Antimicrobial resistance (AMR) is a global problem with rising concern due to increasing resistance to commonly used antimicrobials (O’Neill et al., 2014). The issue is further compounded by a lack of new classes of antimicrobials being developed and authorised, especially for the horse. AMR is abundant across a wide range of equine pathogens, including, Escherichia coli (E. coli), Salmonella, staphylococci, Klebsiella, Pseudomonas species and other opportunistic pathogens. Identification of resistance in high profile pathogens, especially potentially zoonotic bacteria in horses such as E. coli which produce extended spectrum lactamases (ESBL) (Maddox et al., 2011b, 2011a, 2011c), methicillin-resistant Staphylococcus aureus (MRSA) (Weese et al., 2006; Weese & Lefebvre, 2007) and multidrug resistant (MDR, resistance to ≥3 antimicrobial classes) Salmonella (Ward et al., 2005) has increased the attention on antimicrobial resistance in horses. There is strong evidence in human and veterinary studies in other species that antimicrobial use is associated with emergence and dissemination of resistance in ESBL-producing Enterobacteriaceae and MRSA (Wieler et al., 2011; Dunowska et al., 2011). In horses there is also increasing evidence of a similar association being present (Maddox et al., 2011b, 2011c; Weese & Lefebvre, 2007).

Antimicrobial agents act by disrupting specific metabolic and normal functions of bacterial cells. There are 4 predominant targets for antimicrobial action (Neu, 1992):
- disruption of cell wall synthesis
- inhibition of DNA/RNA synthesis
- inhibition of protein biosynthesis
- interference with a vital metabolic pathway

The mechanisms through which bacteria can achieve resistance to antimicrobials can be grouped into 3 major categories (Tenover, 2006):
- protection or alteration of the antimicrobial target site
- exclusion of the antimicrobial agent from the cell interior (via reduced cell permeability or efflux pump expulsion)
- production of antimicrobial inactivating enzymes
Bacterial resistance mechanisms can either occur due to a certain trait common to all bacteria of that group (i.e. intrinsic) or arise from acquired mechanisms found only in some members of a genus or species due to alteration of the bacterial genome (i.e. extrinsic). Acquired resistance can arise from endogenous mutations in chromosomal genes but it is more often achieved by exogenous horizontal acquisition of novel genetic elements. The transferable genetic material participating in exogenous resistance can involve plasmid encoded resistance genes, gene cassettes linked to integrons, transposons and other mobile genetic elements (Roupas & Pitton, 1974; Hall & Collis, 1995). These genetic elements can encode pumps for drug efflux, enzymes for antimicrobial inactivation, alternatives of the antimicrobial target site and as well as mechanisms which provide protection for the molecular target (Tran & Jacoby, 2002; Heikkilä et al., 1990). Exogenous exchange of genetic material may occur between differing strains of the same species or even across genera, and can occur via bacterial transformation (incorporation of exogenous DNA from dead bacteria), conjugation (transfer of plasmids), or transduction (DNA transferred by viral bacteriophages that infect bacteria) (Roupas & Pitton, 1974). Acquired antimicrobial resistance mechanisms are of particular concern, irrespective of their specific origin, as they allow both the emergence and rapid dissemination of resistance in formerly susceptible populations of bacteria. This report will focus on antimicrobial resistance in *E. coli*

