Highlights in this issue:

- Hendra Virus infection: an emerging disease of horses and humans
- Focus article on Glanders
- Update on CEM and EIA outbreaks in the UK

Important note:

The data presented in this report must be interpreted with caution, as there is likely to be some bias in the way that samples are submitted for laboratory testing. For example they are influenced by factors such as owner attitude or financial constraints or are being conducted for routine screening as well as clinical investigation purposes. Consequently these data do not necessarily reflect true disease frequency within the equine population of Great Britain.
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INTRODUCTION

Welcome to the first quarterly equine disease surveillance report for 2010 produced by Department of Environment, Food and Rural Affairs (Defra), British Equine Veterinary Association (BEVA) and the Animal Health Trust (AHT). Regular readers will be aware that this report collates equine disease data arising from multiple diagnostic laboratories and veterinary practices throughout the United Kingdom giving a unique insight into equine disease occurrence on a national scale.

NATIONAL DISEASE OCCURRENCE

Equine Infectious Anaemia (EIA)
As reported in the last quarter’s issue, on 19th January 2010, the Department for Environment, Food and Rural Affairs (Defra) confirmed two cases of Equine Infectious Anaemia (EIA) by positive Coggins (agar gel immunodiffusion assay) in two horses in Wiltshire, England, following importation from Belgium having previously originated from Romania. In line with existing regulations, the infected horses were humanely destroyed. As of 30th April 2010 all remaining horses on the infected premises have been tested with negative results, and no further cases have been reported. The OIE has declared this event resolved.

Contagious Equine Metritis (CEM)
As of 5th March 2010, the Department for Environment, Food and Rural Affairs (Defra) confirmed Contagious Equine Metritis (CEM) in a 5 year-old Arabian stallion stabled in Devon. The stallion showed no clinical signs and it was found to be positive when tested for Taylorella equigenitalis (CEMO) prior to the mating season. A previous test on 21st January 2010 had been negative. The horse originated from another Member State of the European Union. The horse is under restriction and has been treated in line with the HBLB Code of Practice. Further epidemiological investigations are on-going. For more information about this outbreak, click here.
As of 23rd March 2010, Defra confirmed CEM in a 10 year-old Highland mare stabled in Durham, England. The affected mare is under restriction and treatment has commenced in line with the HBLB Code of Practice. A full investigation was undertaken by a veterinary officer on 23rd March 2010; results are pending. Click here for more information about this outbreak.

Atypical Myopathy
The University of Liege’s Equine Atypical Myopathy Alert Group (AMAG) has reported 83 clinical cases of atypical myopathy in the UK and Northern Europe during this spring as of 5th May 2010. Seven cases were reported from United Kingdom, although none of these cases were reported by any of the contributing laboratories in this report. The survival rate in the current European atypical myopathy outbreak is only 5%, lower than the previous epidemic which ended in December 2009 with a survival rate of 22%. For more information, click here.

Equine Influenza (EI)
On 11th May 2010 equine influenza (EI) was confirmed as the cause of a large outbreak of respiratory disease on a single premises in Lincolnshire, England. In addition, as of 21st May 2010 EI has been diagnosed among a small group of horses in a private yard in Shropshire, England.

These outbreaks have been the first alerts for 2010 in the new text alert service sponsored by Merial Animal Health, Tell-Tail. This service alerts practitioners to outbreaks of equine influenza in the UK by a text message...
to the practitioner’s mobile phone. If you are an equine veterinary practitioner and would like to sign up for this scheme, please register here. This service will be also offered to the members of the National Trainers Federation (NTF) in the following weeks.

If you would like to contact us regarding outbreaks of equine influenza virus or would like to sign up for our sentinel practice scheme, please send a message to: equiflunet@aht.org.uk or follow the link to www.equiflunet.org.uk for more information on equine influenza.

INTERNATIONAL DISEASE OCCURRENCE

Avian Influenza virus subtype H5N1
In April 2010 an article by Abdel-Moneim A.S. et al was published in the Journal of Biomedical Science and the abstract was posted in proMED with a link to the full paper (click here). In this article they reported on the isolation and characterization of highly pathogenic avian influenza virus subtype H5N1 from three affected donkeys in Egypt. The disease was recorded on 24th March 2009, one week after an outbreak of H5N1 infection in poultry in the village. The isolation of H5N1 avian influenza virus from donkeys in Egypt raises the concerns about the occurrence of non-H3N8 strains in horses since current vaccines would not be protective. However, the role of donkeys in spreading H5N1 virus to birds, humans or other mammals including equines needs to be assessed.

Contagious Equine Metritis (CEM)
In the U.S., one additional stallion (Holsteiner), currently located in the State of Wisconsin, has been confirmed positive for Taylorella equigenitalis, bringing the total number of carriers associated with the 2008/09 CEM event to 23 stallions and 5 mares. This stallion, though included in the exposed group of stallions and mares since 2009, was not finally tested for CEMO until early 2010. All T. equigenitalis (CEM)-positive horses, and all exposed horses that have been located, are currently under quarantine or hold order until determined as negative for CEMO. Testing and/or treatment protocols are being put into action for all located horses (click here).

Equine Viral Arteritis (EVA)
As of 7th May 2010 SENASA, Argentina’s National Animal and Agriculture Health Board, has suspended all horse transportation in the province of Buenos Aires for at least 2 weeks due to EVA virus. At the moment 8 outbreaks involving 196 cases and 808 susceptible horses have been reported. The index farm had abortions in mares that were with jumping mares artificially inseminated (30 to 40 days before the abortions) with semen imported from Holland. Animals from this farm went to other farms, where investigations are on-going; in some of them serologically positive animals have been found. All detected cases are related to mares inseminated with semen from the same stud. Investigations are ongoing; for more information click here.

On 10th May 2010 an outbreak of EVA has been reported in Claremorris, Co Mayo, West of Ireland. The outbreak has involved two stallions, six naturally infected mares and two test mated mares. The source of the outbreak is a stallion that was imported into Ireland and found to be seropositive on 27th October 2009. In 2010 confirmation was received that this stallion had no history of vaccination; subsequently the animal tested positive on PCR in March 2010 by the Central Veterinary Research Laboratory. Quarantine measures have been applied and investigations are ongoing.
Equine Infectious Anaemia (EIA)

As reported by the Montana Department of Livestock’s Animal Health Division, on 26th April 2010 EIA was reported in Gallatin Gateway, Montana, USA. A horse tested positive following a routine Coggins test prior to movement. As a result of the positive test, two more horses on the premises were tested and another positive was found. The two positive horses have been quarantined for transport to an EIA-positive herd. Fifty neighbouring horses have been tested and, as of 13th May, forty-five of those horses have tested negative. Investigations are ongoing; for more information click here and here.

