FOCUS ARTICLE: Equine Encephalosis – an emerging threat

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Aetiology: The causative agent is Equine Encephalosis Virus (EEV), an orbivirus related to African Horse Sickness Virus (AHSV) and Blue Tongue Virus (BTV) from the family of Reoviridae. It was first isolated in 1967 in South Africa from blood and tissues of an affected horse. Seven non-cross reactive serotypes have been isolated so far (http://www.reoviridae.org/dsRNA_virus_proteins/ReoID/EEV-isolates.htm).
Epidemiology: Equine Encephalosis (EE) originates from southern Africa. EE is a vector borne disease which is transmitted by *Culicoides* midges, similar to AHS and BT and no vaccine is available. EEV appears to infect all equidae, but clinical signs are only seen in horses. Transmission depends on vector activity, which is seasonal in southern Africa (December - July). Studies have shown that around 50 - 60% of donkeys, zebras and horses are seropositive for EEV in South Africa. Serotype 1 seems to be predominant. Antibodies against EEV can be found rarely in elephants too.
Incubation period: Two to six days.
Clinical signs: The name Equine Encephalosis is misleading as it is not a primarily neurological disease.
Clinical signs consist of one to five days of fluctuating fever, accompanied by varying degrees of listlessness and inappetence; elevated heart and respiratory rates and red-brown discoloration of visible mucous membranes as a result of congestion and mild icterus. Most infections are subclinical and affected horses usually show only mild clinical signs and recover uneventfully. Mortality is generally less than 5% of infected animals. Less common but more serious signs can include various degrees of facial swelling; respiratory distress, sometimes with petechial haemorrhages in the conjunctivae and clear or blood-tinged nasal discharge; and signs of chronic heart failure. Pregnant mares may also abort during the first five to six months of gestation. Neurological signs including ataxia (particularly of the hindquarters), depression, frenzy, hyperexcitability and convulsions have been described in single cases but are more likely to be attributable to a cause other than EE (Equine Viral Encephalomyelitis, acute plant (e.g. leukoencephalomalacia following *Fusarium moniliforme* poisoning or chronic seneciosis) or chemical poisoning, Borna disease etc.).
Pathology: Post mortem findings can include lung oedema, hydropericardium, slight hepatomegaly and splenomegaly, petechiae in serosal surfaces (mainly intestines), hyperaemia of the glandular part of the stomach and in some cases congestion and oedema of the brain. Lesions are attributable to severe endothelial damage. There is no encephalitis.
Diagnosis: As infection with EEV is usually subclinical, most cases are confirmed by seroconversion in paired serum samples. Serological tests used include CF test (complement fixation), serotype specific SNT (serum neutralization test) and ELISA. In clinical cases virus can be isolated from heparinized blood and tissues (e.g. spleen, liver, thymus, lung and brain). The most recent outbreak in Israel 2009 was diagnosed by PCR (developed at Onderstepoort, South Africa).
Differential diagnoses: Non-specific febrile diseases, babesiosis, purpura haemorrhagica, AHS (mild form) and in the case of abortion Equine Herpes Virus 1, Equine Viral Arteritis, *Streptococcus. zooepidemicus*, *Klebsiella pneumonia* and others.
Control: Vector control and minimising exposure to infected *Culicoides* (stabling from before sunset to after sunrise, insect repellents).

EEV is not a notifiable disease in the UK or EU or to the OIE.

Importation of horses from affected area:
The rules for the movement and importation of horses into the UK is harmonised through the European Union and subject to European legislation (for further information Click here). EU legislation allows the import of live horses and their germplasm from approved Third Countries or their territories. EU rules require that all imported equidae from Third Countries are immediately checked (i.e. documentary, physical and identity checks) at the port of entry to the EU (Border Inspection Post - BIP) approved for the species. Currently the whole territory of Israel is authorised for imports of all categories of equidae and germplasm. According to TRACES (EU electronic notifications system) there have been no imports of horses from Israel in the recent months.

Literature:
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FOCUS ARTICLE: Surveillance of Equine Viral Arteritis (EVA) in the UK: 2005-2008

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Introduction

Since the first equine disease surveillance report published for the fourth quarter of 2004, Defra, AHT and BEVA have been collating and reporting data for a broad range of equine diseases submitted from a network of UK-based diagnostic laboratories and veterinary practices. Although these data have provided a regular insight into equine disease occurrence on a national scale across the UK, they have not previously been examined longitudinally. To gain insights into UK equine surveillance trends over time data accumulated over the past four years (2005-2008) have begun to be examined and will hopefully form a more regular feature in future reports. The first disease to be assessed in this way is equine viral arteritis (EVA).