*Escherichia coli*

*E. coli* is considered part of the normal gastrointestinal tract flora in horses (van Duijkeren et al., 2000) but despite a predominantly commensal nature, many strains of *E. coli* are capable of causing disease of both gastrointestinal and extra-intestinal sites (Lanz et al., 2003). Antimicrobial resistance is commonly encountered and β-lactam resistance is of particular concern. *E. coli* is intrinsically resistant to penicillin (as it is unable to penetrate their outer membrane) but there is widespread acquired resistance to other β-lactams (mostly via the production of inactivating lactamase enzymes such as TEM-1, TEM-2 and SHV-1, or AmpC β-lactamases, all encoded by various bla resistance genes (Hawkey, 2008; Datta & Kontomichalou, 1965). The extended spectrum β-lactam antimicrobials (including cefotaxime, ceftiofur and cefquinome) were developed to counter resistance seen to the early β-lactams. Resistance to these agents is conferred by bacterial production of ESBL enzymes (Pitout, 2010), many of which are simple mutations of the original TEM/SHV lactamases and only a small number of amino acid substitutions are required to extend their spectrum of resistance to include novel agents (Bradford, 2001). An emergence of a family of ESBL-enzymes that are distinct from SHV and TEM-types has occurred in the last two decades and now predominate within *E. coli* (Tzouvelekis et al., 2000). These enzymes preferentially hydrolyse the extended spectrum β-lactam cefotaxime and are consequently named cefotaximases (CTX-M) and have since been found in both humans and animals (Wieler et al., 2011).
Prevalence and epidemiology of antimicrobial resistant E. coli in horses

E. coli that are resistant to most antimicrobials currently authorised for use in horses in the UK have been identified in previous studies in both clinical (Vo et al. 2007) and commensal isolates (Vo et al., 2007; Dunowska et al., 2006). An increased prevalence of faecal carriage of antimicrobial resistant E. coli has been identified in hospitalised horses compared with those in the community and the same is true for MDR and ESBL-producing E. coli (Maddox et al., 2011c; Ahmed et al., 2010; Johns et al., 2012; Apostolakos et al., 2017).

The prevalence of faecal carriage of MDR and ESBL-producing E. coli has also been shown to increase significantly during hospitalisation (Maddox et al., 2011c; Williams et al., 2013) and some studies have reported a consistent association between antimicrobial exposure in hospitalised horses and increased risk of resistance in faecal E. coli (Ahmed et al., 2010; Maddox et al., 2011c). One study reported an association between overall hospital use of antimicrobials and increased prevalence of resistance, even in horses not actually receiving antimicrobials (Maddox et al., 2011c). Other studies have identified that hospitalisation (even without antimicrobial treatment) is a further risk factor (Bryan et al., 2010; Williams et al., 2013). Being stabled on the same yard as a recently hospitalised horse has also been associated with ESBL-producing E. coli carriage in the equine community (Maddox et al., 2011b) and the identification of continued faecal carriage of MDR bacteria by horses discharged from hospital suggest these horses may act as a reservoir (Johns et al., 2012).

Commensal carriage of ESBL-producing and MDR E. coli has been studied in detail (Maddox et al., 2011c) but their role in clinical infections has not been quantified. Recent publications have reported an increase in clinical infections in horses caused by MDR E. coli (Johns & Adams, 2015) and E. coli accounted for the majority of all surgical site infections (SSIs) following exploratory laparotomy in a recent hospital study (Isgren et al., 2017), but the source of these infections is not yet clear. Monitoring and surveillance of emerging resistance, both in commensals and pathogens, is essential in order to allow us to estimate the growing burden of antimicrobial resistance in the horse population. Apart from the data collated in the AHT/BEVA/DEFRA quarterly surveillance reports and the limited reporting in the UK-VARSS report (VMD, 2017) there is little research or large scale coordinated surveillance of clinical bacterial infections in horses within the UK.
A new surveillance initiative

We are currently undertaking a surveillance project of the AMR profiles of bacterial infections in horses using diagnostic laboratories submissions across a wide geographical range of equine practices. We will report on bacteria commonly associated with clinical infections and their patterns of AMR across most of the UK.

We have also undertaken a multi-centre study investigating risk factors for carriage of ESBL-producing bacteria across five equine referral hospitals in the UK. In this study we obtained daily faecal samples from equine inpatients as well as weekly environmental samples at these hospitals to determine which bacteria reside in the patient faecal flora and hospital environment. We are sampling SSIs from patients at these hospitals in an attempt to determine any link between faecal carriage of MDR E. coli and any role in SSI. Variations in prevalence of carriage and resistance phenotypes among hospitals may be a reflection of different patterns of antimicrobial usage and management. Further analysis will determine risk factors and the geographical distribution associated with carriage of MDR and ESBL-producing E. coli in this population.