As previously reported, following the investigation launched on 20th January 2010 after the UK reported having confirmed the disease in two horses of a consignment from Romania via Belgium, EIA was confirmed in one horse in Belgium on 2nd February 2010. This outbreak is ongoing and no further cases have been reported (click here). On the 18th of March, two separate but epidemiologically linked outbreaks involving one case of EIA each were reported in Fumal and Warsage (Liège), Belgium. The outbreak in Fumal involved a horse which had been imported from Romania in August 2009 and from which a blood sample was taken as a part of an epidemiological investigation on horses imported from Romania. The horse was subsequently euthanised. The outbreak in Warsage occurred in a riding school with 83 horses. Again, the positive case had been imported from Romania in June 2009 and a blood sample was taken on 8th March 2010 as part of an epidemiological investigation on horses imported from Romania. The horse was euthanised. For more information regarding these two outbreaks, click here. All horses having been in contact with the horses from Romania are being traced, movement controls are applied in the farms and the animals are being tested for the disease. In addition, on 25th May 2010, the Réseau d’Épidémio-Surveillance en Pathologie Equine (RESPE) (click here) has reported one case of EIA in West Flanders, Belgium. As with the previous EIA outbreaks in Belgium, this horse had been imported from Romania. There are 3 animals in the premises and no further details on the age, gender or breed of the affected horse have been reported. Investigations are ongoing.

Further to the previously reported EIA cases on premises in two different administrative districts in Germany, notice has been given that the investigations have been completed. The competent authorities considered the disease eradicated in the administrative districts of Kulmbach (Federal State of Bavaria) and Zollernalbkreis (Federal State of Baden-Württemberg). Consequently all implemented quarantine measures on the affected premises and within the established protection zones were removed (click here). As of 14th April 2010 EIA was reported in one horse in Bayern, Germany. There are 15 susceptible horses in the premises. Restrictions have been placed and the confirmed case has been euthanased. The event is still ongoing and the source of the outbreak is unknown (click here).

In France, one case of EIA was identified on 3rd March in Montcaret (Dordogne) and two further cases were diagnosed on one premises on 30th March in French Trotters in Prigonrieux (Dordogne). The outbreak in Prigonrieux is epidemiologically linked to the index outbreak (outbreak in Montcaret) since the affected horse in the index outbreak came from the outbreak in Prigonrieux. The three affected horses were euthanased. The French Ministry is performing an epidemiological inquiry and further information will be reported as it become available. For more information, click here.

On 25th May 2010 RESPE has also reported two cases of EIA in the county of Lika-Senj, Croatia (click here). To date 4 animals have been reported as affected out of a total of 27 animals; no further details on the age, gender or breed of the affected horses have been reported. Movement restrictions and tracing of animals moving from and onto the premises have been implemented.
Equine Infectious Anaemia (EIA) – Control
As of 4th May 2010 the European Union has decided to strengthen the measures taken by the Romanian authorities to prevent the spread of equine infectious anaemia (EIA) to other Member States. The decision is based on a Commission proposal that was endorsed by the Member States during a two-day meeting of the Standing Committee on the Food Chain and Animal Health (SCoFCAH). Equidae from Romania will now be transported to other Member States only from holdings that are certified free of EIA and under a comprehensive and specific regime, which includes double testing before dispatch. This decision reinforces traceability and post-arrival control measures in the Member States of destination. A possible future "regionalisation" of measures within Romania will be allowed in those areas where it is demonstrated that the disease has been successfully eradicated. For access to the press release, click here.

Equine Piroplasmosis
Regarding the Equine Piroplasmosis outbreaks in the US, as of early April 376 horses out of 2,172 tested were confirmed seropositive for Theileria equi. Presently, there are 292 antibody positive horses under quarantine on the index premises in southern Texas. Additional seropositive horses are being held under quarantine in 10 states, including Texas. Only one T. equi seropositive horse was detected out of 861 cohorts tested in 17 states; cohorts refers to horses with recent direct contact with positive trace-out animals. Testing of horses for interstate movement or movement to events has resulted in 17 T. equi positive horses being detected upon entering racetracks in New Mexico. Additionally, 14 seropositive horses epidemiologically unrelated to the index premises have been confirmed in Texas, and one in California. Tracing and testing of horses sold from the index premises in earlier years is ongoing. Additional to Amblyomma cajennense, specimens of Dermacentor variabilis tick removed from horses on the index premises have proved capable of transmitting T. equi. For more information of the US situation, click here.

Vesicular Stomatitis
As reported by the OIE on 27th May 2010, vesicular stomatitis virus (VS) has been confirmed in three horses in Arizona, USA. There are 5 susceptible horses in the premises and the source of infection is still unknown; the USDA Animal Plant Health Inspection Service (APHIS) and the Arizona Department of Agriculture have initiated a comprehensive epidemiological investigation of the event. VS was last reported in the USA in August 2009, and no other cases had been reported since then. The implications of this outbreak for international trade into the EU are still unknown; prior to the reoccurrence of this disease in the USA the European Commission had advised that no safeguard testing for VS would be required for horses certified from the USA coming into the EU after 1st February 2010, and that no pre-import testing for VS would be required for equidae certified from the USA to the EU after the date of 18th February 2010. For more information about this outbreak, click here.

Glanders
As of 21st April 2010 Glanders has been diagnosed in a horse in a university veterinary hospital in Brasilia, Brazil. The Official Veterinary Service began investigations and all the animals having had any contact with the affected horse have been tested with negative results. In Brazil, the disease is limited to certain areas, to some north-eastern States of the country, where it is endemic and, consequently, notified to the OIE in the six-monthly reports as present in a zone of the country. The disease has not been recorded in other regions of the country since 2008, when it was detected in the State of Sao Paulo. The preliminary outbreak assessment can be accessed online here.
On 28th April 2010 Glanders has been reported in Bahrain for the first time. This bacterial disease, caused by *Burkholderia mallei*, has already affected eight horses in the past three weeks, which have been euthanased. In addition, around a dozen horses currently have symptoms of the infection; however some of them are already showing signs of recovery. All horses suspected of either being infected or coming into contact with infected animals are being sampled and analysed in a specialist laboratory in the UAE (United Arab Emirates). Nearly 400 horses have already been sampled and the results from 10 of these horses are negative so far. Movement restrictions have been placed and investigations are ongoing. For more information, click here.

