



**DEFRA / AHT / BEVA
EQUINE QUARTERLY DISEASE
SURVEILLANCE REPORT
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Highlights in this issue:

- **African Horse Sickness: update**
- ***Lawsonia intracellularis***

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 2008 produced by DEFRA, BEVA and the Animal Health Trust. 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.

Equine influenza (EI) remains an important subject for the global equine population. As regular readers will be aware, the outbreak that began in Australia in August 2007 has been heading towards control and eradication over the last few months. The last case was diagnosed on December 25th 2007. At the time of writing there is free movement of horses throughout Australia (free movement began on March 14th) however tracking of animal movements is still in place. If no further cases are identified, Australia will confirm that EI is no longer present on June 30th 2008. Monitoring and surveillance for EI will continue as part of Australia's exotic disease strategy. Background information about Australia's EI outbreak can be found at www.outbreak.gov.au. Japan also suffered an EI outbreak in the latter half of 2007. Control measures have been in place since the outbreak began however sporadic cases continue to be reported among racehorses and riding horses. Surveillance measures remain in place and every effort is being made to eliminate the virus from Japan. In the first quarter of 2008, EI was also prevalent in Sweden with 60 premises in northern Sweden affected in January. The majority of the affected animals had been incorrectly vaccinated.

Equine infectious anaemia (EIA) was diagnosed in a horse in Germany in late 2007 (administrative district Altotting, Federal State of Bavaria). A case was also identified in Germany in February 2008 (administrative district Ansbach, Federal State of Bavaria). Both animals have been euthanased. Investigations into onward spread were immediately conducted in response to both cases. So far all test results have been negative but testing is ongoing at the time of writing.

In South Africa a mild outbreak of African horse sickness (AHS), which began in late 2007, continues to be ongoing in 2008. Numerous premises have been affected, mostly involving non-Thoroughbred, non-vaccinated horses. AHS is endemic in north and eastern South Africa. South Africa hosted a symposium on AHS in February of this year which was attended by an international delegation of veterinary epidemiologists and regulators. A report from the meeting, kindly provided by Dr James Wood, can be found in this edition of the surveillance report. The threat of AHS to the UK equine population has been highlighted by the recent incursion of Bluetongue virus; AHS and Bluetongue are both transmitted by *Culicoides*. In response to the increased awareness of the threat to the UK horse population of exotic diseases, The Horse Trust and the Thoroughbred Breeders Association are holding a one day seminar on emerging equine exotic diseases in Newmarket on June 23rd 2008. The seminar will focus on the threat of AHS and West Nile Virus (WNV) to the UK horse population. Speakers will include international delegates from countries where AHS and WNV are already present alongside UK equine exotic disease experts and representatives from DEFRA. Topics discussed will include the threat to the UK equine population from AHS and WNV, contingency measures should an outbreak occur and the current situation and future possibilities with regards to vaccines. The seminar is open to



veterinarians and the horse-owning public. Further information can be found at www.thetba.co.uk and www.horsetrust.co.uk.

EU member states have reached an agreement on new European Regulations revising existing equine identification legislation. The main change is that microchipping of all foals will become compulsory on July 1st 2009. Member States are allowed to approve alternative identification means to the microchip. The aim of the microchip is to strengthen the identification link between a horse and its passport. This identification will be useful in monitoring and surveillance in the event of an infectious disease outbreak.

The Equine Grass Sickness Fund celebrated its 20th anniversary this year. A meeting attended by HRH, The Princess Royal was held in Edinburgh, in February, to celebrate this event. In addition an Equine Grass Sickness seminar was held at the Royal 'Dick' School of Veterinary Studies in April. The seminar highlighted current research being undertaken in this field including the Equine Grass Sickness Surveillance Scheme which was publicly launched in March 2008. The surveillance scheme is a collaborative project between the Animal Health Trust, the Equine Grass Sickness Fund, the University of Liverpool and the Royal 'Dick' School of Veterinary Studies. The aim of the surveillance scheme is to gather information about the incidence of Equine Grass Sickness in the UK and to try to identify 'high-risk' premises which may be able to be used in vaccine trials at a later date. Further information about this project can be found at www.equinegrasssickness.co.uk.

