Highlights in this issue:

- Update on the EIA outbreak in the United Kingdom
- Defra consultation on Responsibility and Cost Sharing
- Test launched for Foal Immunodeficiency Syndrome

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 fourth quarterly equine disease surveillance report for 2009 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

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 immuno diffusion 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. All remaining horses on the infected premises have been tested with negative results, and no further cases have been reported. The premises on which the two horses were kept are currently under restriction and epidemiological investigations are ongoing.

In the UK, EIA was last confirmed in England in 1976 and in Northern Ireland in 2006. EIA is transmitted by biting insect vectors and, according to the National Equine Welfare Council, the risk of spreading the disease is low given the recent cold weather and its consequences towards the vector population. Exports of horses to Third countries can continue subject to meeting the requirements of the export health certificate of the destination country. For more information on this outbreak, including an epidemiology report and a qualitative risk assessment prepared by Defra, click here.

As of 5th February 2010, the CEM outbreak declared on 22nd October 2009 by the Department for Environment, Food, and Rural Affairs (Defra) on a premises near Milton Keynes, Buckinghamshire, England was declared resolved. Following treatment of the only case, three sets of samples were taken and the final sets of samples were confirmed to be negative for Taylorella equigenitalis by culture and PCR on 22nd December 2009; restrictions were lifted shortly after (click here).

Equine influenza continues to be of importance within the United Kingdom. In this issue we report on several small outbreaks, mainly in unvaccinated horses.

The University of Liege’s Equine Atypical Myopathy Alert Group (AMAG) reported no further cases of atypical myopathy in the UK and in Northern Europe as of 19th December 2009. It seems that the persistent freeze and snow in December stopped the series of cases which started this autumn. According to the AMAG this has been the largest clinical series ever encountered, with 371 cases identified and a survival percentage of 22%. Thirty five cases were reported from United Kingdom. For more information, click here and here.

As in the last report, in this report we include the results from the equine piroplasmosis serology at the VLA.
INTERNATIONAL DISEASE OCCURRENCES

As of 28th December 2009 the CEM outbreak reported in Dubai, UAE in October 2009 was declared resolved. The outbreak involved a Thoroughbred stallion which tested positive for *Taylorella equigenitalis* whilst in pre-export quarantine. This was confirmed by VLA Weybridge by agent isolation and PCR. All in contact horses tested negative for CEM, the affected horse was treated with local and systemic antibiotics and then tested negative for CEM by culture on three occasions; the horse was subsequently exported. The source of the infection remains unknown but is thought to have occurred prior to import into the UAE. (Click here).

In the US, the total number of stallions and mares confirmed positive for *T. equigenitalis* in connection with the 2008/2009 occurrence of the disease remains at 22 stallions and 5 mares. No further carrier animals were detected in the 4th quarter 2009 (click here).

Following the investigation launched on 20th January 2010 after the UK reported having confirmed the disease in two horses of a consignment from Belgium, EIA has been confirmed in one horse in Belgium on 2nd February 2010. On 21st October 2009, 18 horses arrived from Romania at a dealer’s establishment at Drongen. On 22nd December 2009, nine of these horses were sent to the UK, where the disease was diagnosed. The investigation revealed that the other nine horses were sold in mid-November to the same person at Assebroek. This person sold then one horse to his brother at Meetkerke; no movement of horses is permitted to or from these facilities. All horses having been in contact with the horses from Romania are being traced, movement controls will be applied in the farms and the animals will be tested for the disease. The positive animal is due to be euthanased. For more information on this outbreak, click here.

From October 2009 5 outbreaks of EIA have been reported in Germany. Two of these outbreaks have been resolved as of January 2010 whereas the remaining outbreaks are ongoing. The total number of susceptible horses accounts for 165, with 8 positive cases being destroyed. The affected premises are quarantined and epidemiological investigations are ongoing (click here).

The *equine piroplasmosis* (EP) outbreak confirmed in September by the Department of Agriculture, Fisheries and Food in an equine facility in county Meath, Ireland was declared resolved on 9th December 2009. The outbreak involved 458 susceptible equides on 11 premises. Of these, 50 tested positive for *T. equi* on 6 premises. The origin of the outbreak was not confirmed, but it was thought to be associated with an animal that had returned to Ireland having spent a prolonged period in an endemic area abroad. All premises restrictions were lifted and a register of sero-positive horses was created. (Click here).

Regarding the EP outbreak in Texas, US, as of 20th January 2010 over 1600 horses have been tested and piroplasmosis has been found in 364 horses. The positive horses are currently located in 12 states, with 289 of them still on the index ranch in Texas. The remaining horses are on premises in Texas, Alabama, California, Florida, Indiana, Louisiana, Minnesota, North Carolina, New Jersey, Tennessee, Utah and Wisconsin. Georgia is now free of equine piroplasmosis positive horses.
Furthermore, in late December, three additional cases of *Theileria equi* infection were detected in conjunction with a racetrack screening program in New Mexico, none of which displayed any clinical evidence of EP. There was no epidemiological connection between this group of positive horses and the ongoing investigation of widespread *T. equi* infection on the index premises in Southern Texas. As of 25th January 2010, 13 horses have tested positive and 5 have been euthanased in New Mexico. For more information of the US situation, click here.

The eight *Venezuelan Equine Encephalitis* (VEE) outbreaks reported last quarter in three districts in Belize and the 3 outbreaks reported in Costa Rica are continuing. No information is available on the virus subtype involved or whether the horses involved had been vaccinated against VEE. As reported by the OIE on February 2010, the event in Costa Rica is unlikely to be contained and the infection is considered to be endemic. (Click here).

On 2nd November 2009 the OIE reported the first occurrence of *West Nile Virus* (WNV) in Costa Rica. WNV was diagnosed by immunocapture ELISA in the National Veterinary Services Laboratory (OIE’s Reference Laboratory); at the moment three outbreaks have been reported in a single district of the country, involving in total 90 susceptible horses, 4 cases and 3 deaths. Measures applied include quarantine, as well as movement control inside the country and control of arthropods. As of 22nd February 2010 the OIE has declared the event resolved. (Click here).