**Hendra Virus**

As of 20th May a horse has been confirmed positive for *Hendra Virus* in Queensland, Australia; subsequently it has been euthanased. As reported by Queensland Health, there is no indication of infection in any of the people on the property where the horse was kept. Even though there is another horse living in the adjoining property, it is not believed to be under threat. The source of infection is still unknown; however it is suspected that the virus could have been transmitted by a fruit bat colony in the area. For more information about this outbreak, click here.

**Focus articles**

In this report we are pleased to include two focus articles. Following the outbreaks in Brazil and Bahrain, in our first focus article Fatima Cruz from the AHT gives an overview on Glanders, an equine bacterial disease caused by *Burkholderia mallei*. We would like to acknowledge Ian Mawhinney from the Veterinary Laboratories Agency (VLA) for providing us with valuable comments and Holger C. Scholz from the Bundeswehr Institute of Microbiology in Munich, Germany for the pictures included in this focus article.

For our second focus article and, given the importance of this disease for the equine industry and the very recent outbreak in Australia, James R. Gilkerson and Colin R. Wilks from the University of Melbourne, Australia have prepared a focus article on Hendra Virus, a zoonotic viral disease affecting equids.

We reiterate that the views expressed in these focus articles are the authors’ own and should not be interpreted as official statements of Defra, BEVA or the AHT.

Access to all of the equine disease surveillance reports can be made on a dedicated page on the Animal Health Trust website at [http://www.aht.org.uk/equine_disease.html](http://www.aht.org.uk/equine_disease.html) or via the BEVA and Defra websites:


We would remind readers and their colleagues that a form is available on the AHT website for registration to receive reports free of charge, via e-mail, on a quarterly basis. The link for this registration form is available via

Virology Disease Report for the First Quarter of 2010

The results of virological testing for January to March 2010 are summarised in Table 1 and include data relating to Equine Viral Arteritis (EVA), Equine Infectious Anaemia (EIA) and West Nile Virus (WNV) from the Veterinary Laboratories Agency (VLA), Weybridge. The sample population for the VLA is different from that for the other contributing laboratories, as the VLA’s tests are principally in relation to international trade (EVA and EIA). VLA now provides testing for WNV as part of clinical work up of neurological cases on specific request and provided the local DVM has been informed.

Table 1: Diagnostic virology sample throughput and positive results for the first quarter 2010

<table>
<thead>
<tr>
<th>Serological Tests</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td>EVA ELISA</td>
<td>5486</td>
<td>103#</td>
<td>4</td>
</tr>
<tr>
<td>EVA VN</td>
<td>3191</td>
<td>71#</td>
<td>3</td>
</tr>
<tr>
<td>VLA EVA VN</td>
<td>543</td>
<td>31#</td>
<td>1</td>
</tr>
<tr>
<td>EHV-1/-4 CF test</td>
<td>542</td>
<td>28*</td>
<td>1</td>
</tr>
<tr>
<td>EHV-3 VN test</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>ERV-A/B CF test</td>
<td>268</td>
<td>5*</td>
<td>1</td>
</tr>
<tr>
<td>Influenza HI test</td>
<td>287</td>
<td>8*</td>
<td>1</td>
</tr>
<tr>
<td>EIA (Coggins)</td>
<td>3170</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>EIA ELISA</td>
<td>3732</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>VLA EIA (Coggins)</td>
<td>875</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>VLA WNV (PRNT)</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Virus Detection</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>EHV-1/-4 PCR</td>
<td>88</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>EHV-2/-5 PCR</td>
<td>7</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Influenza NP ELISA**</td>
<td>199</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Influenza Directigen</td>
<td>104</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Influenza VI in eggs</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>EHV VI</td>
<td>241</td>
<td>47</td>
<td>1</td>
</tr>
<tr>
<td>EVA VI/PCR</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>VLA EVA VI/PCR</td>
<td>6</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Rotavirus</td>
<td>74</td>
<td>38</td>
<td>7</td>
</tr>
</tbody>
</table>

ELISA = enzyme-linked immunosorbent assay, VN = virus neutralisation, VLA = Veterinary Laboratories Agency, CF = complement fixation, HI = haemagglutination inhibition, Coggins = agar gel immuno diffusion test, PCR = polymerase chain reaction, NP = nucleoprotein, VI = virus isolation, EVA = equine viral arteritis, EHV = equine herpes virus, ERV = equine rhinitis virus, EIA = equine infectious anaemia

# = Seropositives include vaccinated stallions, * = Diagnosed positive on basis of seroconversion between paired sera

** = The relatively high number of NP ELISA tests performed is largely due to requirements for international equine movement. All horses travelling to Australia must now have 2 NP ELISA tests performed prior to travel. The figures above include tests performed for international trade purposes.
Of the 31 EVA VN positives detected by the VLA, 19 were samples for private testing, 6 were export samples, 2 were serum samples from a stallion for artificial insemination (AI) certification, 3 samples were from overseas and one sample was from an imported horse. The 6 semen samples received for EVA testing were all negative on virus isolation and RT-PCR.

The 875 agar gel immuno diffusion tests for EIA (AGID; Coggins) were conducted for international trade purposes. Two samples from imported horses tested positive (please see the EIA outbreak in the UK explained in the National Disease Occurrence in the introduction of this report).

**Virological Diagnoses for the First Quarter of 2010**

**Equine Infectious Anaemia (EIA)**
As previously reported, two cases of EIA were confirmed in this quarter. For more information please refer to the National Disease Occurrence in this issue (page 3).

**EHV-1 Abortion**
One small outbreak, nine single cases of EHV-1 abortions and a case of neonatal death due to EHV-1 have been reported in this quarter. The small outbreak reported involved two abortions in vaccinated Thoroughbred mares on a stud farm. Of the nine single cases of EHV-1 abortions, one was diagnosed in a stud farm which reported another EHV-1 abortion in December 2009. All the mares involved in these nine single abortions were Thoroughbreds, with the exception of two. EHV-1 was diagnosed by PCR and virus isolation in placenta and/or fetal tissues. In a Thoroughbred stud farm a case of neonatal death was diagnosed to be due to EHV-1 by histopathology in fetal tissues.

**EHV-1 Neurological Disease**
As previously reported, in this quarter neurological EHV-1 was diagnosed by PCR in spinal cord tissues taken from a seven year-old Thoroughbred mare sent for post-mortem examination with a history of recumbent paralytic disease requiring euthanasia. The mare was in race training in a yard in southern England and all horses on the yard were current for EHV-1/-4 vaccination.