Comparison of AMR in commensal E. coli versus those found in clinical infections and in the equine hospital environment will be key to our understanding of reservoirs of infection and transmission. Interventions can then be developed to help mitigate transmission, which will aid prevention of resistant infections in our equine population.

If any hospital has a suspected outbreak of MDR E. coli from surgical site infections we would be happy to receive and process samples free of charge and feedback results. For more information please contact Cajsa Isgren on cisgren@liverpool.ac.uk or Dr Gina Pinchbeck on ginap@liverpool.ac.uk / 0151 7946195

References

References are available on request, please contact maire.o'brien@aht.org.uk

Important note

The views expressed in this focus article are the author’s own and should not be interpreted as official statements of APHA, BEVA or the AHT.
A summary of diagnostic toxicosis and parasitology testing undertaken by contributing laboratories is presented in Tables 14 and 15 respectively. Results for toxicosis are based on histopathologically confirmed evidence of disease only (where applicable).

### Table 14: Diagnostic toxicosis sample throughput results for the first quarter 2018

<table>
<thead>
<tr>
<th>Disease</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Labs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grass Sickness</td>
<td>17</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>Hepatic toxicoses</td>
<td>42</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Atypical myopathy/Seasonal Pasture Associated Myopathy</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

### Table 15: Diagnostic parasitology sample throughput and positive results for the first quarter 2018

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Number of Samples Tested</th>
<th>Number Positive</th>
<th>Number of Contributing Labs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Endoparasites</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascarids</td>
<td>4774</td>
<td>60</td>
<td>18</td>
</tr>
<tr>
<td>Stronglyloides</td>
<td>5825</td>
<td>588</td>
<td>16</td>
</tr>
<tr>
<td>Strongyles (large/small)</td>
<td>6545</td>
<td>2799</td>
<td>22</td>
</tr>
<tr>
<td>Tapeworms ELISA serum</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tapeworms ELISA saliva</td>
<td>4793</td>
<td>1420</td>
<td>12</td>
</tr>
<tr>
<td>Tapeworms Faecal exam</td>
<td>3563</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>Dictyocaulus arnfieldi</td>
<td>70</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Oxyuris equi</td>
<td>746</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Fasciola hepatica</td>
<td>500</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>Coccidia</td>
<td>1138</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Cryptosporidia</td>
<td>178</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td><em>Theileria equi</em> cELISA</td>
<td>341</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td><em>Babesia caballi</em> cELISA</td>
<td>341</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td><em>Theileria equi</em> (APHA) CFT</td>
<td>115</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><em>Theileria equi</em> (APHA) IFAT</td>
<td>164</td>
<td>22</td>
<td>1</td>
</tr>
<tr>
<td><em>Theileria equi</em> (APHA) cELISA</td>
<td>142</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><em>Babesia caballi</em> (APHA) CFT</td>
<td>115</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><em>Babesia caballi</em> (APHA) IFAT</td>
<td>164</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td><em>Babesia caballi</em> (APHA) cELISA</td>
<td>142</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Dourine(APHA) IFAT</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Dourine (APHA) CFT</td>
<td>282</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><strong>Ectoparasites</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mites</td>
<td>247</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Lice</td>
<td>249</td>
<td>19</td>
<td>8</td>
</tr>
<tr>
<td>Ringworm</td>
<td>337</td>
<td>69</td>
<td>14</td>
</tr>
<tr>
<td>Dermatophilus</td>
<td>39</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Candida</td>
<td>77</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

CFT = Complement Fixation Test - CFT suspect/positive samples are tested by IFAT test, IFAT = Indirect Fluorescent Antibody Test, cELISA = competitive Enzyme-linked immunosorbent assay
Grass sickness surveillance data for the second quarter 2018

The nationwide Equine Grass Sickness surveillance scheme (http://www.equinegrasssickness.co.uk/) was established in spring 2008 to facilitate the investigation of changes in geographical distribution and incidence of the disease in Great Britain. Data gathered by this scheme is collated in a strictly confidential database.