As reported by the internet platform Promed (click here), since September 2009 13 horses have died in Nova Scotia, Canada, as a result of *Eastern Equine Encephalitis* (EEE). Infected horses show sleepiness or depression, difficulty walking, seizures, muscle twitches and other neurological signs such as pressing their heads against solid objects. The outbreak has remained contained, with no horses affected in other areas.

**DEFRA BUSINESS**

On 25th January 2010, Defra published for pre-legislative scrutiny and public consultation a draft Animal Health Bill to help implement its plans for responsibility and cost sharing (RCS) to deliver improved animal health and welfare in England. Following this publication, in this issue we include a focus article on responsibility and cost sharing consultation by Defra.

Defra is seeking views from all interested parties on whether the proposed legislation successfully achieves the desired outcomes of the RCS agenda.

If you would like to read the proposals, further information on the draft Bill and how to respond to the consultation are available from the RCS pages of the Defra website http://www.defra.gov.uk/foodfarm/policy/animalhealth/sharing/ahbill/index.htm. The deadline for responses is 19th April 2010.
FOCUS ARTICLES

In this report we are pleased to include three focus articles. In our first focus article, the Responsibility and Cost Sharing (RCS) team at Defra provides the details of Defra consultation on responsibility and cost sharing.

Following the focus article published for the first quarter in 2009 in which data of Equine Viral Arteritis (EVA) surveillance activity from 2005 to 2008 was assessed; in this issue, Fatima Cruz in collaboration with Richard Newton from the Animal Health Trust review the data collated and reported by Defra, AHT and BEVA over the period from 2005 to 2009 for Taylorella equigenitalis (the contagious equine metritis organism – CEMO), and data collated and reported over the period from 2008 to 2009 for K. pneumoniae and P. aeruginosa.

Finally, in our third focus article Laura Fox-Clipsham from the Animal Health Trust has prepared an overview on the Foal Immunodeficiency Syndrome (Fell Pony Syndrome) and the recent success in the development of a DNA test for detecting carriers of this disease.

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 or via the BEVA and Defra websites:

http://www.beva.org.uk/


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

FOCUS ARTICLE: DEFRA CONSULTATION ON RESPONSIBILITY AND COST SHARING

Responsibility and Cost Sharing (RCS) team, Defra.

On 25th January 2010, Defra published for pre-legislative scrutiny and public consultation a draft Animal Health Bill to help implement its plans for responsibility and cost sharing (RCS) to deliver improved animal health and welfare in England.

The main provisions of the draft Bill will:

- establish the ‘Animal Health Organisation’ to take over from Defra responsibility for animal health policy and delivery in England;
- put on a statutory footing the role of the Chief Veterinary Officer (UK), who will be based in Defra;
- simplify existing provisions on payments for animals slaughtered, or things seized or destroyed, for disease control purposes in England and Wales. It will also introduce express provisions to allow reductions in payments where a person has contributed to the spread of disease or breached relevant regulations; and
- broaden existing powers in England and Wales to collect and test veterinary samples and vaccinate animals to help in disease management.

The proposed animal health organisation would be led by an independent chair and board. The board would have an understanding of animal health, including knowledge of industry across all species, financial, corporate governance, public sector management, public health and veterinary science, and wider issues including wildlife and consumers. Recruitment would follow the Nolan principles, with appropriate input from industry experts in the recruitment and selection of suitable candidates. The board would be accountable to Parliament and Ministers, animal keepers, the wider public and other stakeholders, and subject to external scrutiny and challenge.

Defra consider that building greater responsibility sharing through the establishment of the proposed Animal Health Organisation will only help to bring about the essential behaviour change sought in the livestock sector in relation to risk if suitable financial contributions and incentives are also introduced. Defra consulted last year on measures requiring keepers to pay for some of the annual costs of surveillance and preparedness for exotic disease outbreaks. The intention is that keepers of all disease susceptible species should share these costs. The necessary cost sharing measures will be introduced by the Government under a future Finance Bill.

Two good examples of where the equine sector has benefitted from Defra’s disease prevention and control measures, together with new arrangements to ensure improved partnership working are (1) the recent detection of equine infectious anaemia in horses in Wiltshire as a result of post import testing, and the subsequent management of the disease incident; (2) the development of the African Horse Sickness control strategy.
Defra has established a joint industry-Government advisory group to help work together to set-up the proposed new body. It will advise on the detailed design and ways of working, to ensure that a new independent body has the confidence of industry and the public. The establishment of an Advisory Group demonstrates Government’s commitment to working with industry, and it responds directly to calls from many organisations for a greater involvement in policy development. This group is chaired by Rosemary Radcliffe, and Prof. Tim Morris of the British Horse Industry Confederation and Prof. Bill Reilly from the British Veterinary Association sit on the group.
**Virology Disease Report for the Fourth Quarter of 2009**

The results of virological testing for October to December 2009 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>
<thead>
<tr>
<th>Table 1: Diagnostic virology sample throughput and positive results for the fourth quarter 2009</th>
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<tbody>
<tr>
<td><strong>Serological Tests</strong></td>
</tr>
<tr>
<td>EVA ELISA</td>
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<tr>
<td>EVA VN</td>
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<td>VLA EVA VN</td>
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<tr>
<td>EHV- 1/-4 CF test</td>
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<td>EHV-3 VN test</td>
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<tr>
<td>ERV-A/B CF test</td>
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<tr>
<td>Influenza HI test</td>
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<tr>
<td>EIA (Coggins)</td>
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<tr>
<td>EIA ELISA</td>
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<tr>
<td>VLA EIA (Coggins)</td>
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<td>VLA WNV (PRNT)</td>
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<tr>
<th><strong>Virus Detection</strong></th>
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<tbody>
<tr>
<td>EHV-1/-4 PCR</td>
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<tr>
<td>EHV-2/-5 PCR</td>
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<tr>
<td>Influenza NP ELISA**</td>
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<tr>
<td>Influenza Directigen</td>
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<tr>
<td>Influenza VI in eggs</td>
</tr>
<tr>
<td>EHV VI</td>
</tr>
<tr>
<td>EVA VI/PCR</td>
</tr>
<tr>
<td>VLA EVA VI/PCR</td>
</tr>
<tr>
<td>Rotavirus</td>
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</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 28 EVA VN positives detected by the VLA, 13 were export samples, eight were samples for diagnosis, one was a serum sample from a stallion for artificial insemination (AI) certification, three samples were from overseas, 2 were private and one sample was from an imported horse. The 12 semen samples received for EVA testing were all negative on virus isolation and RT-PCR.