Our second focus article highlights disease caused by *Lawsonia intracellularis*. This condition has recently been gaining increased attention in the UK and is of considerable importance on affected farms.

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/> <http://www.defra.gov.uk/animalh/diseases/vetsurveillance/species/horses/index.htm>

We would remind readers and their colleagues that a form is available on the AHT website for registering to receive reports free of charge, via e-mail, on a quarterly basis. The link for this registration form is available via http://www.aht.org.uk/equine_disease_registration.html.



Virology Disease Report for the first quarter of 2008

The results of virological testing for January – March 2008 are summarised in Table 1 and include data relating to equine viral arteritis virus 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. Of the 29 EVA VN positives detected by the VLA, 14 were among export samples, 2 from imports and the remainder were private requests. The 6 semen samples received for virus isolation were all negative for EVA virus isolation after 3 passages in RK13 cell culture and negative for EVA by the one-tube RT-PCR

Table 1: Diagnostic virology sample throughput and positive results for the first quarter 2008

	Number of Samples Tested	Number Positive	Number of Contributing Laboratories
<u>Serological Tests</u>			
EVA ELISA	6620	235 [#]	3
EVA VN	3620	265 [#]	2
VLA EVA VN	1342	29 [#]	1
EHV-1/-4 CF test	782	5 [*]	1
EHV-3 VN test	8	0	1
ERV-1/-2 CF test	494	1 [*]	1
Influenza HI test	448	1 [*]	1
EIA (Coggins)	1969	0	1
<u>Virus Detection</u>			
EHV-1/-4 PCR	110	7	1
EHV-2/-5 PCR	0	0	0
Influenza NP ELISA**	347	0	1
Influenza VI in eggs	0	0	0
EHV VI	263	7	1
EVA VI/ PCR	2	0	1
VLA EVA VI/ PCR	6	0	1
Rotavirus	227	192	5

VN = virus neutralisation, ELISA = enzyme-linked immunosorbent assay, CF = complement fixation, HI = haemagglutination inhibition, PCR = polymerase chain reaction, NP = nucleoprotein, VI = virus isolation
EVA = equine viral arteritis, EHV = equine herpes virus, ERV = equine rhinovirus,
EIA = equine infectious anaemia, # = Seropositives include vaccinated stallions
* = Diagnosed positive on basis of seroconversion between paired sera

**Regular readers may note a large increase in the number of NP ELISA tests performed in this quarter. This increase is largely due to new 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.



Virological Diagnoses for the Third Quarter of 2007

EHV-1 Abortion

Eight EHV associated abortions were identified in this quarter. Three of the abortions occurred in Thoroughbreds; the breeds of the other mares are unknown. EHV-1 was identified using PCR on foetal tissue in 4 of these cases. Virus isolation was also positive in 3 of the cases. EHV-4 was identified using PCR from the placenta in one case.

EHV-1 Neurological Disease

No cases of EHV associated neurological disease were reported in this quarter.

EHV-1 Respiratory Disease

Two cases of respiratory disease associated with EHV were identified this quarter. EHV-4 was isolated from a nasopharyngeal swab from a pony with pyrexia and signs of respiratory disease. Several in contact horses also had signs of respiratory disease but did not test positive by virus isolation. EHV-4 was isolated from a horse on a second yard. This animal was pyrexia. No other animals on the yard were affected.

EHV-3 Coital Exanthema

No cases of EHV-3 coital exanthema were identified in this quarter.

Equine Influenza

No cases of influenza were identified in this quarter.