EHV-1 was confirmed by virus isolation from nasopharyngeal (NP) swabs and/or heparinised blood samples in 12 of the 32 animals in the premises. The yard and their veterinary surgeons worked closely with the British Horseracing Authority and the Animal Health Trust in conducting further serological and virological laboratory tests, which provided the all clear as of 18th February. No further cases have been reported and restrictions have been lifted.

**EHV-4 Respiratory Disease**
EHV-4 was isolated from nasopharyngeal swab in a 15 year-old mare which showed pyrexia, cough, mucopurulent nasal discharge, tachypnea, conjunctivitis and swollen lymph nodes.

EHV-4 was also isolated from nasopharyngeal swab in a 5 year-old gelding showing clinical signs of inappetance, serous nasal discharge, lethargy and swollen hind limbs.
In September 1994 an emergency response was initiated to investigate an outbreak of acute, fulminant respiratory disease in a group of racehorses in South-Eastern Queensland, Australia. After ruling out African Horse Sickness, virologists from the Australian Animal Health Laboratory identified a new virus, which was initially called equine morbillivirus, but subsequently named Hendra virus after the Brisbane suburb of Hendra where the outbreak occurred. Hendra virus is now classified in the Henipavirus genus in the Family Paramyxoviridae. This first outbreak affected 20 horses, 7 of which died within 12 hours of showing any clinical signs of illness. Unfortunately, the outbreak also affected two people, one of whom, the trainer in charge of the stable, subsequently died. Although it was not known at the time, these people were not the first to be infected by the Hendra virus. In August 1994 a veterinarian in the north Queensland town of Mackay conducted a post-mortem examination of a horse that had died suddenly. The veterinarian’s husband, who assisted with the necropsy, developed a fever and other “flu-like” symptoms shortly afterwards and although he recovered from this acute episode, he subsequently died from Hendra virus encephalitis some 14 months after the original infection.

Following the original outbreak in Hendra a large-scale investigation was conducted to identify the source of the virus and any potential reservoir or vector species involved in the epidemiology of the disease with a view to preventing any further outbreaks. Serological studies of a very large number of possible vertebrate host species in and around the racing stables found no evidence of Hendra virus infection in cats, dogs or horses in stables other than the outbreak stable, but antibodies to Hendra virus were found in local fruit bats. Subsequent studies have found that the natural reservoir of Hendra virus is fruit bats of the genus Pteropus in the Order Chiroptera. There are four species of these bats distributed around the northern and eastern coast of Australia, and all four species have serological evidence of Hendra virus infection. Hendra virus has been detected in foetal and neonatal lung and from uterine fluid and renal tissue of adult bats. Hendra virus has also been detected in bat urine and faeces. From these data it is proposed that horses become infected from being exposed to virus infected bat faeces, urine or birthing fluids when sheltering or feeding under trees in which bats are roosting. Currently, it is not understood precisely how Hendra virus jumps species from bats to horses. It is also not known how Hendra virus is transmitted from horses to humans, or indeed if infection of horses is necessary for human infection to occur, although all human cases to date have been linked to clinically infected horses. There are many investigators around the world currently studying bat paramyxoviruses, such as Hendra virus and the closely related Nipah virus, in order to improve our knowledge of how these viruses infect humans.

Between the first outbreak in 1994 and 2008 there were a number of individual cases of equine sudden death that were diagnosed, following post-mortem examination and further testing, as Hendra virus deaths. Fortunately, these were all single horse events with no associated human infection. In 2008, however, another multiple horse outbreak was reported, this time in a veterinary hospital in Brisbane. In this outbreak, all horses were in residence at the veterinary hospital at the commencement of the outbreak and affected horses presented with signs of central nervous system disease, rather than the previously reported signs of respiratory
disease. Five horses were affected, out of a population of more than 30 horses. Four of the five horses were so severely ill that they were euthanased for humane reasons. The surviving horse was also euthanased due to public health concerns. In this outbreak a veterinarian and a veterinary nurse became infected, both were admitted to hospital and the veterinarian subsequently died from viral encephalitis. It was likely that both affected people were exposed to the virus prior to the infected horses showing any obvious clinical signs of disease.

The 2008 outbreak in Brisbane highlighted the risks facing veterinarians and veterinary staff due to the lack of knowledge regarding mode(s) of transmission of Hendra virus from bats to horses and from horses to humans and the appropriate preventive methods they need to take. Experimental challenge studies to describe the pathogenesis of Hendra virus infection in horses and the routes of virus excretion were conducted, but unfortunately, before these results could be widely disseminated another veterinarian, in Rockhampton, was exposed to Hendra virus, and subsequently died from viral encephalitis, after performing an endoscopic examination on a clinically normal horse.

To date, of the seven people confirmed to have been infected with Hendra virus, four have died. The case-fatality rate in horses is approximately 80%, and the horses that have survived the acute stage of disease have been euthanased subsequently for public health reasons. Another two incidents of equine Hendra virus infection have been reported in 2009 and 2010. No human cases have been diagnosed in either event, but tests are still ongoing on the people exposed in the most recent episode.

Horse infection studies have been conducted recently at the Australian Animal Health Laboratory high biosecurity facility. Horses were exposed to Hendra virus via the respiratory tract and swab samples collected to determine the route of virus excretion. While no infectious virus was detected in tissue culture assays prior to onset of clinical signs, the nucleic acid of Hendra virus was detected by polymerase chain reaction (PCR) in nasal and oral swabs, as well as in blood and urine prior to the onset of fever. Samples collected from the respiratory tract of the experimentally infected horses were consistently PCR positive following the first febrile episode until they were euthanased on humane grounds. Samples were collected from multiple organ systems during post-mortem examination and infectious virus was isolated from a large number of tissues indicating that during infection the virus spreads widely throughout the horse’s body. Importantly, however, there were large amounts of virus detected in the endothelial cells lining the blood vessels of the mucosa of the upper respiratory tract, which may explain how veterinarians performing endoscopy or lavage procedures might become exposed to infectious virus prior to the onset of obvious clinical signs.

No vaccine is currently available to protect either horses or humans against disease caused by Hendra virus. There are several groups working towards a vaccine and hopefully clinical trials will be commenced in the near future, now that funding has been secured from the Queensland state Government and the Australian federal Government.

Hendra virus is an important emerging disease of horses and humans and highlights the disease dangers that arise when humans and their companion animals move into ecological niches previously only occupied by native animals and their attendant viruses. Unfortunately, we know little to nothing about the viruses carried by most wildlife species until there is a spill-over of one of their viruses into humans or our domestic animals and a “new” disease appears, as has occurred several times in recent years.
**Bacteriology Disease Report for the First Quarter 2010**

A summary of the diagnostic bacteriology testing undertaken by different contributing laboratories is presented in Table 2. For contagious equine metritis (CEM) 25 of 28 HLB approved laboratories in the UK contributed data.