The 1982 agar gel immuno diffusion tests for EIA (AGID; Coggins) were conducted for international trade purposes and they were all negative.

**Virological Diagnoses For The Fourth Quarter Of 2009**

**EHV-1 Abortion**
One case of EHV-1 abortion in a Thoroughbred mare was diagnosed by virus isolation in fetal tissues. The mare was current for vaccinations against EHV-1/4.

**EHV-1 Paralytic disease**
On 4th January 2010 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-2**
EHV-2 infection was diagnosed by virus isolation from nasopharyngeal swabs in two colts (16 and 4 months old respectively).

**EHV-3**
One horse showed a seroconversion to EHV-3 on the virus neutralization (VN) test. No further information could be obtained regarding this case.

**EHV-4 Respiratory infection**
EHV-4 was isolated from a nasopharyngeal swab in a 18 month-old gelding which showed mild pyrexia, cough, serous nasal discharge and swollen lymph nodes.

**Equine Influenza**
Seven outbreaks of equine influenza were reported in this quarter.

**Outbreak descriptions**
In Monmouthshire, Wales two ponies (28 and 19 years old respectively) and a horse (unknown age), which showed pyrexia, mucopurulent nasal discharge, frequent coughing at rest and anorexia, tested positive for equine influenza by nucleoprotein (NP) ELISA on a nasopharyngeal swab. The virus was isolated and sequenced from only one horse. Serology by haemagglutination inhibition (HI) was consistent with recent equine influenza viral activity. The outbreak occurred in a livery yard with 47 animals of which 30
unvaccinated horses showed clinical signs whereas 12 previously vaccinated horses were not clinically affected.

In Lanarkshire, Scotland a 10 year old unvaccinated horse was positive for equine influenza by NP ELISA on a nasopharyngeal swab. The horse had been in contact with a horse which came back from a show several days previously and subsequently developed respiratory signs.

In Nottinghamshire, England an unvaccinated mare which had been out hunting with other horses from different premises showed respiratory signs and pyrexia and tested positive for equine influenza by NP ELISA on a nasopharyngeal swab. No further cases were reported from the hunt.

In Dorset, England two ponies showing clinical signs tested positive for equine influenza by NP ELISA on a nasopharyngeal swab. Two weeks before this two other ponies had been shipped over from different locations in Ireland. Five ponies were involved in the outbreak, 3 of them un-vaccinated and all were affected. Virus was isolated and sequenced from one animal and paired serology by HI in 4 of the 5 ponies showed seroconversion.

In a livery yard in Perthshire, Scotland a mare which had been to a show jumping yard the previous week tested positive for equine influenza by NP ELISA on a nasopharyngeal swab. Another 5 year old horse which showed more severe signs was negative for equine influenza at the time of sampling.

After attending a hunt event in Yorkshire, England 12 un-vaccinated horses from 3 different yards showed respiratory signs. Four of them were swabbed and 3 of them tested positive for equine influenza by NP ELISA. Paired serology by HI was performed in 9 of the 12 horses and results showed seroconversion to H3N8 equine influenza virus consistent with infection in 6 horses and titres consistent with infection prior to the time of the first sample in the other 3 animals.

Two horses among a group of 30 in Bridgend, Wales tested positive for equine influenza by NP ELISA on a nasopharyngeal swab. Cases showed classical signs of influenza with 20 unvaccinated horses in the group being affected. None of the animals had apparently left the premises in the previous few months and it was believed that the virus could have been introduced indirectly by human contact.

Equine influenza virus characterisation

Genetic characterisation of the isolates obtained from the outbreaks in Monmouthshire, Lanarkshire, Nottinghamshire, Dorset and Bridgend showed that they belonged to the Florida sublineage clade 1 of the American lineage of H3N8 equine influenza virus. In contrast the isolates from the outbreaks in Yorkshire and probably Perthshire (for which only partial HA sequence was available) belonged to Florida sublineage clade 2 of the American lineage of H3N8 equine influenza virus. This suggests that clade 1 viruses are becoming more numerous and widespread within the United Kingdom compared to previous years since 2003 when the Florida sublineage first appeared in the UK. The virus responsible for the outbreak in Dorset may have been introduced with ponies imported from the Republic of Ireland.
Bacteriology Disease Report for the Fourth Quarter 2009

A summary of the diagnostic bacteriology testing undertaken by different contributing laboratories is presented in Table 2. For contagious equine metritis (CEM) 25 of 27 HBLB approved laboratories in the UK contributed data.

VLA CEMO Data for the period October to December 2009

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. 3), in this quarter one isolate was identified as CEMO positive by the VLA in a seven year old non-Thoroughbred mare in Buckinghamshire, England. The diagnosis was made on the basis of initial agent identification (Taylorella equigenitalis) in a sample submitted to a private Horserace Betting Levy Board (HBLB) quality approved laboratory with subsequent confirmation by culture and qPCR by the Veterinary Laboratories Agency.

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 third quarter 2009

<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>CEMO (HBLB)</td>
<td>1088</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>CEMO (VLA)</td>
<td>1735</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Klebsiella pneumoniae#</td>
<td>1089¹</td>
<td>7</td>
<td>18</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>1101¹</td>
<td>23</td>
<td>18</td>
</tr>
<tr>
<td>Strangles* culture</td>
<td>1407</td>
<td>90</td>
<td>16</td>
</tr>
<tr>
<td>Strangles PCR</td>
<td>663</td>
<td>55</td>
<td>1</td>
</tr>
<tr>
<td>Strangles ELISA</td>
<td>849</td>
<td>132</td>
<td>1</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>412</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>MRSA</td>
<td>87</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
<td>116</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>*Clostridium difficile (toxin by ELISA or immunochromatography)</td>
<td>100</td>
<td>12</td>
<td>7</td>
</tr>
<tr>
<td>Borrelia (by ELISA)</td>
<td>29</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Lawsonia intracellularis**</td>
<td>55</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 = methicillin resistant Staphylococcus aureus. **Lawsonia intracellularis identified using PCR applied to faeces; 1 reproductive tract samples only

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**Borrelia**

*Borrelia burgdorferi* is the causative agent for Lyme disease. Clinical disease in horses is associated with lameness, stiffness, joint swelling, lethargy, fever, weight loss, uveitis, and potentially with neurologic disease and foal mortality. Results provided in this table for *Borrelia* were tested by an ELISA test kit. When this test kit was evaluated in 164 equine serum samples from northeastern United States in relation to Western Blot Assay, results showed 100% sensitivity and 95% specificity*.