EVA overview

EVA is an infectious disease of equidae caused by equine arteritis virus (EAV). Clinical signs include pyrexia and depression, frequently accompanied by marked conjunctivitis ("pink eye"). Swelling of the area around the eyes, the lower limbs, brisket, mammary glands in mares and sheath or scrotum in males is seen. EAV may cause abortion if it infects pregnant mares and can kill young foals. Spread is by both respiratory and venereal routes and persistent infection may occur that can be maintained for several years in the accessory glands of carrier stallions. Serological surveys in the USA, Australia, New Zealand and the UK during the last two decades all showed a markedly higher percentage of seropositive Standardbreds than Thoroughbreds, particularly among breeding Standardbreds more than racing horses. Recent EVA outbreaks in the USA, France, Israel and Croatia highlight the potential significance of this disease to horse breeding industries across the word and the need to remain vigilant to changing circumstances.

The UK situation

The first confirmed outbreak of EVA in the UK occurred in 1993 after which there was heightened awareness about control and prevention of the disease. EVA is specifically dealt with by the HBLB Codes of Practice (Click here) and the infection has been notifiable in stallions under the Equine Viral Arteritis Order (Click here) since 1995. The principal means of control in the UK are based on establishment of freedom from infection, achieved by pre-breeding serological testing of stallions and mares and vaccination of stallions. An inactivated whole virus vaccine (Artervac; Fort Dodge Laboratories) became available following the outbreak in 1993 and has since been used since then, especially in Thoroughbred stallions. The vaccine is generally not used in mares, however.

EVA testing data

During the period 2005-2008 data on serological testing of antibodies to EAV have been supplied by the Veterinary Laboratories Agency (VLA) and several commercial laboratories, including the AHT (together referred to as "non-VLA" laboratories). The VLA conduct serological testing principally for international trade purposes based on the OIE prescribed virus neutralization (VN) test. Following the emergence of problems with widespread use of the VN test for routine screening, especially among recently EHV-1 vaccinated mares, non-VLA laboratories now conduct testing using a screening ELISA,

followed where necessary by a confirmatory VN test conducted by a limited number of laboratories.
A reverse transcriptase polymerase chain reaction (RT-PCR) assay is used for detection of EAV in clinical and *post-mortem* specimens and has been used principally by the VLA and AHT to detect EAV in the semen of stallions. Samples tested by RT-PCR also undergo confirmatory virus isolation (VI) using rabbit kidney cell culture (RK-13). All 135 PCR/VI tests performed by VLA and AHT during 2005-2008 were negative for EAV.

Graphical representation of EVA data: 2005-2008

Figure 1 represents a summary by quarter for 2005-2008 of the total number of serological tests conducted by VLA and non-VLA laboratories (green and blue bars) and the two quarter rolling average proportion of positives identified among these tests (yellow and red lines).