A total of six cases of equine grass sickness (EGS) were reported during the second quarter of 2018, of which one case occurred in April, one case in May and four cases in June. These figures are lower than expected for the time of year, where previously the highest number of cases seen per month each year has been in May, with an average of 30 reported each May since 2008.

Three cases were reported in England, one in Scotland and two in Wales. Of the six cases, three premises reported a history of EGS, one premises in England, one in Scotland and one in Wales.

The cases comprised of four mares and two geldings, with a median age of four years (range 1-9 years). Affected breeds were Cob (n=2), Native (n=2) and two cross breeds.

Type of EGS was given for five of the six cases; three were diagnosed with acute EGS and two with sub-acute EGS. Diagnostic information was provided for all reported cases, of which five cases were diagnosed on clinical signs alone and one case was diagnosed by histopathological confirmation of biopsies taken post-mortem (ileum, paravertebral trunk and mesenteric ganglia).

The Peak Season for Equine Grass Sickness - Early Summer: A Case Report

Introduction
Equine grass sickness (EGS), also known as equine dysautonomia, is a frequently fatal neurodegenerative disease of horses, donkeys and mules (Newton, et al., 2004; Girling, et al., 2017). As suggested in the name, the disease is seen predominantly in horses with access to grazing, accounting for >99% of cases (Newton et al., 2010; Wylie et al., 2011).

Clinical presentation of the disease includes an increased heart rate, muscle fasciculations, patchy sweating, dysphagia, reduced intestinal motility, signs of colic and weight loss (Doxey et al., 1991; Milne., 1996).

Horses presenting with the acute and subacute form of the disease will invariably die or require euthanisia (Wylie et al., 2009). Treatment for select cases of horses with chronic EGS may be successful through intensive nursing and monitoring. Recent analysis of the disease has found that survival rates are now at 16% for chronic cases (Wylie et al., 2011).

Horses of all ages can be affected by EGS, but young horses between the ages of two and seven years-old have a higher risk of contracting the disease (Wylie et al., 2009).

The high risk EGS season lies in early summer, with the largest number of cases being reported in May.

Case Presentation and History
A two-year-old Welsh mountain pony filly presented with clinical signs consistent with EGS on 5 June 2018 in Suffolk, UK. The filly had muscle fasciculations of the hindquarters, a short gait, rhinitis sicca, tachycardia (consistently 80bpm), rectal temperature of 38.3°C, ptosis of both upper eyelids and absence of borborygmi. Rectal examination revealed a very large impaction within the pelvic flexure and faeces in the rectum were clay-like.

The filly had been on the premises for a year and a half in total and had been grazing the affected paddock for four weeks before falling ill. Prior to that, the field had been rested over winter with adequate grass coverage and the field had been harrowed in the spring. Droppings were removed via mechanical sweeping six days prior to the filly falling ill. There had been a spell of hot dry weather followed by light rain in the preceding days. The farm had a history of EGS.
Diagnosis
Microscopic findings of the ileum included; paucity of neurons of the submucosa and myenteric plexuses and concomitant mild increased number of glial cells. Paravertebral trunk and mesenteric ganglia findings included; neurons characterized by multifocal chromatolysis, hypereosinophilia and cytoplasmic vacuolization, with concomitant increased number of glial cells.

A summary of ileal neuronal loss and neuronal degeneration of the paravertebral trunk with necrosis and gliosis was reached, consistent with EGS.

Outcome
Euthanasia and post-mortem. The five in-contacts were removed from the field and were closely monitored.