*R. Chandrashekar and D. Daniluk, ACVIM congress 2004, abstract 268.

**VLA Salmonella results**

From the 10 strains typed by the VLA the serotypes reported were S. Newport (one case), S. Enteritidis (one case), S. Stourbridge (one case), *Salmonella* serotype 4,5,12:i:- (2 cases), S. Bovismorbificans (one case), S. Dublin (one case), S. Typhimurium (one case), *Salmonella* serotype 44:-:- (one case) and S. Muenster (one case). Each of the ten 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: SURVEILLANCE OF CONTAGIOUS EQUINE METRITIS (CEM), KLEBSIELLA PNEUMONIAE AND PSEUDOMONAS AERUGINOSA IN THE UNITED KINGDOM: 2005-2009

Fatima Cruz, DVM, MRCVS, MPhil, Animal Health Trust; in collaboration with Richard Newton, BVSc, MSc, PhD, DLSHTM, DipECVPH, FRCVS, Animal Health Trust.

Introduction

Following the focus article published for the first quarter in 2009 in which Equine Viral Arteritis (EVA) was assessed, and in order to gain insights into the UK equine surveillance trends over time, here we review the data collated and reported by Defra, AHT and BEVA over the period from 2005 to 2009 (including this quarter) for Taylorella equigenitalis (the contagious equine metritis organism – CEMO), and data collated and reported over the period from 2008 to 2009 (including this quarter) for K. pneumoniae and P. aeruginosa.

Overview

CEM is an infectious disease of equidae caused by T. equigenitalis (CEMO). K. pneumoniae capsule types 1, 2 or 5 and P. aeruginosa can also cause venereal disease. Clinical signs vary between mares and stallions; in mares there are two states of infection, the active state in which the main clinical sign would be vulval discharge, and the carrier state in which there are no outward signs of infection but the mare remains capable of shedding. Stallions do not generally show clinical signs of infection but can become carriers and therefore will be able to transmit the infection. Occasionally the bacteria can colonize the stallion’s sex glands, causing pus and bacteria to contaminate the semen.

Transmission of the disease can occur directly or indirectly during mating or teasing by genital or naso-genital contact, but also by means of contaminated semen used for artificial insemination (AI) or indirect, iatrogenic transmission via the hands and equipment of staff or veterinary surgeons having handled the tail or genitalia of an infected horse.

CEM, caused by T. equigenitalis, occurs in the non-Thoroughbred population, and only to a limited extent in Thoroughbreds. Both K. pneumoniae and P. aeruginosa occur sporadically within Europe. Recent CEM outbreaks in France, Germany, United Arab Emirates, USA and lately in the UK in October 2009 highlight the potential importance of this disease.

The UK situation

CEM was first reported in 1977 on studs in England and was found in 1978 in horses imported from Europe into the state of Kentucky in the USA. The emergence of this new disease triggered the development of the first HBLB (Horserace Betting Levy Board) Code of Practice for control of this infection in conjunction with the other bacterial venereal pathogens such as K. pneumoniae and P. aeruginosa (click here). In the UK, isolation of the CEMO is notifiable by law under the Infectious Diseases of Horses Order 1987 (click here) and
any positive samples must be reported by the testing laboratory to a Divisional Veterinary Manager (DVM) or equivalent such as the Duty Veterinary Officer at the local Animal Health office of the Department for Environment, Food and Rural Affairs (Defra). The main preventive measures for this disease are based on establishing freedom from infection before and during breeding activities, and exercising strict hygiene measures during breeding activities. Whilst Thoroughbred breeders have adopted a zero-tolerance to CEMO globally, many non-Thoroughbred breeds remain endemically affected and there are no measures adopted to screen and clear breeding animals. Consequently, the effective absence of restrictions on trade in horses within the EU places the UK at risk of importation of CEMO infected horses from mainland Europe.

**CEM testing data**

CEM is diagnosed by means of microaerophilic culture from clitoral or endometrial swabs in mares and swabs taken from the urethra, urethral fossa and penile sheath, plus pre-ejaculatory fluid when possible in stallions. Currently CEM can now be diagnosed by quantitative/real time PCR (qPCR) in some laboratories; this technique increases sensitivity and speed of diagnosis. Every swab taken for official export health certification, and also every swab with a positive result from a HBLB certified laboratory must be sent to the designated laboratory within the Veterinary Laboratories Agency (VLA).

**Graphical presentation of CEMO data: 2005-2009.**

Figure 1 represents a summary by quarter for 2005-2009 of the total number of CEMO cultures conducted by a network of UK-based diagnostic labs and vet practices certified by the HBLB (together referred to as “CEMO HBLB”) and by the VLA (referred to as “CEMO VLA”). The lines represent the number of positives from both the HBLB labs and the VLA. The numbers on top of the CEMO HBLB bars correspond to the number of HBLB certified laboratories submitting data in each quarter.

![Graph of CEMO culture data by quarter for 2005-2009 for VLA and HBLB certified laboratories.](image-url)
The results of note in this summary for CEMO culture data from 2005 to 2009 are:

- The consistent seasonal trend in the number of cultures conducted within each year, mostly from 2006 on, which was different between VLA and HBLB certified laboratories. The pattern in HBLB certified laboratories demonstrated a peak in the 1st quarter, declining to 3rd quarter and with a small increase in numbers in the 4th quarter. This pattern would be consistent with pre-breeding testing in the first two quarters, with a predominance of Thoroughbred samples tested in the 1st quarter and some cultures conducted after December 1st in the 4th quarter. The VLA pattern would be different since VLA cultures are principally in relation to international trade (most of which is done between October and December) and outbreak investigations, showing lower numbers of cultures overall and a less pronounced peak in the final quarter, consistent with international trade testing requirements.