Planning for African horse sickness in Europe: a workshop in South Africa

**Dr. James Wood BVetMed, BSc, MSc, DLSHTM, DipECVPH, MRCVS
Director, Cambridge Infectious Disease Consortium**

The dramatic and still unexplained arrival of Bluetongue serotype 8 in northern Europe in 2006 and its dramatic spread in 2007, including to the UK, raised the spectre of the virus' close relation, African horse sickness (AHS), both arriving and then being able to spread in the same region. The Horse Trust has been active in trying to increase awareness of AHS and has coordinated a working party in the UK to help establish contingency plans for the event of AHS arriving in the UK. Three members of The Horse Trust's AHS working party attended a workshop at the annual South African Equine Veterinary Association's congress that was dedicated to the discussion of the challenges that this virus poses. The workshop took place in February of this year. Speakers included workshop organiser, Professor Alan Guthrie, international AHS expert (University of Pretoria, Onderstepoort), Professor Jim MacLachlan (Washington State University), Dr Alf Fuessel (European Commission), Dr James Gilkerson (University of Melbourne), Professor Uli Wernery (Central Veterinary Research Laboratory, Dubai) and Dr Jules Minke (Merial). Attendees also included Tony Kettle (Dubai), Mauricio Lopez (defra), Lynn Hillyer (British Horseracing Authority), Dr Richard Newton (Animal Health Trust) and Dr James Wood (Cambridge University and BEVA). There were many other South African delegates, several of whom are also acknowledged experts in the field of AHS and who made significant contributions to the workshop.



Sessions at the workshop covered details of AHS control, vaccination and laboratory diagnosis. The discussions were far ranging, but the workshop was carefully organised and produced many useful conclusions. Some of the findings from the workshop are outlined below.

The control session identified that movement restrictions and vaccination are critically important in AHS control. Surveillance and animal movement restrictions imposed as part of control measures require sensitive and specific, validated diagnostic tests, along with detailed information of horse location and movement in each area. Detailed information about the distribution and movement of the equine population is currently unavailable for nearly all areas of the world, including the UK. There was a consensus at the workshop that depopulation of domestic horses is contraindicated in the face of AHS, especially during outbreaks when animal movement restrictions are imposed.

The vaccination session heard how modified live vaccines are used in many countries where AHS is endemic. Such vaccines are, however, unlikely to be used in Europe due to the reluctance of the international community to use products containing live virus. Most vaccines used in endemic regions are polyvalent as they need to protect against all the different serotypes of AHS present in these areas. Inactivated vaccines have been used effectively and safely, e.g. in Spain in 1990-1991, but they are unlikely to be used in endemic regions because of their greater cost. These vaccines also need to be produced in Biosafety Level 3 production facilities, such as at Pirbright.

Vaccination requires safe and efficacious vaccines. In particular there is a need for cheap, safe products that allow discrimination between vaccinated and infected animals; no suitable products are currently available. The ability to discriminate between vaccinated and unvaccinated animals would be of critical importance if AHS was ever to be eliminated from currently free regions that become affected. There is considerable interest in what new vaccine technologies, such as pox virus vectored vaccines, have to offer for AHS. It was agreed at the workshop that international collaboration would be essential if international vaccine banks or vaccine seed banks were ever to be established; the availability of such banks would hugely add to the control of the disease.

In the laboratory diagnosis session, it was agreed that there was an urgent need for the establishment and validation of an internationally accepted group specific RT-PCR that reacts with all currently conceivable AHS viruses. There was also consensus that there was a need for an international accepted group specific ELISA for surveillance and trade purposes with published validation data. It is essential that when such tests become available the reagents needed to conduct the tests are available internationally. It is also essential that a test strategy to allow surveillance in the presence of a vaccinated population is determined.

This workshop provided an invaluable resource for the members of the Horst Trust AHS working party and it has served to focus the subsequent discussions held by the group.



Bacteriology Disease Report for the first quarter 2008

A summary of the diagnostic bacteriology testing undertaken by different contributing laboratories is presented in Table 2. For contagious equine metritis (CEM) 11 of 28 HBLB approved laboratories contributed data.

VLA CEMO Data for the period January – March 2008

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. Four isolates were identified as CEMO positive by HBLB laboratories. One sample was found to be negative on further testing by the VLA. Three samples were confirmed positive by the VLA. Two of these isolates were from animals not currently resident in the UK. The third sample was from a UK stallion. The results of post treatment swabbing will be available in the next edition of this report.