Discussion
Previous epidemiological studies have highlighted risk factors for EGS development (Newton et al., 2004; Wylie et al., 2009; Wylie et al., 2011) and a number of these were present in the history and presentation of this case. Horses aged between two and seven years old are at a greater risk of developing EGS, the affected animal fell into this young age category. The premises has a history of EGS which increases the risk of further cases occurring. Differing management practices can also increase the risk of EGS occurrence such as continuous turnout and movement onto new pastures, the affected animal had been moved to a new pasture within the preceding four weeks and was turned out all the time. Managing pastures with mechanical dropping removal aids such as sweepers has been found to increase the risk of EGS, thought to be a result of paddock and soil disturbance whilst sweeping, the farm the affected animal was housed at regularly swept the paddock. Weather conditions can also increase the risk of EGS occurring alongside the time of year. The affected animal fell ill in June, which is within the high risk period of early summer and the prevailing weather conditions had been predominantly dry, followed by light rain. Previous studies have found that an increase in cases of EGS occurred after two weeks of predominantly dry weather (Wood et al., 1998). Future preventative measures have been put in place at the premises, including resting fields for a period after mechanical sweeping.

References
References are available on request, please contact maire.o'brien@aht.org.uk
The caseload of post-mortem examinations reported below have been obtained from one UK Veterinary School and six of the other contributing laboratories to this report.

**East Anglia**
_A total of 41 cases were examined by post mortem._

A total of 17 aborted fetuses and fetal membranes were examined.

**Table 16: Summary of post-mortem findings for aborted fetuses in East Anglia for the second quarter 2018.**

<table>
<thead>
<tr>
<th>Post Mortem Diagnosis</th>
<th>Total</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placentitis</td>
<td>4</td>
<td>Placentitis confirmed to be secondary to bacterial infection, with aerobic cultures reported in one case, isolating E. coli</td>
</tr>
<tr>
<td>Equine Herpes Virus-1</td>
<td>6</td>
<td>Confirmed by histopathology and PCR on fetal and placental tissues</td>
</tr>
<tr>
<td>Placentopathy</td>
<td>1</td>
<td>Allantois had multifocal villous atrophy and multifocal squamous metaplasia. Villi were variably blunted, multifocally lost, with interspersed trophoblastic necrosis and squamous metaplasia</td>
</tr>
<tr>
<td>Placental Damage</td>
<td>1</td>
<td>Presence of placental damage, most likely secondary to terminal hypoxia</td>
</tr>
<tr>
<td>Placental Insufficiency</td>
<td>2</td>
<td>In the first case, histological findings supported the gross assessment of placental pathology and probable placental insufficiency. Given the extent of placenta grossly affected by the histologically confirmed changes, placental insufficiency was considered the most likely cause of foetal death and abortion. In the second case, inappropriate mineralisation was present over extensive regions, resulting in placental insufficiency</td>
</tr>
<tr>
<td>Umbilical Cord Torsion</td>
<td>1</td>
<td>None</td>
</tr>
<tr>
<td>Excessive Umbilical Cord Length</td>
<td>1</td>
<td>Intrapartum death, suspected to have resulted in cord compromise at foaling</td>
</tr>
<tr>
<td>No final diagnosis*</td>
<td>1</td>
<td>Infectious causes ruled out</td>
</tr>
</tbody>
</table>

*Where cases had no final diagnosis reached, hypotheses were made for each case with the intention for interpretation by the submitting veterinarian, relating post mortem findings to concurrent clinical history to affirm the most likely conclusion. For every post mortem, congenital and common infectious causes have been ruled out.

Three cases of neonatal death were examined. One case was found to have dysmaturity and sepsis associated with in-utero funisitis (inflammation of umbilical cord). One case examined was a sudden death with uncertain aetiology. One case was a sudden death with severe acute pulmonary oedema, suspected to be as a result of cardiogenic issues or possibly from anaphylaxis.

Nine gastrointestinal cases were examined. One case had acute gastric rupture and associated peritonitis. One case was found to have rupture of the ileum and associated peritonitis. Four cases were diagnosed with severe necrotising enteritis and associated peritonitis and one of these cases concurrently had nephritis, pneumonia and pulmonary arterial thromboemboli. One case of small intestinal volvulus was examined. Two cases with large intestinal pathology were examined and a diagnosis of caecal rupture and associated acute septic peritonitis was made in both.
One hepatic disease case was examined and found to have a hepatopathy consistent with ragwort toxicity.