- For the period between the 2nd quarter 2005 (when the VLA started submitting data) and the 4th quarter of 2009, the proportion of positive results for both the VLA and HBLB certified laboratories were reasonably closely matched. This could be due to the fact that every positive result from a HBLB laboratory must be repeated and confirmed by the VLA. Overall there was a very low level of positivity for CEMO among the screened population in the UK, and most of the positive cases were horses from mainland Europe and staying in the UK for international transfer.

- Regarding the number of HBLB certified contributing laboratories, there was a peak in the 1st and 2nd quarters and a decrease in the 3rd quarter since many of the laboratories won’t submit data if they haven’t conducted any CEMO culture. Interestingly, even when in 2009 there was a rise in the number of contributing labs reaching nearly the 100% of contributions (due to the efforts of HBLB), the addition of laboratories to the reporting scheme has actually made little difference to the coverage of samples – probably because the scheme already covered the major contributors by means of numbers of cultures conducted; in addition, there was a small decrease on the numbers of cultures conducted by these major contributors in 2009 (which could reflect the impact of the prevailing adverse economic climate on the number of breeding animals being tested) and the new laboratories filled this gap. Such a good coverage since 2005 permitted the analysis of the graph as a whole despite the increase on the number of laboratories reporting in 2009.

Figures 2 and 3 represent a summary by quarter for 2008 and 2009 of the total number of *K. pneumoniae* and *P. aeruginosa* cultures conducted by a network of UK-based diagnostic labs and vet practices (dark blue bars). It should be noted that data provided here for *K. pneumoniae* only relates to the pathogenic capsule types (1, 2 and 5). The lines represent the percentage of positive cultures. The numbers on top of the bars correspond to the number of laboratories submitting data in each quarter.
The results of note in this summary for *K. pneumoniae* and *P. aeruginosa* culture data from 2008 to 2009 are:

- As in Figure 1, the consistent seasonal trend in the number of cultures conducted within each year. This similarity would be explained by the fact that the pre-breeding testing is the same as for CEMO.

- The percentage of positive results for both *K. pneumoniae* and *P. aeruginosa* were reasonably closely matched with the exception of the 4th quarter 2009. There was a peak in the percentage of positives in the 3rd quarter 2008 and 2009 for *K. pneumoniae* and the 3rd quarter 2008, 3rd and 4th quarter 2009 for *P. aeruginosa*, but the high percentages of positives could be due to the low numbers of samples being tested in these quarters.

- With regards to the number of contributing laboratories, there are differences with respect to CEMO since even when the major contributors report for CEMO, *K. pneumoniae* and *P. aeruginosa*, some HBLB certified laboratories only report for CEMO. Furthermore, there are other non-HBLB laboratories which report only cultures conducted for *Klebsiella* and *Pseudomonas*. As for CEMO, there was an increase in the number of contributing laboratories in 2009 which has not affected the coverage in terms of number of cultures conducted.

In conclusion, the data presented in this article confirms the on-going application of pre-breeding recommendations resumed in the HBLB Codes of Practice. These recommendations have led to a **zero level of positivity in the Thoroughbred population in the UK**, where zero-tolerance to CEMO has been adopted. Overall the positive cases were **sporadic** and were detected in **non-Thoroughbred horses**, especially in those from mainland Europe and transiting UK for international transfer.

The efforts of the HBLB as the operator of the laboratory approval scheme have led to reporting almost 100% coverage in 2009; therefore the data presented here is representative of the UK situation and should be encouraging for stakeholders and international trade partners for breeds undertaking pre-breeding CEM screening.
TOXIC AND PARASITIC DISEASE REPORT FOR THE FOURTH QUARTER 2009

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 fourth quarter 2009

<table>
<thead>
<tr>
<th>Condition</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>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hepatic toxicoses</td>
<td>30</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Atypical myopathy</td>
<td>88</td>
<td>18</td>
<td>5</td>
</tr>
<tr>
<td>Tetanus</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4: Diagnostic parasitology sample throughput and positive results for the fourth quarter 2009

<table>
<thead>
<tr>
<th>Parasites</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Laboratories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endoparasites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascarids</td>
<td>906</td>
<td>36</td>
<td>13</td>
</tr>
<tr>
<td>Cyathostomes</td>
<td>835</td>
<td>228</td>
<td>10</td>
</tr>
<tr>
<td>Dictyocaulus</td>
<td>595</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Strongyles</td>
<td>1666</td>
<td>266</td>
<td>19</td>
</tr>
<tr>
<td>Tapeworms (ELISA based testing)*</td>
<td>21</td>
<td>15</td>
<td>4</td>
</tr>
<tr>
<td>Tapeworms (faecal exam)</td>
<td>1356</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Trichostrongylus</td>
<td>34</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Strongyloides</td>
<td>713</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Oxyuris equi</td>
<td>50</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Fasciola</td>
<td>109</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Coccidia</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cryptosporidia</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>VLA Theileria equi (CFT)*</td>
<td>707</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>VLA Theileria equi (IFAT)**</td>
<td>792</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>VLA Theileria equi (cELISA)***</td>
<td>535</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>VLA Babesia caballi (CFT)*</td>
<td>706</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>VLA Babesia caballi (IFAT)**</td>
<td>793</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>VLA Babesia caballi (cELISA)***</td>
<td>535</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Ectoparasites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mites</td>
<td>20</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Lice</td>
<td>398</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Ringworm</td>
<td>397</td>
<td>135</td>
<td>14</td>
</tr>
<tr>
<td>Dermatophilus</td>
<td>189</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>Candida</td>
<td>63</td>
<td>6</td>
<td>2</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 15 EGS cases have been received for the fourth quarter (October-December 2009), making a total of 128 reports in 2009. The type of grass sickness was reported for 14 cases with 8 (57%) acute cases, 4 (29%) chronic cases and 2 (14%) subacute cases. All of the cases in this quarter were fatal. Only one of the 15 cases was diagnosed by post-mortem examination and ganglia examination whereas 14 cases were diagnosed based on clinical signs.

The locations of two cases were not disclosed, with 5 cases reported from England, 6 from Scotland and 2 from Wales.