**VLA CEMO Data for the period January to March 2010**

We are again pleased to include data relating to CEM testing from the Veterinary Laboratories Agency (VLA), in this quarterly report. The sample population for the VLA is different from that for the other contributing laboratories as the VLA tests are principally in relation to international trade and/or outbreak investigations.

As already mentioned in the last report (Vol.5, No.4), in this quarter one isolate was identified as CEM positive by the VLA in a 5 year-old Arabian stallion stabled in Devon. Also in this quarter CEM was diagnosed by the VLA in a 10 year-old Highland mare stabled in Durham, England.

**Strangles**

Strangles remains endemic in the UK, especially among parts of the non-Thoroughbred horse population. Diagnoses are confirmed in the UK based on traditional culture of *S. equi* and qPCR on respiratory samples and/or seroconversion using a serological ELISA.

**Table 2: Diagnostic bacteriology sample throughput and positive results for the first quarter 2010**

<table>
<thead>
<tr>
<th></th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CEMO (HBLB)</strong></td>
<td>10307</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td><strong>CEMO (VLA)</strong></td>
<td>457</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>Klebsiella pneumoniae</strong></td>
<td>10308&lt;sup&gt;1&lt;/sup&gt;</td>
<td>4</td>
<td>25</td>
</tr>
<tr>
<td><strong>Pseudomonas aeruginosa</strong></td>
<td>10297&lt;sup&gt;1&lt;/sup&gt;</td>
<td>28</td>
<td>25</td>
</tr>
<tr>
<td><em><em>Strangles</em> culture</em>*</td>
<td>1281</td>
<td>97</td>
<td>16</td>
</tr>
<tr>
<td><strong>Strangles PCR</strong></td>
<td>796</td>
<td>93</td>
<td>2</td>
</tr>
<tr>
<td><strong>Strangles ELISA</strong></td>
<td>1067</td>
<td>237</td>
<td>1</td>
</tr>
<tr>
<td><strong>Salmonellosis</strong></td>
<td>378</td>
<td>13</td>
<td>17</td>
</tr>
<tr>
<td><strong>MRSA</strong></td>
<td>148</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td><strong>Clostridium perfringens</strong></td>
<td>126</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td><strong>Clostridium difficile</strong></td>
<td>105&lt;sup&gt;1&lt;/sup&gt;</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td><strong>Borrelia (by ELISA)</strong></td>
<td>13</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td><strong>Rhodococcus equi</strong></td>
<td>504</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td><strong>Lawsonia intracellularis</strong></td>
<td>135</td>
<td>27</td>
<td>3</td>
</tr>
</tbody>
</table>

CEMO = contagious equine metritis organism (*Taylorella equigenitalis*); HBLB = HBLB accredited laboratories; # = capsule type 1,2,5; VLA = VLA reference laboratory; *Streptococcus equi* subsp. *equi*; MRSA = meticillin resistant *Staphylococcus aureus*. **Lawsonia intracellularis** identified using PCR applied to faeces; 1 reproductive tract samples only.
**Rhodococcus equi**

For the first time in this report we include the results for *Rhodococcus equi*. This bacteria is an important cause of disease and death in foals 3 weeks to 6 months worldwide. The most common clinical manifestation of this disease is pyogranulomatous pneumonia, and it has the potential to cause significant losses, especially on farms where it is enzootic. It is normally diagnosed by culture from a tracheobronchial aspirate, although a number of PCR techniques and serologic tests have been developed. All the contributing laboratories that reported data in this quarter diagnosed *R. equi* by culture.

**VLA Salmonella results**

From the 15 strains typed by the VLA the serotypes reported were *S. Typhimurium* (ten cases), *S. Oslo* (three cases), *S. Newport* (one case) and *S. Nagoya* (one case). Each of the 15 positive samples represents one incident.

The following definition of an incident applies: “An incident comprises the first isolation and all subsequent isolations of the same serovar or serovar and phage/definitive type combination of a particular *Salmonella* from an animal, group of animals or their environment on a single premises, within a defined time period (usually 30 days).”

For more information from Defra about *Salmonella* in the UK, please [click here](#).
FOCUS ARTICLE: GLANDERS

Fatima Cruz, DVM, MRCVS, MPhil, Animal Health Trust.

**Introduction**

Glanders is a bacterial infection caused by *Burkholderia mallei* (previously known as *Pseudomonas mallei*) which mainly affects *equidae* (horses, mules and donkeys) and has a zoonotic potential.

The most common source of infection is the ingestion of contaminated food or water via discharges from the respiratory tract or ulcerated skin lesions from carrier animals; however subclinical carriers often prove to be more important in the transmission of disease than clinical cases.

Glanders was widespread in Great Britain in the nineteenth century and was finally eradicated from this country in 1928. However, it still continues to be reported from various areas of the Middle East, Asia, Africa and South America and has been reported very recently in Brazil and Bahrain.

**Clinical signs and lesions**

According to the OIE, the incubation period of glanders varies depending on the route and intensity of exposure and intrinsic factors of the host; therefore it can range from a few days to many months.

According to the location of the primary lesions, there are three described forms of the disease: nasal, pulmonary and cutaneous (also known as farcy).

**Nasal form:**

- Clinical signs: At the beginning only pyrexia, loss of appetite and laboured breathing with coughing may be present. Affected animals show a highly infectious, yellowish-green mucopurulent discharge; a purulent ocular discharge may also be present. Nodules in the nasal mucosa may produce ulcers.
- Lesions: Ulcers of the nasal area, trachea, pharynx and larynx that may resolve in the form of star-shaped scars.

**Pulmonary form:**

- Clinical signs: Usually requires several months to develop; initial signs are pyrexia, dyspnea and a persistent dry cough. Diarrhea and polyuria may also occur; all leading to a progressive loss of condition.
- Lesions: Lung lesions commence as small light-coloured nodules surrounded by a haemorrhagic zone or as a consolidation of pulmonary tissue and a diffuse pneumonia; pulmonary nodules then progress to caseous or calcified state eventually discharging their contents and spreading the disease to the upper respiratory tract. Nodules can also be found in the liver, spleen and kidneys.