Two musculoskeletal cases were examined. In one case, a diagnosis of severe damage of the distal phalanx made, with osteomyelitis and a comminuted fracture of the distal phalanx, locally extensive, severe, chronic tenosynovitis and suspect bone loss of the navicular bone. The macroscopic examination confirmed the clinical diagnosis of severe damage of the distal phalanx and the adjacent articular and periarticular structures. The other case was found to have a pelvic fracture and secondary haemabdomen.

Two neurological cases were examined. In one, a diagnosis of Wobbler syndrome was made. Narrowing of the vertebral canal was detected on mid-sagittal section of the spine, at the level of C3-C4 and C6-C7. The cervical spinal cord in these areas was compressed mainly secondary to upward bending of the floor of the spinal canal. Exacerbation of the compression was produced by ventral flexion of the spine. In the other case, a severe encephalomyelitis was found. Histological findings included; spinal: haemorrhagic with perivascular mononuclear cuffing, predominantly grey matter, and brain: multifocal necrosis and supplicative foci and mononuclear perivascular cuffing. The case was negative for EHV, WNV, Borna disease and Listeria.

Four respiratory cases were examined. One case was diagnosed with Rhodococcal pneumonia. One case had a severe acute diffuse interstitial and bronchointerstitial pneumonia with no aetiological agent identified. One case had a severe septic pleuropneumonia. The final case was an anaesthetic-related death.

One case was examined for an unexpected death, the case presented with dysphagia. No diagnosis was reached and there was no specific aetiology found.

Two cases of sudden death were examined. In the first, there were histopathologically detected myocardial changes but these were minimal and unlikely to be the only cause of the sudden death. For this reason potential concomitant damage and alteration of the conduction system may have possibly played a role. The post-mortem examination in this case could not determine the precise cause of sudden death but a primary acute cardiac insufficiency is the most likely precipitating cause. In the second case, mild inflammatory changes of the myocardium could have caused alteration of the conduction system causing a deadly arrhythmia, but diagnosis was not definitive.

Home Counties
A total of 16 cases were examined.

One case of abortion was examined and diagnosed with placentitis. The placenta had necrosuppurative placentitis with intra-lesional gram-positive cocci. A positive culture isolated Streptococcus zooepidemicus and Staphylococcus aureus.

Three cardiac cases were examined. The first was found to have petechiaton and severe acute multifocal extensive, monophasic acute degeneration and necrosis of the right atrial and ventricular epicardium. The second case had a marked serosanguinous effusion of the pleural and peritoneal cavities secondary to cardiac failure, with the heart displaying multifocal interstitial fibrosis. The final case was diagnosed with a thrombus affecting the deep circumflex iliac artery with secondary acute, marked haemabdomen.

Six cases of gastrointestinal disease were examined. There was one case that had a markedly impacted and distended stomach with no obvious precipitating cause. One case was found to have macroscopic segmental oedema and mural thickening of the small intestine, caecum and colon and ulcerative colitis. Salmonella was isolated from the small intestine and serotyping identified the isolate as Salmonella typhimurium I B. One case was found to have very large numbers of adult Parascairis equorum worms within the intestine. One case was examined for colic but no cause of the colic was identified, a mild abdominal effusion was present. Two cases were diagnosed with mesenteric lipomas causing segmental infarction and necrosis of the small intestine and mid-jejunum.

One hepatic case was examined and found to have a diffusely firm and nodular liver with severe, chronic bridging fibrosis, biliary hyperplasia with regenerative nodules, hepatocellular megalocytosis, vacuolar degeneration and necrosis.

Two neoplastic cases were examined. One was diagnosed with disseminated haemangiosarcoma affecting the vertebral body, heart, kidney, liver, spleen and left ovary. The other case was diagnosed with multifocal lymphoma.
affecting the intestine, jejunum and colon.

One reproductive case was examined and diagnosed with a non-healing scrotal wound with purulent material, fibrinous deposition and marked swelling.

Two welfare cases were examined. Both were found to be emaciated with diffuse muscle atrophy and evidence of parasitism.