Of the affected horses 40% were geldings, 40% were mares and 20% were stallions. A range of ages was reported (1 year – 18 years) with the mean age being 7.3 years and the median 4 years. The breed of one horse was unknown, with 1 cross breed and 13 pure breeds reported in this quarter.

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.
Foal immunodeficiency syndrome (FIS), formally known as Fell Pony syndrome, was first recognised in UK Fell Pony foals in 1998, described as a syndrome of anaemia and immunodeficiency. Since, FIS cases in the Fell Pony have been reported across Europe and United States of America. Until recently the disease had only been reported in the Fell Pony, but in June 2008, the same condition was confirmed in a Dale Pony foal.

The disease manifests at 2-6 weeks after birth, the foals being apparently normal when born. The first clinical signs of this disease include diarrhoea, nasal discharge, poor growth, pale gums and decreased appetite. A progressive profound anaemia is a notable early feature of this disease (PCV<20%). Lymphocyte sub-population analysis show normal circulating T-lymphocyte numbers and severely reduced B-lymphocytes numbers.

Attempts at symptomatic treatment have been made; these include rehydration, antibiotics, vitamin and mineral (Se) supplements, analgesia, blood transfusions, erythropoietin injections. Despite extensive treatment and supportive therapy, foals die or are euthanased on the basis of lethargy, severe anaemia and persistent infections before sixteen weeks of age. To date there have been no validated cases where foals have survived this disease, FIS has a 100% mortality rate.

Breeders noted a familial pattern, with repeat matings producing both affected and unaffected foals. This together with examination of the stud book, which reveals a small effective breeding population and genetic bottlenecks in recent years, strongly suggests that FIS is an inherited disease caused by an autosomal recessive mutation. Research into the genetics of FIS, funded by The Horse Trust, has been performed at the Animal Health Trust, in collaboration with the University of Liverpool. Recent advances in equine genetics enabled this project to advance rapidly and in November 2009, a single point mutation was identified on chromosome 26 that segregated with the disease. The mutation was screened in 23 obligate carriers, including the sire and dam of an FIS affected Dales foal, all were heterozygous for the mutation. Thirty-one samples from affected animals were screened, including the affected Dales foal and all were homozygous for the mutation. Ninety-six samples from breeds which are unlikely to have interbred with the Fell or Dales, including Thoroughbred, Appaloosa, Arab and Sport Horses, were screened for the mutation, all were homozygous for the wild-type allele.

FIS carrier prevalence in the Dales and Fell population has been estimated by analysing a random anonymous selection of samples submitted to the Animal Health Trust for parentage verification. Two-hundred and fourteen Fell Pony samples were screened, of which eighty-two were carriers of the mutation (38%). Eighty-seven Dales Pony samples were screened, of which sixteen carried the FIS mutation (18%).

This breakthrough has led to the development of a test which can be used to screen the DNA of individual
ponies for the presence or absence of the mutation. The test, which is available from the Animal Health Trust, will enable breeders to make informed breeding decisions.

By identifying carriers, subsequent carrier-carrier matings can be avoided, since it is pairings of this type only which lead to FIS. We would urge any breeders of Fell or Dales ponies to utilise the test and also encourage using the test for any mixed breed ponies with Fell Pony or Dales Pony ancestry.

Since the launch of the FIS test, forty-one Fell Pony samples have been submitted for testing, twenty-two were carriers (54%) of FIS. Twenty-eight Dales Pony samples have been submitted for testing, three were carriers (11%).

Previously there has been no definitive test for an FIS foal so diagnosis was made on clinical signs and clinical history. In 2010, the test will act as a diagnostic aid to confirm suspected syndrome foals. Foal samples submitted will be processed as urgent and the result available in three working days from receipt of sample.

Our breeding advice is that all animals should remain actively breeding to avoid the loss of desirable breed traits and prevent a loss of genetic diversity in the population. Carriers can still be bred to tested clear ponies. On average, 50% of the offspring will be clear and 50% will be carriers; there can be no affected animals produced from a carrier-clear mating. Foals which will be used for breeding themselves can be DNA tested to determine whether they are clear or a carrier. In time, with careful breeding, the disease gene may eventually be eradicated from the population.

For further information on FIS testing please contact the Fell Pony Society, the Dales Pony Society or refer to the Animal Health Trust website at http://www.aht.org.uk/genetics_tests.html#equine

References:


REPORT ON POST-MORTEM EXAMINATIONS FOR THE FOURTH QUARTER 2009

East Anglia

A total of 80 cases were examined including 54 aborted fetuses.

Of the aborted fetuses examined this quarter, umbilical cord torsion was suspected as the precipitating cause in 21 of 54 cases. Placentitis was found to be the cause in 8 cases of abortion, whereas EHV-1 was confirmed by PCR and virus isolation in one fetus and EHV-4 was confirmed by PCR and histology in another fetus. No definitive cause was determined for 23 cases of abortion, however infectious agents were excluded and the most likely causes were placental insufficiency or umbilical cord torsion.

A case of neonatal death was associated to dystocia.

There were two neurological cases examined this quarter, one of them was diagnosed of a pedunculated lipoma located in the caudal cervical spinal cord (C5-C6) and which might be an unusual presentation of dynamic Wobbler’s syndrome in this horse.

Eight horses were examined following gastrointestinal disease, causes of death were as follows: Two gastric ruptures (in one of them there was no histopathological evidence of grass sickness), Two caecal ruptures, one case of typhlocolitis, two cases of peritonitis (one of them septic), and one case of colonic ischemia following correction of colon torsion.

A 3.5 month old part-bred foal suffered severe clinical illness of a mainly respiratory nature for a period of only approximately 24 hours prior to death. Failure to identify an infective agent as the cause of death and the fact that this was the seventh foal death in the premises since July suggested the possibility of a pneumo-toxic plant-based agent/s.

Post mortem examination of a horse showed an aortic rupture as the cause of death, whereas the examination of another horse with congestive heart failure revealed atrial fibrillation, mitral valve regurgitation and left heart enlargement.

Following post-mortem examination and histology, lymphoma was found to be the cause of death in a horse.

Atypical myopathy was diagnosed in three cases. Other musculoskeletal cases reported were an oblique, complete fracture in the left tuber coxae and a pony filly which was found dead and post-mortem findings showed a significant and widespread subacute myopathy affecting the striated muscles and the heart muscle.