*Picture 1: Nodules in the spleen*
Cutaneous form:

- Clinical signs: This form develops insidiously over an extended period; it also often begins with coughing and progressive debilitation. Pyrexia and enlargement of the lymph nodes may also be present.
- Lesions: Nodules begin to appear in the subcutaneous tissue along the course of lymphatic vessels of the legs, costal areas and ventral abdomen and upon rupturing excrete an infectious purulent, yellow exudate. Infected lymphatic vessels may result in swollen, thickened, cord-like lesions (often referred to as “farcy pipes”). Nodular lesions can also be found in the liver and spleen. Orchitis may also be present in affected stallions.

Diagnosis

In most countries these days diagnosis of any suspected cases would be undertaken by a government veterinary service according to approved methods available at the time. However the following summarises some of the methods available or traditionally employed in the past.

Clinical signs alone do not allow a definitive diagnosis of glanders since the respiratory form can be confused with equine viral arteritis (EVA), strangles (Streptococcus equi equi) and fungal pneumonia whereas the cutaneous form of the disease (farcy) may be confused with epizootic lymphangitis (Histoplasma farciminosum), ulcerative lymphangitis (Corynebacterium pseudotuberculosis) or pseudotuberculosis (Yersinia pseudotuberculosis). Specific diagnosis can be done by either identification of the agent, the mallein test or by serological tests.

Identification of the agent:

The ultimate confirmation of any infection is agent identification. However this can be quite a challenge with glanders. The optimal sample for isolating the organism is pus recovered from lung, choanal and organ abscesses or nasal mucosa. Even multiple sampling can give a disappointing identification rate since these abscesses do not contain many bacteria. In addition, these samples are often contaminated with other bacterial species such as Pseudomonas spp. and Pasteurella spp. which makes isolation very difficult. Subcutaneous abscesses contain good numbers of the pathogen whereas ulcers are usually free of B. mallei. As glanders has a zoonotic potential, all samples must be handled with great care in a laboratory that meets the requirements for “containment group 3” pathogens, and equally serious concerns exist over the conditions and justification for carrying out necropsy of suspected cases.

Other methods of identifying the agent are the confirmation by conventional polymerase chain reaction (PCR) or quantitative (real-time) PCR, and the intraperitoneum inoculation of suspected material into a male guinea pig.
pig and observation for peritonitis and orchitis has been reported (B. mallei must be re-isolated from the lesions).

**Mallein test:**
This test is one of the tests recommended by the OIE for the diagnosis of carriers and it consists of the evaluation of the hypersensitivity of the horse after the inoculation of a small volume of a mallein purified protein derivative (PPD) intradermally into the lower eyelid (intradermo-palpebral test), into the eye at the canthus (ophthalmic test) or subcutaneously in the middle of the neck (subcutaneous test). The intradermo-palpebral test is preferred since it is more sensitive, reliable and specific. The test was used extensively in the past, but has been superceded by serological testing in much of the western world. There has been concern that the test can interfere with subsequent serological diagnosis since a seroconversion can occur after subcutaneous injection.

**Serological tests:**
The complement fixation (CF) test is an accurate serological test that has been used for glanders diagnosis for many years. Serum is positive within one week of infection and remains positive (sometimes) in chronic cases. It is presently the only test prescribed by the OIE for international trade of equids. The specificity of the CF test is very high, but it is known that occasional equine sera show false positive reactions (probably cross reactions).

Experimental Enzyme-linked immunosorbent assays (ELISAs) and membrane (blot) serology tests have also been developed; none of them has yet been extensively validated and are not fully recognized by the OIE, but there may be further developments along these lines in the future.

**Control**
As a result of the severity of the effects of glanders and its social and economic impact, it is listed as notifiable by UK law, and also by the World Organisation for Animal Health (OIE), by the European Commission in Brussels under Directive 92/25/EC. This means that, without exception if there is any suspicion of glanders, a suitable authority such as Divisional Veterinary Manager of Defra (Animal Health) must be notified immediately. Imported horses from countries outside the European Union are subjected to import controls and risk-based testing for glanders.

No vaccines are available against glanders. The disease can only be prevented by biosecurity and sanitary measures. Although B. mallei is sensitive to many antibiotics, treatment probably leads to chronic and occult cases. Treatment is somewhat academic since most countries have a slaughter policy for glanders.

For more detailed information on this disease, see the OIE Technical Disease Card for glanders at http://www.oie.int/eng/maladies/en_technical_diseasecards.htm

**Acknowledgements**
We would like to acknowledge Ian Mawhinney from the Veterinary Laboratories Agency (VLA) for providing us with valuable comments and Holger C. Scholz from the Bundeswehr Institute of Microbiology in Munich, Germany for the pictures included in this focus article.
A summary of diagnostic toxicosis and parasitology testing undertaken by contributing laboratories is presented in Tables 3 and 4 respectively. Results for toxicosis are based on histopathologically confirmed evidence of disease only (where applicable).

### Table 3: Diagnostic toxicosis sample throughput and positive results for the first quarter 2010

<table>
<thead>
<tr>
<th>N/P</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grass Sickness</td>
<td>15</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Hepatic toxicoses</td>
<td>5</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Atypical myopathy</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tetanus</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

### Table 4: Diagnostic parasitology sample throughput and positive results for the first quarter 2010

<table>
<thead>
<tr>
<th>N/P</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Endoparasites</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascarids</td>
<td>1881</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>Cyathostomes</td>
<td>1115</td>
<td>290</td>
<td>12</td>
</tr>
<tr>
<td>Dictyocaulus</td>
<td>826</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Strongyles</td>
<td>2785</td>
<td>566</td>
<td>20</td>
</tr>
<tr>
<td>Tapeworms (ELISA based testing)*</td>
<td>26</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>Tapeworms (faecal exam)</td>
<td>1940</td>
<td>20</td>
<td>11</td>
</tr>
<tr>
<td>Trichostrongylus</td>
<td>34</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Strongyloids</td>
<td>368</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td><em>Oxyuris equi</em></td>
<td>35</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Fasciola</td>
<td>128</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>Coccidia</td>
<td>66</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Cryptosporidia</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>VLA Theileria equi (CFT)</strong></td>
<td>257</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>VLA Theileria equi (IFAT)</strong></td>
<td>311</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td><strong>VLA Theileria equi (cELISA)</strong></td>
<td>114</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td><strong>VLA Babesia caballi (CFT)</strong></td>
<td>257</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><strong>VLA Babesia caballi (IFAT)</strong></td>
<td>313</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><strong>VLA Babesia caballi (cELISA)</strong></td>
<td>114</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>Ectoparasites</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mites</td>
<td>27</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Lice</td>
<td>411</td>
<td>26</td>
<td>13</td>
</tr>
<tr>
<td>Ringworm</td>
<td>534</td>
<td>57</td>
<td>17</td>
</tr>
<tr>
<td>Dermatophilus</td>
<td>367</td>
<td>23</td>
<td>9</td>
</tr>
<tr>
<td>Candida</td>
<td>73</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

*Complement Fixation Test; CFT suspect/positive samples are tested in IFAT test
**Indirect Fluorescent Antibody Test; ***competitive Enzyme-linked immunosorbent assay; positive cELISA results are not undergoing confirmatory testing
Grass sickness surveillance data (www.equinegrasssickness.co.uk):

A total of eight equine grass sickness cases have been reported for the first quarter (January to March 2010). The type of grass sickness was reported for all cases with two (25%) acute cases, four (50%) subacute cases and two (25%) chronic cases. One horse died naturally, six cases were euthanased and one chronic case has survived to date. Only one of the eight cases was diagnosed by post-mortem and ganglia examination, whereas seven cases were diagnosed based on clinical signs alone.