Table 2: Diagnostic bacteriology sample throughput and positive results for first quarter 2008

	Number of Samples Tested	Number Positive	Number of Contributing Laboratories
CEMO (HBLB)	11380	4	11
CEMO (VLA)	702	3	1
<i>Klebsiella pneumoniae</i> [#]	12068	23	10
<i>Pseudomonas aeruginosa</i>	12069	82	11
Strangles*	2644	183	10
Strangles PCR	1518	68	1
Salmonellosis	1055	43	11
MRSA	947	1	5
<i>Clostridium perfringens</i>	24	1	1
<i>Clostridium difficile</i> (toxin by ELISA)	58	5	3
Cryptosporidium	0	0	0
<i>Lawsonia intracellularis</i> **	4	0	1

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

Of the 43 samples testing positive for *Salmonella* spp., the serotype of 16 are known after further testing by the VLA. Of the 16 typed strains there were 11 of *S. typhimurium*, 1 of *S. anatum*, 1 of *S. durham*, 1 of *S. enetritidis*, 1 of *S. infantis* and 1 of *S. newport*.

Regular readers will note the addition of *Lawsonia intracellularis* (*L. intracellularis*) in this section. This bacterium is identified in the faeces using a PCR technique that is available at the Scottish Agricultural College. Further information about this disease can be found in the focus article later in this report.



LAWSONIA INTRACELLULARIS

Julie Ross MA, VetMB, MRCVS, Dip.ACVM

Lawsonia intracellularis (*L. intracellularis*) is an obligate intracellular gram negative bacterium that is most commonly associated with disease (proliferative enteropathy) in pigs. Porcine proliferative enteropathy is a proliferative disease affecting the ileum and, in some cases, the large intestine.¹ The bacteria infect gastrointestinal mucosal cells, specifically crypt cells, resulting in the loss of normal villi architecture in affected areas. Loss of villi leads to the loss of brush-border structure resulting in malabsorption. In horses, the pathogenesis of *L. intracellularis* is similar to the pathogenesis in pigs, although disease is confined solely to the small intestine.

L. intracellularis classically affects young horses, especially weanlings. Several breeds are reported to have been affected. Classical clinical signs in affected animals include weight loss, dull demeanour, dependant oedema, rough hair coat and diarrhoea. The severity and range of clinical signs varies extensively, ranging from profuse diarrhoea and colic to dullness and weight loss with normal faeces. A retrospective review¹ of 57 confirmed *L. intracellularis* cases revealed the most common clinical signs to be oedema, diarrhoea and fever. Clinical pathology classically shows profound hypoalbuminaemia and hypoproteinaemia (all horses in the previously mentioned study were hypoalbuminaemic; 74% had albumin levels <2mg/dl (reference range 3.4-4.1 mg/dl)). An association between affected horses and exposure to pigs is not a consistent finding.

The differential diagnoses for horses presenting with hypoalbuminaemia, with or without diarrhoea include, in addition to *L. intracellularis*, parasitism, right dorsal colitis, NSAID toxicity, *Rhodococcus equi*, sand enteropathy, peritonitis, alimentary lymphosarcoma and other infiltrative bowel diseases such as lymphocytic-plasmacytic enteritis. Colitis of any form can also lead to hypoproteinaemia. It should be remembered that severe pleuritis and protein losing nephropathy can also lead to hypoproteinaemia and if there are any concerns that the urinary or respiratory systems are affected, appropriate investigation should be performed. *L. intracellularis* should be considered as an important differential in any young horse presenting with diarrhoea, weight loss or chronic colic and hypoproteinaemia. Further diagnostics should be carried out to more definitively diagnose the cause of clinical signs.

Standard work-up for a case presenting with hypoproteinaemia (with or without diarrhoea/weight loss) might include abdominal ultrasonography, abdominocentesis, rectal exam, faecal exam and faecal culture and rectal biopsy. A glucose tolerance test may also be indicated if weight loss is the predominant sign. The most useful diagnostic test for the diagnosis of *L. intracellularis* is abdominal ultrasonography.