**Northern England**

* A total of one case was examined.

One musculoskeletal case was examined and diagnosed with a right ileal wing fracture.

**Northern Ireland**

* A total of two cases were examined.

One case of neonatal death was examined and a diagnosis of sepsis made. The liver and adrenal glands contained numerous bacteria and bacterial culture isolated *Pasteurella multocida*.

One case of lymphoreticular disease was examined and a diagnosis of mesenteric suppurative lymphadenopathy made. Bacterial culture isolated *Pasteurella multocida*.

**Scotland**

* A total of 17 cases were examined.

One neonatal death case was examined and diagnosed with patent ductus arteriosus and pulmonary atelectasis.

Seven cases of gastrointestinal disease were examined. One was diagnosed with an ileo-caecal intussusception. One case had a diagnosis of marked lymphoplasmacytic and eosinophilic enteritis with erosive typhilitis of moderate severity, mild granulomatous colitis with intralesional nematodes (cyathostomes), cestode infestation (*Anoplocephala perfoliata*) and multiple chronic renal infarcts. One case had a perforation at the pelvic flexure of the large colon with associated peritonitis and pelvic cellulitis. One case was a six week old male Warmblood with an intestinal motility disorder and a right dorsal impaction identified, histopathology was not performed. There were three cases of equine grass sickness all with the findings of caecocolic impaction with small intestinal and gastric reflux. Histopathology was not performed.

Four musculoskeletal cases were examined. One was found to have septic arthritis of the tarsus. One case was diagnosed with a right hind fracture. One case was diagnosed with a penetrating hoof injury. There was one case of traumatic injury with a fracture of thoracic vertebral body (T4) and associated fractures of costovertebral junctions (right T4 and left T5).

One neoplastic case was examined and diagnosed with a pancreatic mass and secondary haemabdomen.

One ophthalmic case was examined which had a clinical history of blindness, no significant findings were made on post-mortem examination.

One respiratory case was examined and diagnosed with bronchopneumonia and pleurisy.

Two cases of sudden death were examined and both were found to have a fracture of one thoracic vertebral body, bilateral rib fractures and rupture of the aorta with secondary haemothorax.
South West England
A total of five cases were examined.

One cardiac case was examined and diagnosed with mitral valve degeneration.

One case suffering from gastrointestinal disease was examined and diagnosed with a small colon impaction.

One case suffering from neoplasia was examined and diagnosed with an oral papilloma.

Two cases with dental disease were examined and one had a chronic focal ulcer and the other had severe dental disease.
ACKNOWLEDGEMENTS

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

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 AHVLA, which acts as the reference laboratory.

Agri-Food and Biosciences Institute of Northern Ireland
Animal Health Trust Diagnostic Laboratory Services
Animal and Plant Health Agency
Austin Davis Biologics Ltd
Axiom Veterinary Laboratories Ltd.
Biobest Laboratories Ltd.
BioTe Veterinary Laboratories.
B & W. Equine Group Ltd.
Carmichael Torrance Diagnostic Services
Chine House Veterinary Hospital
The Donkey Sanctuary
Donnington Grove Veterinary Group
Endell Veterinary Group Equine Hospital
Hampden Veterinary Hospital
IDEXX Laboratories
JSC Equine Laboratory
Lab Services Ltd.
Liphook Equine Hospital
Minster Equine Veterinary Clinic
NationWide Laboratories
Newmarket Equine Hospital
Oakham Veterinary Hospital
Rainbow Equine Hospital
Rossdales Laboratories
Royal Veterinary College
Sussex Equine Hospital
Three Counties Equine Hospital
Torrance Diamond Diagnostic Services (TDDS)
University of Glasgow
Valley Equine Hospital

The Animal Health Trust (AHT) is extremely grateful to the Horserace Betting Levy Board (HBLB), Racehorse Owners Association (ROA) and Thoroughbred Breeders’ Association (TBA) for their continued combined contribution to the AHT’s Equine Infectious Disease Service.

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

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