen was detected in some of the infected horses, no significant correlation between neurological disease and ocular disease was detected. This lack of viral antigen in the eyes of some of the horses exhibiting neurological disease may have been due to the time point of euthanasia and a lack of sensitivity of the assays selected.

A NOVEL STRATEGY TO BOOST ANTIBODY PRODUCTION TO EQUINE HERPESVIRUS TYPE 1 (EHV-1) IN FOALS. G.A. Perkins, L.B. Goodman, S. Babasyan, H. Freer, A. Keggan, A. Glaser, S. Torsteinsdóttir, V. Svansson, S. Björnsdóttir, B.A. Wagner. Cornell University, College of Veterinary Medicine, Ithaca, NY.

Foals respond poorly to existing vaccines that are optimized for the adult immune system. Our hypothesis is that foals can be protected against EHV-1 infection by a vaccination that activates T-cell independent B-cell memory and antibody production.

Iceland is considered free of EHV-1. Pregnant Icelandic mares (n=15) were imported to an isolation facility in February 2012. The foals born in that spring suckled colostrum that was free of maternal EHV-1 antibodies. Foals either received equine IgE at birth by nasogastric tube and 2 days later recombinant EHV-1 glycoprotein C (gC) antigen intramuscularly (IM); or received EHV-1 gC antigen IM on day 2 of life; or received no treatment. At 7-months of age, foals were weaned and infected with EHV-1. Physical examinations were done daily. EHV-1 viral DNA and anti-EHV-1 gC antibodies were measured in the blood and nasal secretions before and after infection. Repeated measures ANOVA with Dunn's post-tests to compare individual groups were done.

All foals developed a fever. At 36-h post-infection (p.i.), the vaccinated foals had a significantly lower temperature ($p<0.05$). EHV-1 DNA was detected by qPCR in nasal swabs for the first week p.i. and there was no significant difference between groups. Serum and nasal anti-EHV-1 gC antibodies increased by day 8 p.i. and this was significantly higher in magnitude in the vaccinated foals ($p<0.001$).

Our novel equine neonatal vaccination provided some protection against EHV-1 clinical disease and prepared the foal's adaptive immune system to respond more rapidly and robustly to EHV-1 infection.

COMPARISON OF INNATE IMMUNE RESPONSES IN EQUINE RESPIRATORY EPITHELIAL CELLS TO MODIFIED-LIVE EQUINE INFLUENZA VACCINE AND RELATED WILD-TYPE INFLUENZA VIRUS. H.L. Pecoraro[1], D. Koch[1], G. Soboll Hussey[2], L. Bentsen[1], G.A. Landolt[1]. [1]Colorado State University, Fort Collins, CO., [2]Michigan State University, East Lansing, MI.

Equine influenza virus (EIV) is an important equine respiratory pathogen that continues to cause disease and suffering in horses. In order to design effective EIV control strategies, it is crucial to understand how to invoke long-term protective immunity. While systemic IgG titers have long been used as a correlate for protection from EIV infection, systemic antibody titers are not always accurate predictors of clinical protection. The cold-adapted, modified-live virus (MLV) vaccine (FluAvert I.N.®, Merck Animal Health) induces only weak systemic antibody responses. Despite this, the vaccine has been shown to provide clinical protection for up to 12 months after a single vaccine administration. This suggests that mechanisms other than humoral responses are critically important in EIV immunity. While the adaptive immunity to EIV has been well characterized, there is a lack of information regarding the innate immune responses occurring at the airway epithelium during EIV infection.

The objective of this study was to compare the innate immune responses of primary equine respiratory epithelial cell (EREC) cultures following infection with either the MLV vaccine strain or a closely related wild-type EIV strain (A/Equine/Kentucky/1/91; Eq/KY). It was our hypothesis that infection with the MLV vaccine strain elicits similar innate immune responses in equine airway cells as seen following infection with the wild-type virus.

Equine airway epithelial cells were collected from the upper and lower respiratory tract of respiratory healthy animals and the cells were grown at the air-fluid interface until fully differentiated. Subsequently, cell cultures were inoculated with a multiplicity of infection (MOI) of 10 with either the MLV strain or Eq/KY and incubated at either 30°C or 37°C. Twelve, 24, and 48 hrs after inoculation, cell culture subnatants and cells were collected and assayed for protein and mRNA expression of IFN-α, IFN-γ, IL-4, IL-6, IL-10, IL-17, TNF-α, and TLR3 & 9.

Inoculation of ERECs with either virus resulted in up-regulation of both mRNA and cytokine expression. The degree of up-regulation was dependent on virus strain, site of cell collection (upper versus lower respiratory tract) and incubation temperature used. Moreover, the expression levels appeared to be associated with viral replication efficiency.

These results highlight the fact that immunity to influenza virus is multifaceted and involves several arms of the host's immune response. Whether innate, antibody-independent mechanisms play a role in the observed clinical protection following intranasal vaccination with the MLV vaccine remains to be investigated.

INFECTIOUS DISEASES OF WORKING EQUIDS: HAVEMEYER WORKSHOP REPORT. N. Aklilu[1], A. Stringer[1], D.P. Lunn[2]. [1]SPANA, Ethiopia, [2]NCSU, Raleigh, NC.

A workshop was held in November, 2013 in Addis Ababa, Ethiopia, attended by 35 international delegates representing academia, NGO's, the OIE and government agencies. The goal was to identify ways to reduce the burden of infectious diseases in working equids worldwide.