Hydatid cysts were found in the liver of a horse in the post-mortem examination and confirmed by later gross histology examination.

Three welfare/neglect cases have been reported in this quarter. The first one was a pony which had suffered from a severe cachexia and typhlocolitis associated with numerous intrallesional parasites. The animal had been in a severely catabolic state which would have resulted in decompensation of cardio-respiratory and
brain function. The second case was a female pony which was found lying flat on her left side and was thought to have been recumbent for more than 24 hours. Post-mortem examination revealed carcase emaciation with generalised atrophy of body fat deposits, purulent endometritis with placental remnants from an aborted pregnancy, an empty stomach with ulcers in both the oesophageal and pyloric regions, and parasitic lesions in the large bowel mucosa, cranial mesenteric and colic arteries. PCR testing of fresh tissues including CNS was negative for EHV-1 and EHV-4. The third and last case was a pony which died following a period of violent struggling after entrapment of its head and neck, and resulted in neck and shoulder trauma. Post-mortem examination showed the presence of local haemorrhage around the atlas and deeper regions of the neck, and haemarthrosis in one atlanto-occipital joint. A minimally displaced L-shaped fracture of the left wing of the atlas provided further confirmatory evidence of recent severe trauma at that site.

Other cases reported include a horse euthanased due to laminitis and a case of acute renal failure in which a pigment tubular nephropathy was diagnosed.

Home Counties

Ten cases were examined in this quarter.

One abortion was reported in this quarter, which was confirmed to be due to a fungal placentitis in the mare. In addition, a neonatal death was a result of enteritis, as diagnosed by post-mortem examination and histology.

Two neurological cases were reported. Encephalitozoon cuniculi encephalitis was confirmed in one of the cases by histology, whereas a pituitary adenoma was found in the second case.

Post-mortem examinations and histology in five gastrointestinal cases showed a pedunculated lipoma, colitis in two cases (one of them was confirmed to be due to bacterial infection), colic disease and a case of heavy intestinal parasitism.

Finally, the tenth reported case in this quarter was a neoplasia which was confirmed to be a pheochromocytoma.

South West

Nine cases were examined in this quarter.

One abortion was reported to be due to EHV infection. After gross post-mortem examination, EHV infection was diagnosed by PCR and histology in the fetus.

Gastrointestinal cases included a case of gastric stasis and ulceration in which dental disease was also present, and a case of a perforating ulcer in the large colon with subsequent peritonitis.

A case of pneumonia with associated pleurisy was diagnosed in a horse. In addition, another horse collapsed at a race; after gross post-mortem examination a massive, exercise-induced pulmonary haemorrhage (EIPH) was suspected to be the cause of death.
After gross post-mortem examination a comminuted fracture at the fourth cervical vertebra was diagnosed in a horse, which presented a wound at the forehead with entry into the frontal sinus.

Furthermore, a racehorse at exercise fractured its pelvis. Necropsy in this horse also revealed haemorrhage into muscles caused by the fracture.

Two cases of hepatic disease were reported in this quarter. One of them presented concurrent laminitis, whereas in the second case an abscess was found in the liver.

**Northern England**

*Four cases were examined in this quarter.*

Necropsy in two gastrointestinal cases revealed a ruptured stomach in one horse, and a ruptured large colon in another horse.

Mild hepatic fibrosis was diagnosed by histopathology in the third case.

In the fourth case, pyelonephritis was diagnosed following urine culture and inspection at necropsy, which also revealed a concurrent mild hepatic fibrosis.

**West Midlands**

*One case was examined in this quarter.*

One case of gastrointestinal disease was reported in which the post-mortem examination revealed a ruptured liver.

**Scotland**

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

A 17 year old grey pony mare presented to the equine hospital with tachypnoea, a marked bilateral serosanguinous pleural effusion, and signs consistent with Horner’s syndrome. The mare also had a left mammary mass, which had previously been diagnosed as mammary adenocarcinoma. Thoracoscopy revealed numerous nodular masses expanding the pleural surfaces, and approximately 30 litres of serosanguinous pleural fluid was drained. The histological appearance of samples of the pleural nodules obtained at this time was consistent with metastatic carcinoma with a marked desmoplastic reaction. Necropsy examination revealed a firm mottled and partially cystic mass within the left mammary gland, and enlargement of the inguinal and iliac lymph nodes. Numerous nodular masses were scattered throughout the pleura, with two larger lobulated masses in the cranial mediastinum partially surrounding the left vagosympathetic trunk. The liver contained numerous firm raised and often umbilicated masses, and similar nodular masses were present over the capsular surfaces of both kidneys. The final diagnosis was mammary gland adenocarcinoma with multiple organ metastases.
A four month old filly foal presented with a history of weight loss, lethargy and diarrhoea. Haematology and biochemistry revealed neutrophilia, hypoalbuminaemia and azotaemia, and electrolyte imbalances. Trans-abdominal ultrasound showed thickening of the small intestinal wall. The foal died shortly following admission. Necropsy examination revealed marked segmental thickening of the jejunum, with a corrugated mucosa, and stenosis of the intestinal lumen. Histopathology of the thickened intestine revealed a proliferative enteritis, and silver impregnation staining of sections demonstrated organisms typical of *Lawsonia intracellularis* within intestinal crypts. Faecal PCR was positive for *L. intracellularis*. Other gastrointestinal cases reported include four cases of colitis (one of them was a right dorsal colitis), one case of jejunal rupture, one case of gastric rupture, an ileal rupture which was suspected to be related to an epiploic foramen entrapment, and a rectal diverticulum with a 360° circumferential grade 3 rectal tear and resultant fibrinous peritonitis. The inciting cause of the rectal diverticulum / tear was not determined.

Eight musculoskeletal cases were reported. Five horses (two Cobs, one Thoroughbred cross, one Appaloosa and one mixed breed) from different premises were presented for necropsy examination following signs of acute myopathy. The most consistent gross findings were generalised pallor of the axial and appendicular muscles. Other findings included subcutaneous and pulmonary oedema, myoglobinuria and multiple petechiae or ecchymotic haemorrhages. Histological examination confirmed acute myopathy in all horses, which was considered to be consistent with atypical (pasture) myopathy. Atypical myopathy was also diagnosed in two other horses in this quarter, making a total of 7 cases. The eighth musculoskeletal case was a horse with fetlock joint disease.