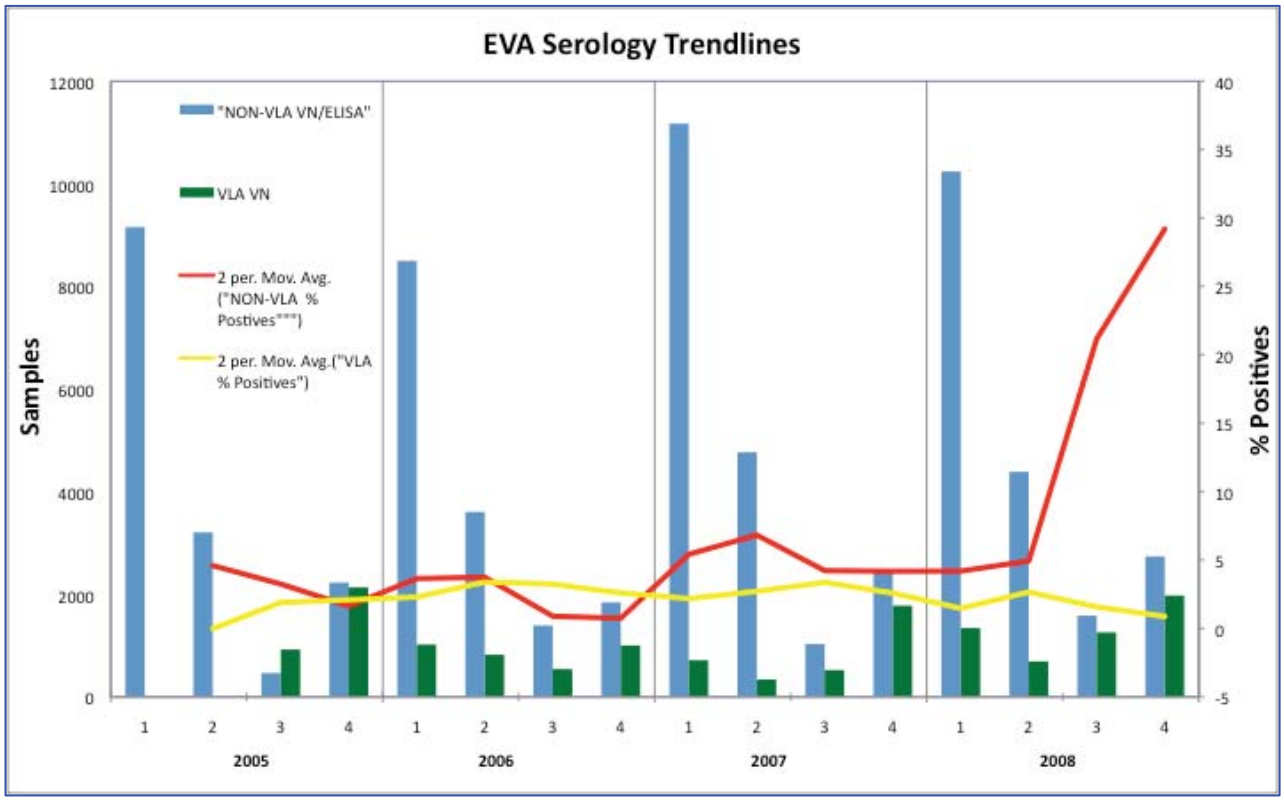

Figure 1: EVA serological tests and two period moving average proportion positive tests by quarter for 2005-2008 for VLA and non-VLA laboratories*

*No truly positive animals have been identified during this time period. All VN-positive animals were followed up according to a standard protocol to differentiate between seropositivity due to previous infection, vaccination or active infection/carrier status (stallions).

Features of interest to note in this summary for 2005-2008 are:

- The consistent seasonal pattern of numbers of tests conducted within each year, which was somewhat different between VLA and non-VLA laboratories. The pattern among non-VLA laboratories demonstrated a peak and subsequent decline from 1st to 3rd quarters with a pick up in numbers for the 4th quarter. This was consistent with pre-breeding testing in the first two quarters, with a predominance of Thoroughbred samples tested early in the first quarter. The fourth quarter increase represented pre-autumn sales testing as required by the major sales companies. VLA testing showed lower numbers of tests overall with a less pronounced peak

usually in the final quarter, consistent with less seasonally variable international trade testing requirements.

- The higher proportion of positive results seen in the first half of the year among the non-VLA samples during 2005-2007 was consistent with pre-breeding serological testing of stallions that had been vaccinated using Artervac EVA vaccine, in addition to testing of serologically positive mares, predominantly imported from other parts of the EU.
- For the period between the 2nd quarter 2005 and the 2nd quarter 2008, the proportion of positive results for both VLA and non-VLA samples were reasonably closely matched.
- In the second half of 2008 there was a marked rise in proportion positive among non-VLA samples that was not reflected among VLA samples. However, the explanation for this apparent peak of EVA positivity is largely artefactual related to changes in EVA testing practice during this period. Transfer of screening testing to in-house commercial ELISA-kits at several large veterinary practice laboratories that previously sent samples to specialist non-VLA laboratories, resulted in notably increased proportions of positive samples tested by confirmatory VN test at the specialist laboratories.

In conclusion, the data summarised in Figure 1 represent the considerable effort made among parts of the equine industry in the UK to maintain its vigilance against EVA. This is done in accordance with the recommendations that are outlined consistently each year in the HBLB Codes of Practice for annual serological retesting of breeding stock and re-vaccination of stallions and for the requirements also to test horses going through the sales ring. The data demonstrate consistent and predictable seasonal patterns of sample submission and seropositivity but also highlight the need when interpreting surveillance outputs for awareness about changes in practices that might notably influence underlying patterns in data.