- Abdominal ultrasonography in classical cases of *L. intracellularis* reveals marked thickening of the small intestinal wall. It is impossible to visualise the entire length of the small intestine using ultrasound, thus the identification of normal small intestine on ultrasonographic exam does not exclude *L. intracellularis* as a cause of disease.



Ultrasonographic image of thickened loops of small intestine in a horse suffering from *L. intracellularis*. Image kindly provided by Dr. Joann Slack



Diffuse thickening and corrugation of the small intestine mucosa in *L. intracellularis*. Image kindly provided by Dr. Perry Habecker,

- Abdominocentesis would be expected to be within normal limits in *L. intracellularis*.
- Faecal culture should be performed to test for *Salmonella* and *Clostridial* species. Due to the intermittent shedding of *Salmonella* in infected animals, 3-5 cultures should be performed. Faecal floatation to identify parasites should also be conducted. Cyathostomiasis and tapeworm infestation may not be detected using this technique. Testing can also be performed for Rotavirus and Cryptosporidium.
- Rectal exam should be within normal limits in horses with *L. intracellularis*. Thickening of the small intestine may be palpable but the small intestine should not be distended.
- Rectal biopsy should only be performed if infiltrative enteropathy is suspected. *L. intracellularis* is limited to the small intestine thus rectal biopsy should be normal. Rectal biopsy in horses with diarrhoea should only be carried out if considered essential due to the friable nature of the rectal mucosa.

A presumptive diagnosis of *L. intracellularis* can be made based on signalement, clinical pathology and abdominal ultrasound findings. Definitive diagnosis requires demonstration of *L. intracellularis* organism in the wall of the small intestine, requiring *post mortem* examination or surgical biopsy. In most clinical cases diagnosis must be made using less invasive tests. A PCR is available that detects *L. intracellularis* organisms in faeces. This test has good specificity; however false negatives are possible due to intermittent organism shedding and decreased shedding with antimicrobial treatment.² Serological testing is can also be used to identify antibodies against *L. intracellularis* in recently exposed horses. In pigs, serology is considered more sensitive than PCR; this has not been investigated in equines. PCR testing is available at the Scottish Agricultural College. Serological testing is not currently available in the UK but can be performed at the Veterinary Diagnostic Laboratory, University of Minnesota.

L. intracellularis was first demonstrated to be a cause of equine disease in 1996³ (the first case was actually described in 1982⁴, but was not identified as *L. intracellularis* for several years). The disease has since been reported in Europe, Canada and Australia. The incidence of the disease appears to be increasing however this may be due to increased awareness of disease and increased availability of diagnostic tests. The true prevalence of *L. intracellularis* in horses is unknown.



The epidemiology of *L. intracellularis* has not been fully investigated and questions remain about the mechanism of transmission between horses. Infected animals shed organisms in their faeces resulting in contamination of soil. Studies in pigs have revealed that the *L. intracellularis* would not be expected to survive for longer than 2 weeks in the environment⁵. Cases on horse farms are often seen with longer periods between clinical cases or after there have been no horses present on a premise for several years. These findings suggest an environmental reservoir may exist, possibly in the wild deer population. Further work is needed in this area.

L. intracellularis infection can be fatal in equines if no treatment is instituted, however properly managed cases appear to have a high survival rate (in a referral hospital population, 93% of treated cases survived¹). Treatment recommendations focus on elimination of *L. intracellularis* from the gastrointestinal tract using antimicrobial therapy, colloidal support and general supportive care. Antimicrobials must achieve and maintain high intracellular concentrations in order to be effective against *L. intracellularis*. Commonly used therapies include erythromycin² (with or without rifampin) and oxytetracycline⁶. Licensing laws and potential side effects must be taken into account when deciding on which antimicrobial is most suitable. Readers should specifically remember that due to the high incidence of colitis in weaned horses treated with erythromycin, this drug should be used with great care. In the author's opinion, the use of this drug is contraindicated in animals over one year old due to the potential for fatal colitis. Oxytetracycline is a nephrotoxic drug thus appropriate fluid therapy should be initiated in hypovolaemic or dehydrated animals receiving treatment. Prolonged therapy is often essential in *L. intracellularis* cases (4-6 weeks) and the use of oral doxycycline in place of oxytetracycline has been described.⁶