A horse showed asphyxia and haemorrhage in the neck during a veterinary procedure.

A 35 year old grey gelding was euthanased due to old age. At necropsy multiple melanomas were present around the perianal region, and within several peripheral lymph nodes, the masseter muscles, lips and pharynx.

After post-mortem examination, two cases of hepatic disease were reported. In the first case death was related to liver torsion, whereas in the second case necropsy revealed hepatic lipidosis, colitis and metastatic calcification.

Other necropsy cases included one horse with chronic interstitial nephritis.

*Biopsies from 16 equids were submitted this quarter.*

Surgical biopsy samples were submitted from 16 horses. Four muscle biopsies were examined, two of which were within normal limits, one was consistent with exertional rhabdomyolysis, and in one case the diagnosis remains open. Two cases of equine grass sickness were diagnosed from ileal biopsies. Other diagnoses included metastatic pleural carcinoma (tissue from the case of mammary carcinoma described above), penile squamous cell carcinoma, penile papilloma, lymphocytic enteritis, pyogranulomatous dermatitis (suggestive of collagenolytic granuloma), and an intestinal vascular hamartoma.

The four remaining biopsies were either non-diagnostic or open diagnoses.
Northern Ireland

Nineteen cases were examined during this quarter.

Four aborted fetuses were examined. No significant pathogens were identified in these cases.

A Strongyloides worm count of 290,000 was detected on post-mortem examination of a four-month-old foal that was euthanased after developing nervous signs and becoming recumbent.

A high number of cyathostome and strongyle eggs were detected in the large intestine of a two-year-old mare that died after developing weakness and severe abdominal pain. On post-mortem examination gas and dark watery content were seen in the small intestines and the uterus contained serosanguinous fluid heavily populated by *Escherichia coli* and *Streptococcus* spp.

A nine-year-old pony was euthanased after developing colic. On post-mortem examination the small intestine was distended with gas and watery contents. The terminal ileum was darkened and devitalised in appearance. High levels of *Escherichia fergusonii* were isolated from gut contents.

Intestinal displacement was diagnosed in a four-year-old gelding euthanased after developing colic. On post-mortem examination the caecum and colon were distended with gas and watery contents. Haemorrhage and mesenteric venous distension were detected between the loops of the colon at level of the ileo-caecocolic junction.

Gastric impaction with peritonitis was detected on post-mortem examination of a six-month-old foal. A focus of severe peritonitis was observed around the stomach with a large volume of fluid and fibrin present in the abdomen. The stomach was markedly enlarged and filled with dry ingesta. Mucosal haemorrhage and ulceration was present at the pylorus.

A five-month-old foal with a history of hind limb ataxia was found dead in a field. On post-mortem examination a 30cm-long tear was present in the dorsal neck muscles. There were changes in the spinal cord consistent with a recent compressive injury. Dynamic vertebral instability, with accompanying neck muscle damage, was diagnosed suggesting a possible traumatic exacerbation leading to death.

A two-year-old gelding was euthanased after developing colic. A well-demarcated thickened white annular constricting ring was present in the duodenum with an anterior plug of fibrous gut contents. A diagnosis of parasitic enteritis was made.

A large pedunculated mesenteric lipoma strangulating the ileum was seen in a twenty-year-old mare. The mare had developed severe colic symptoms and became recumbent over three days.
Multiple deep haemorrhagic ulcers of the glandular portion of the stomach were detected on post-mortem examination of a two-year-old gelding which had been ill for several days. A large number of nematodes were observed close to areas of ulceration. *Trichostrongylus axei* was suspected on histology. A large number of cyathostomes was also observed in the small intestine and caecum.

Gastric distension and rupture with associated peritonitis was observed in a six-month-old foal. There was a 30cm-long tear in the greater curvature of the stomach adjacent to the splenic ligament with haemorrhage at the torn edges. The stomach was markedly distended and contained 16kg of slightly moist, packed ingesta, some of which had contaminated the abdominal cavity. The stomach contents consisted predominantly of fermented grass. The intestinal contents were sparse and dry ingesta coated the mucosa of the colon.

Yew poisoning was detected on post-mortem examination of an eight-year-old donkey with a history of sudden death. Multiple yew fronds were present in the stomach and large intestine.

A six-month-old foal died following sudden onset of scour, ataxia and anorexia. The small intestines, caecum and colon were distended with watery contents. The caecal and colonic mucosa were irregularly thickened with moderate eosinophil infiltrate. Prominent submucosal lymphoid tissue was present throughout the caecum and colon. Enteritis with a possible parasitic or hypersensitivity component was suspected.

Equine hyperlipaemia and hepatic lipidosis was diagnosed in a six-year-old Shetland pony that was found dead. There was histological evidence of moderate to large, clear intracytoplasmic vacuoles displacing hepatocyte nuclei.

Gastric impaction was observed on post-mortem examination of two two-year-old mares originating from the same farm. Both mares had a history of sudden death. The stomachs were markedly distended and filled with solid, dry short fibrous material.
ACKNOWLEDGEMENTS

This report was compiled by the Animal Health Trust.
We are extremely grateful to the following laboratories for contributing data for this report.

Animal Health Trust Diagnostic Laboratory
Avonvale Veterinary Practice
Agri-Food and Biosciences Institute of Northern Ireland
Arundel Equine Hospital
Axiom
Baskerville Horgan and Partners
Beaufort Cottage Laboratories
BioBest Laboratories Ltd
Capital Diagnostics, Scottish Agricultural College
Central Animal Pathology Laboratories (CAPL Ltd)
Carmichael Torrance Diagnostic Services
Chine House Veterinary Hospital
Compton Paddock Laboratories
Endell Veterinary Group
JSC Equine Laboratory
Hampton Veterinary Group
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:

Animal Health Trust
Lanwades Park, Kentford, Newmarket, Suffolk, CB8 7UU
Telephone: 01638 750659
Fax: 01638 555659
E-mail: equinesurveillance@ah.t.org.uk
Website: www.ah.t.org.uk