The location of one case was not disclosed, with three cases reported from England and four from Scotland.

Of the affected horses 25% were geldings, 50% were mares and 25% were stallions. A range of ages was reported (7 months to 22 years) with the mean age being 10.2 years and the median 9.8 years. The breed type of three horses was provided with one Irish Draught and two Thoroughbreds reported.

Whilst reports of grass sickness is rarely reported to occur in horses less than one year old, subacute grass sickness was confirmed in a seven month-old cob-cross colt. Confirmation of diagnosis was undertaken by post-mortem examination of ganglia following euthanasia.

It should be noted that the grass sickness surveillance scheme receives data from a wider population in comparison to the data presented in Table 3 and different diagnostic criteria were used. For more information about the grass sickness surveillance please refer to previous reports published in Vol.4 No.2 and Vol.2 No.4.
REPORT ON POST-MORTEM EXAMINATIONS FOR THE FIRST QUARTER 2010

East Anglia

A total of 97 cases were examined including 60 aborted fetuses.

Of the aborted fetuses examined this quarter, umbilical cord torsion was suspected as the precipitating cause in 10 of 60 cases. Placentitis was found to be the cause in 14 cases of abortion, whereas EHV-1 was confirmed by PCR, virus isolation and histopathology in 14 fetuses. Premature placental separation was diagnosed as the cause of 3 abortions, and in one case the abortion was due to the presence of twin fetuses. No definitive cause was determined for 18 cases of abortion, however infectious agents were excluded.

There were six cases of neonatal death reported in this quarter. Three of these cases were associated to dystocia whereas one case was diagnosed to be due to sepsis; no definitive cause was determined for the remaining two cases.

Following post-mortem examination and confirmation by PCR a horse which was euthanased due to neurological signs was diagnosed of EHV-1 paralytic disease.

Ten horses were examined following gastrointestinal disease, causes of death were as follows: Two cases of Salmonellosis (one of them was confirmed to be due to Salmonella typhimurium), one case presenting a colonic ulcer and intestinal haemorrhage, one case of atresia coli in a neonatal foal, one case of idiopathic gastric rupture in an adult horse and five cases which were euthanased and presented a heavy worm burden (Cyathostoma in three cases, Strongyles in one case; no information available for the fifth case).

There were two respiratory cases reported. The first case was diagnosed of a bronchointerstitial pneumonia; this horse also presented disseminated intravascular coagulation (DIC). In the second case a paranasal sinus squamous cell carcinoma was diagnosed.

Following post-mortem examination and histology of a horse with cardiac disease, the heart showed focal fibrosis, myxomatous stroma and cartilaginous metaplasia.

One case of neoplasia in a horse was reported in this quarter. Post-mortem examination and histology showed that lymphoma was the cause of death.

Musculoskeletal cases reported in this quarter include a severe acute fracture of the ischium that led to haemorrhage into the pelvic canal and abdomen resulting in hypovolaemic shock and death in a 3 year-old colt, a case of compression spinal injury affecting the sixth and seventh cervical vertebrae and a case of hypertrophic osteopathy.

Following post-mortem examination and histology, ragwort was confirmed to be the cause of death in three cases. One of these cases also presented a heavy worm burden.

Seven welfare/neglect cases have been reported in this quarter. The first one was a 27 year-old gelding with a moderate diffuse eosinophilic and lymphoplasmacytic enteritis consistent with enteric parasitism. In this first case histology in the liver showed previous exposure to ragwort. The second case was a 7 month-old colt which showed a subacute myopathy. Given the young age, body condition and severe endoparasitism of this
second case, nutritional deficiency (vitamin E and/or selenium) was considered to be an underlying cause of myositis. The third case was a pony mare which presented a severe ulcerative osteoarthritis in the left shoulder. Histology in this third case showed a moderate to marked chronic proliferative synovitis. The fourth case was a yearling which showed recumbency and inability to rise. Following post-mortem examination and histology in this case an acute myopathy was considered to be the most likely cause of the clinically reported recumbency. The fifth case was a 15 year-old pregnant mare which collapsed during advanced pregnancy and showed dehydration secondary to scours. Histological changes in the liver suggested grazing exposure to pyrrolizidine containing plants (e.g. Ragwort). The sixth case was a 6 year-old pony mare with a history of pregnancy, parasitic infection and severe abdominal colic symptoms. Following post-mortem examination, the cranial mesenteric artery showed a proliferative endarteritis which was highly suggestive of *Strongylus vulgaris* infestation, and was the most likely cause of colic reported clinically. Finally, the seventh case was reported to show laminitis.

Other cases reported include a midline uterine artery rupture, a mare which showed haemoperitoneum post foaling and a bisphenoid/occipital fracture of the skull.

**Home Counties**

*Fourteen cases were examined in this quarter.*

Two abortions were reported in this quarter. One of them was confirmed to be due to a bacterial placentitis in the mare whereas the second one was due to a mycotic placentitis in the mare.

A neurological case was reported to be due to a non-suppurative encephalitis.

Post-mortem examinations in five gastrointestinal cases revealed colic disease in three cases (one of which was due to a pedunculated lipoma), a small intestine entrapment/incarceration in one case and a gastric rupture in the fifth case.

Pneumonia was reported in a horse after post-mortem examination. The causal agent was found to be *Pasteurella pneumotropica*.

Neoplasia cases include a melanoma in one case and an adrenal tumour in another case.

Other cases reported were a horse with hepatopathy and two cases of hydatidosis due to *Echinococcus granulosus*.

**South West**

*Fourteen cases were examined in this quarter.*

Five abortions were reported to be consistent with EHV infection; however confirmation by PCR and virus isolation was made only in four of these cases.