Colloidal support is indicated in animals where albumin concentration is very low. Plasma transfusions or the use of synthetic colloids (i.e. pentastarch) help transiently alleviate the severe bowel oedema and peripheral oedema that can develop. Colloid administration can provide significant benefit early in the course of treatment. Further supportive care that may be indicated includes fluid therapy (if necessary due to dehydration caused by diarrhoea), nutrition and general supportive care. Protein and albumin levels should be monitored during treatment, however rapid increases should not be expected.

The long term outcome for young animals affected by *L. intracellularis* has not been studied, however current knowledge suggests that animals that survive do not have significant long term problems with growth, abdominal disease or performance. Further work is needed in this area, along with further investigation of the epidemiology of this disease and potential preventative measures.

¹ How to diagnose and treat *Lawsonia intracellularis* Frazer ML

Proceedings of the 53rd annual convention of the AAEP, Florida, 2007 pp236-239

² Equine proliferative enteropathy: a cause of weight loss, colic, diarrhea and hypoproteinaemia in foals on three breeding farms in Canada

Lavoie JP, Drolet R, Parsons D *et al* *Equine Vet J* 2000 ;32 :418-425

³ Proliferative enteropathy in a foal caused by *Lawsonia intracellularis*-like bacterium.

Williams NM, Harrison LR, Gebhart CJ. *J Vet Diagn Invest.* 1996 Apr;8(2):254-6.

⁴ Intestinal adenomatosis in a foal

Duhamel GE, Wheeldon EB *Vet Pathol* 1982;19:447-450

⁵ An outbreak of *Lawsonia intracellularis* infection in a standardbred herd in Ontario

Kimberly M, McGurrian J, Vengust M *et al* *Canadian Vet J* 2007;48:927-930

⁶ Tetracycline therapy of *Lawsonia intracellularis* enteropathy in foals.

Sampieri F, Hinchcliff KW, Toribio RE. *Equine Vet J.* 2006 Jan;38(1):89-92



Toxic and Parasitic Disease Report for the First Quarter 2008

A summary of diagnostic toxicosis and parasitology testing undertaken by contributing laboratories is presented in Tables 3 and 4 respectively. Results are based on histopathologically confirmed evidence of disease only (where applicable).

Table 3: Diagnostic toxicosis sample throughput and positive results for first quarter 2008

	Number of Samples Tested	Number Positive	Number of Contributing Laboratories
Grass Sickness	8	4	4
Hepatic toxicoses	9	3	3
Atypical myopathy	0	0	0
Ryegrass staggers*	1	1	1

* See brief article below for information on this condition.

GRASS STAGGERS

Grass staggers are caused by a number of related products of plant or fungal metabolism. These toxic products act at the γ -aminobutyric (GABA) receptors of the internuncial neurones. Toxicity results in signs of released inhibition of these neurones. There are 2 types of 'staggers' associated with ryegrass; perennial ryegrass staggers and annual rye grass staggers. Clinical signs are similar in both, including hypermetria, muscle tremors (worse when excited), trunkal sway, intention tremor and wide-based stance. Some animals will fall over and the limbs remain stiff during recumbency.

Diagnosis is usually based on clinical signs. There are no characteristic clinical pathological or histopathological changes associated with this condition. Examination of the pasture and hay should be conducted to try to identify the causative agent. Differential diagnoses would include severe electrolyte imbalances (i.e. hypocalcaemia) and cerebellar disease. Identification of the tremorogenic agent responsible for grass staggers is difficult and the help of a pasture management expert is recommended when an investigation is required.

No specific treatment is available for rye grass staggers. Case attack rate varies in different outbreaks and is likely to be related to the percent of the available forage that is affected. Case attack rate can reach 100%. Fatality rate ranges from 0-90%. Horses can recover from ryegrass staggers, although recovery can be slow. When a case is suspected, all horses should be removed from the affected pasture and a new hay source should be used while investigation occurs.



Table 4: Diagnostic parasitology sample throughput and positive results for the first quarter 2008

	Number of Samples Tested	Number Positive	Number of Contributing Laboratories
<u>Endoparasites</u>			
Ascarids	1232	32	9
Cyathostomes	1096	332	7
Dictyocaulus	450	3	5
Strongyles	1670	147	8
Tapeworms (ELISA based testing)*	1709	682	1
Tapeworms (Faecal exam)	1157	19	6
Trichostrongylus	549	14	5
Strongyloides	567	2	5
<u>Ectoparasites</u>			
Mites	277	6	8
Ringworm	326	25	7
Dermatophilus	23	0	1

* Regular readers will notice that the number of tapeworm samples submitted has increased significantly in this quarter. This is due to submissions from a new contributor. Our new contributing laboratory conducts the ELISA test for tapeworms and tested 1709 samples this quarter. The remainder of tapeworm testing was done using floatation techniques.

Report on *Post Mortem* Examinations for First Quarter 2008

East Anglia

87 cases were examined, including 66 fetuses.

Sixty six fetuses were examined following abortion; the cause of abortion was identified in 49 cases. The most common cause of abortion was umbilical cord torsion. This was identified as the cause of abortion in 18 cases. Other non-infectious causes of abortion included early placental separation (8 cases), placental mineralisation (5 cases), placental insufficiency (2 cases), twins (1 case) and placental hydrops (1 case). Infectious causes of abortion included equine herpes virus (8 cases) and placentitis (6 cases). The causative agent was identified as *Streptococcus equi* subsp. *zooepidemicus* in 3 placentitis cases.

Nine neonates were presented having died during dystocia. Six of these neonates had hypoxia associated changes identified on *post mortem*. Severe congenital malformations were present in one dystocia case (scoliosis and marked curvature of the right hind limb). Two foals were examined during this quarter. *Post mortem* findings in the first case included septic arthritis of the coxofemoral joint. The second case had evidence of impaction of the stomach (impacted with hay) and subsequent gastric rupture and peritonitis. This foal was 13 days old.

Four adult gastrointestinal cases were examined. Findings included grass sickness (2 cases) and severe cyathostomiasis (2 cases). One of the cases with cyathostomiasis also



had evidence of mesenteric artery thrombosis. Three neurological cases were examined. Sudden onset ataxia was the reason for euthanasia in 1 of the cases. *Post mortem* findings in this case included a synovial pseudocyst at C6-C7 causing cervical spinal cord compression. The remaining two cases had clinical signs associated with cerebral disease. The first case was ataxic and also suffered seizure like activity. *Post mortem* examination revealed Alzheimer type II changes in astrocytes consistent with hyperammonaemia. Blood ammonia concentration was increased, confirming this diagnosis. No hepatic changes were identified in this case. Hyperammonaemia associated with gastrointestinal disease, or of unknown cause, has been previously described. The third neurological case had been suffering from strangles-like symptoms when neurological signs developed (including blindness in the right eye, head tilt to the left and circling to the right). *Post mortem* examination revealed an abscess in the left caudal cerebrum with compression of the ventral cerebrum and right cerebral hemisphere.

Hepatic disease was identified in an aged donkey that was presented for *post mortem* examination. Findings were consistent with chronic hepatic disease but no cause was identified. Two cases of sudden death were examined. Recent neck and head trauma was identified as the cause of death in the first case. The cause of death was not identified in the second case.

Home Counties

18 cases were examined, including 2 fetuses.

Two fetuses were examined in this quarter. The first was aborted at 150 days gestation. The fetus was too autolysed to allow determination of cause of abortion. The second case was a term fetus that was dead on delivery. *Post mortem* revealed intra-abdominal haemorrhage possibly consistent with intra-uterine trauma.