Five gastrointestinal cases were examined; these included a case of cyathostomosis, a horse that failed to respond to treatment after surgery for non-resected volvulus (histology of the small intestine was non-diagnostic), a case of reoccurrence of epiploic foramen entrapment three weeks after surgery, a case of a
horse with an enterolith which also showed a necrotic bowel and a case of caecal displacement which also presented gastric ulceration.

Following sudden death in a horse, the cause of death could not be determined but the post-mortem examination showed emphysema in the lung.

A case of hyperlipaemia in a donkey was reported; however the post-mortem findings were inconclusive.

Two cases of chronic hepatopathy were reported and were most likely due to ragwort. Both cases showed portal hepatitis with fibrosis and bile duct proliferation, moderate colitis and also a choroid plexus cholesteatoma.

**Northern England**

*Three cases were examined in this quarter.*

Necropsy in two gastrointestinal cases revealed cyathostomosis in one horse, and a hyperaemic small intestine in a horse which had presented persistent ileus.

The remaining case was a neonatal death. The cause of death wasn’t reported in this case.

**West Midlands**

*One case was examined in this quarter.*

Post-mortem examination in a young horse revealed a severe fibrinous pericarditis with abscessation of the lungs. A sample from the pericardial fluid was cultured and there was a profuse growth of *Actinobacillus equuli*.

**Scotland**

*17 post-mortem examinations were reported in this quarter.*

Following a prolonged history of pyrexia of unknown origin, a six month old pony filly was presented to the equine hospital. The filly was dull and obtunded, with evidence of central blindness, and numerous skin and mucocutaneous lesions varying form alopecia to crusting and erosion. Liver enzymes and bile acids were raised. On necropsy examination several peripheral lymph nodes were enlarged, with innumerable gritty pinpoint yellow foci within the parenchyma. The liver was markedly enlarged, diffusely yellow / brown, with rounded borders and multiple irregular depressions which overlay regions of fibrosis. The hepatic parenchyma was firm and occasionally gritty. The meninges within both posterior cranial fossae contained multifocal irregular granular nodules. Histopathology revealed a granulomatous lymphadenitis and meningitis with mineralisation, ulcerative dermatitis with dermal fibrosis, and severe, centrilobular and periportal hepatic fibrosis. The aetiology of these changes remains undetermined. The hepatic changes were considered most likely to be primary in this case. Possibilities considered were idiosyncratic drug reactions and various hepatotoxins.

A nine year old Shetland pony mare was presented for necropsy examination, having recently aborted a foetus, and developed septic peritonitis. The abdominal cavity contained a moderate amount of red to brown cloudy fluid. The left uterine horn was dilated and contained a moderate amount of grey, cloudy malodorous fluid. A partial thickness tear was present within the wall of the right uterine horn, which resulted in the
formation of a thin walled pocket within the wall of the uterus. Adjacent jejunum was loosely adhered to the uterine serosa at this point. Although the uterine tear was only of partial thickness it was considered to have resulted in the local spread of infection from the uterine lumen into the abdominal cavity.

A sixteen year old, grey Highland pony mare was presented for necropsy examination. Multiple round black, spherical nodules were present on the surface of the mammary gland, ventral tail base, perineum, subcutis of the forehead, submucosa of the lower eyelid, and caudal to the atlanto-occipital joint. Further nodules were found within the salivary glands, oropharyngeal lymphoid tissue, the fascia of the cervical neck, the left kidney, pelvic cavity and sub-lumbar region, thoracic cavity and dura mater. The final diagnosis was malignant melanoma with multiple metastases.

Gastrointestinal cases reported included one case of Equine Dysautonomia (grass sickness), one case of unexplained gastric rupture, one case of esophageal rupture, one case of right dorsal colonic displacement which also presented gastric rupture, one case of colonic impaction, and four cases of small intestinal strangulation, two of which were due to mesenteric lipomas whereas one was due to an epiploic foramen entrapment.

Other necropsy cases reported included one elderly pony which was presented with multiple joints affected by degenerative joint disease and a case of septic tendonitis in a horse. Three cases were recorded as having an open diagnosis.

*Biopsies from 22 horses were submitted this quarter.*

Surgical biopsy samples were submitted from 22 horses. Nine muscle biopsies were examined, four of which were within normal limits. One was consistent with equine motor neuron disease, one with rhabdomyolysis, and the diagnosis remains open in three cases. Diagnoses from other samples included one case of grass sickness, one case of eosinophilic colitis, squamous cell carcinoma of the third eyelid, and two cases of equine sarcoïds. Diagnosis remained open in 8 biopsies.

**Northern Ireland**

*Five post-mortem examinations and one biopsy were reported this quarter.*

Three aborted fetuses were examined. Umbilical torsion was suspected as the cause of abortion in one case which had a long cord with multiple torsions and focally extensive haemorrhage within the cord. The mare’s serum sample was positive for EHV antibodies also in this case. No significant pathogens were isolated in the other two cases.

An adult male horse died after an acute case of colic. Torsion of the ascending and descending colon with volvulus, venous infarction and caecal displacement were found on post mortem examination. Histologically renal tubular necrosis associated with hypovolaemic shock was found.

An adult male horse was euthanized after presenting with acute severe colic. On post mortem examination ileal entrapment in the mesenteric fold was diagnosed.

One equine biopsy was submitted. Sections from a pedunculated mass from the penile sheath were diagnosed as fibroblastic type sarcoïd.
ACKNOWLEDGEMENTS

This report was compiled by the Animal Health Trust.
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Capital Diagnostics, Scottish Agricultural College
CAPL Ltd.
Carmichael Torrance Diagnostic Services
Compton Paddock Laboratories
Endell Veterinary Group
Hampton Veterinary Group
IDEXX
JSC Equine Laboratory
Liphook Equine Hospital
Minster Equine Clinic
NationWide Laboratories
Newmarket Equine Hospital
O’Gorman Slater & Main Veterinary Surgery
Oakham Veterinary Hospital
The Donkey Sanctuary
The Royal Veterinary College
Three Counties Equine Hospital
Torrance Diamond Diagnostic Services (TDDS)
University of Bristol, Department of Pathology
University of Edinburgh
University of Glasgow
Veterinary Laboratories Agency

All laboratories contributing to this report operate Quality Assurance schemes. These schemes differ between laboratories, however, all the contagious equine metritis testing reported was accredited by the Horserace Betting Levy Board with the exception of the VLA, which acts as the reference laboratory.

We would also like to acknowledge the contribution of the Horserace Betting Levy Board CEMO-scheme.

We would welcome feedback including contributions on focus articles and/or case reports to the following address:

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