Eight gastrointestinal cases were examined. Five cases had lesions associated with the small intestine. Findings included muscular hypertrophy of the ileum with secondary rupture (1), jejunal rupture (1) and small intestinal adhesions (1). The remaining 2 cases had clinical ileus post enterectomy. No further cause of ileus was identified at *post mortem*. Findings in the remaining gastrointestinal cases included gastric rupture (1), colon rupture (1) and chronic colitis (1). Three cases with a history of neurological disease were examined. Two of these cases had a history of ataxia. Findings in one of the ataxic cases included non-suppurative meningitis. The cause of ataxia in the second case was not identified. The third neurological case had a history of pyrexia and recumbency. *Post mortem* revealed acute meningoencephalitis. Bacterial culture was negative.

Two respiratory cases were examined. Findings included classical strangles lesions (1) and pulmonary abscessation (1). Two sudden death cases were examined. Findings in the first case included necrotising gastritis and evidence of toxemia. This animal had previously undergone tube feeding. No diagnosis was reached in the second case. One case was submitted following euthanasia on welfare grounds due to weakness and poor body condition. *Post mortem* revealed cyathostomiasis.

South West

Four donkeys and nine horses were examined.

Six gastrointestinal cases were examined. Findings included chronic ulcerative colitis (1), ulcerative enteritis and fibrinous peritonitis (1), epiploic foramen entrapment (1), partial colon volvulus (1), impaction of the large colon (1) and abdominal abscessation (1). Hepatic



lipidosis was identified in the case of ulcerative enteritis. Three musculoskeletal cases were examined. Findings included severe polyarthritis (1), myopathy of unknown cause (1) and myositis (1). Findings in the remaining cases included severe hyperlipaemia and hepatic failure (in a donkey) (1), pleural effusion with mineralisation of the lung (1) and retroperitoneal abscess (1). Cause of death was not identified in one case.

Scotland

Fourteen cases were examined including two fetuses

Two fetuses were examined during this quarter. Twisting of the umbilical cord was the cause of foetal death in one case. The cause of abortion was not identified in the other case. Four gastrointestinal cases were presented. Findings included acute grass sickness (1), large colon volvulus (1), jejunal entrapment (1) and nematode associated enteritis (1). Four musculoskeletal cases were examined. Findings included chronic laminitis (1), fracture of the 6th lumbar vertebrae (1), septic arthritis (1) and exsanguination following laceration of the right hind limb (1). The latter case was trauma associated. One further trauma associated case was examined; the cause of death was splenic rupture. Two cases with respiratory disease were presented. *Post mortem* examination revealed tracheal collapse with pulmonary haemorrhage (1) and inhalation pneumonia (1). One neurological case was examined. The cause of death was not identified in this case.

Northern Ireland

Fifteen cases were examined, including eight fetuses.

Eight fetuses were examined this quarter. *Leptospira* antigens were identified using immunofluorescence in 2 cases. In the first case, antigens were detected in the lung and kidney of the fetus. In the second case, *Leptospira* antigens were detected in the placenta. EHV antigens were also identified in the placenta in this case. Septicaemia associated with *Aeromonas hydrophilia* was identified as the cause of death in a third case. No significant findings were identified in the remaining cases.

A two day old foal with a history of diarrhoea was examined. Findings included marked haemorrhagic enteritis; *Clostridium difficile* and *Clostridium perfringens* toxins were identified in the ceecal contents.

Two sudden death cases were presented. Findings included cyathostomiasis (1) and severe malnutrition (1). One case presented after an episode of acute colic. *Post mortem* examination revealed gastric rupture with secondary fibrinous peritonitis.

The remaining cases included hepatic lipidosis in an inappetant donkey (1), partial urethral obstruction with secondary bladder distension (1) and suppurative bronchopneumonia with pleural abscessation. *Streptococcus zooepidemicus* was isolated from the lungs in this case.



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**We would welcome feedback including contributions on focus articles
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