NSW GOVERNMENT

Information contributed by staff of
NSW Department of Primary Industries
and Local Land Services

NEW SOUTH WALES

ANIMAL HEALTH SURVEILLANCE

January– June 2019 » Issue 2019/1

In this issue

Background to the NSW DPI-LLS animal disease and pest surveillance program

The NSW Department of Primary Industries (DPI) is obliged under the *Biosecurity Act 2015* to detect and manage notifiable animal disease outbreaks. This obligation is met by government veterinary officers, from Local Land Services (LLS) and DPI that are required to investigate potential notifiable disease outbreaks and unusual diseases that may be new, emerging or difficult to diagnose. They also conduct targeted surveillance projects, inspections of stock at saleyards and monitoring of compliance programs.

The desired outcome is the early detection of notifiable diseases, including exotics, and thus minimisation of negative impacts, and accurate, verifiable data on the animal disease and pest status of NSW. Reports are collated at the state level, for subsequent official reporting to the National Animal Health Information System (NAHIS), which is managed by Animal Health Australia. The NSW surveillance program is supported by Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) and by research staff who design and improve diagnostic tests and, working with field veterinarians, investigate the epidemiology of diseases that may have significant biosecurity impacts.

Surveillance by NSW Government in Q1 and Q2 (January – June)

The key outputs of surveillance by the government veterinary service in NSW are the detection and exclusion of exotic and endemic notifiable diseases so that disease-control strategies can be put in place to minimize the impact of these diseases if detected, and to support claims of disease freedom through evidence of absence of disease when excluded.

In Q1 and Q2 of 2019 (Jan – June) more than 1,200 disease investigations were conducted by the NSW Government. The most common species investigated were cattle and sheep (See Figure 1 and 2).

The most commonly investigated syndromes in cattle and sheep were found dead, sudden death, illthrift / weight loss / poor body condition and nervous signs. In 74% and 81% of cases respectively for Q1 and Q2, more than one syndrome was described (see Figure 3).

This data is only an analysis of investigations by the NSW Government and does not include those undertaken by private practitioners.

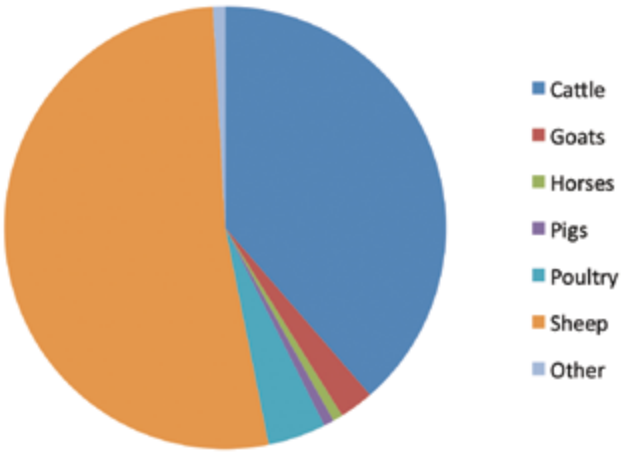

Figure 1: Q1 (Jan-Mar) 2019 Diagnostic Events by NSW Government. Graph by C. Harrison.

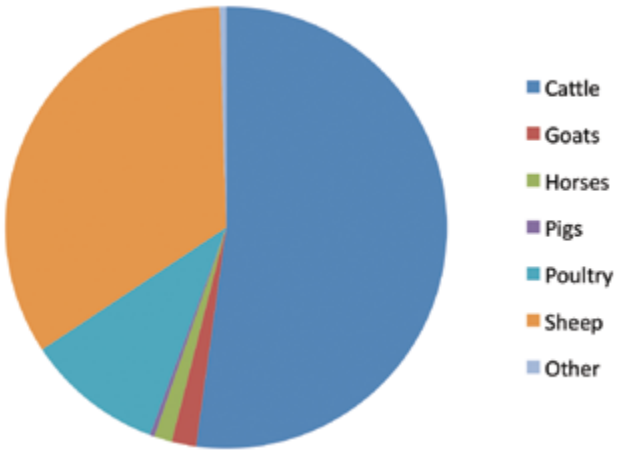

Figure 2: Q2 (Apr - Jun) 2019 Diagnostic Events by NSW Government. Graph by C. Harrison.

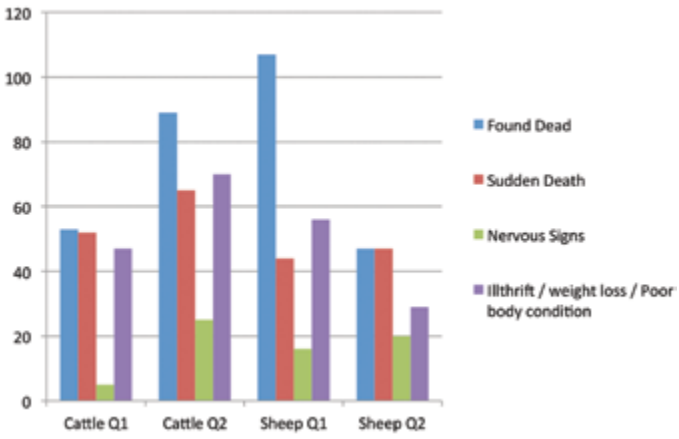

Figure 3: Most commonly described syndromes in cattle and sheep for Q1 and Q2 2019. Graph by C. Harrison.

For further information contact Claire Harrison, Veterinary Policy & Project Officer, NSW DPI Animal Biosecurity, Orange NSW, on (02) 6391 3490.

NSW Anthrax Report

Jan - March 2019 (Q1)

There was one presumptive diagnosis of anthrax during the quarter. In early February a producer from the Nyngan district noticed 100 dead lambs after being away for a few days. One lamb was taken to a private practitioner who performed a necropsy. During the procedure, the anthrax immunochromatographic test (ICT) was positive two times. Samples were not taken for laboratory confirmation because the carcass was immediately sealed in a bag and a decision was made to not reopen it.

More than 300 lambs eventually died from a total of 3,000 sheep at risk on the property. Biosecurity directions were applied to both the source property and the veterinary practice as per the NSW DPI anthrax procedure. Two people were exposed to the open carcass and were followed up by NSW Health.

Carcasses were disposed of by burning, and 4,000 sheep were vaccinated. No movements on or off the property were recorded on NLIS in the past four months.

Fifty other mortality investigations excluded anthrax during the quarter. Thirty-four of these were in cattle where alternate diagnoses included bloat, pneumonia, metabolic disease, and toxicities due to cyanobacteria, nitrate/nitrite, and urea. Fifteen investigations involved sheep where alternate diagnoses included clostridial infection, lactic acidosis, salmonellosis and toxicities due to nitrate/nitrite and pyyolizidine alkaloids (*Heliotropium* spp). One investigation involved a kangaroo where no alternative diagnosis was found.

The ICT was conducted in twenty-six of the cattle investigations with negative results. Twelve of these investigations were confirmed negative by laboratory investigations (polychrome methylene blue, PCR or both). The ICT was used in ten of the sheep exclusions, of which three were confirmed negative by laboratory testing.

The remaining five sheep and eight cattle exclusions were based on alternate diagnoses or clinical grounds. The kangaroo was excluded by laboratory testing.

April - June 2019 (Q2)

There were no diagnoses of anthrax during the quarter.

There were fifty-nine mortality investigations during the quarter. Of these fifty-three involved cattle where alternative diagnoses included Clostridial infection, toxicities due to Sorghum spp, ptaquiloside - *Cheilanthes sieberi* and ptaquiloside - *Pteridium esculentum*, hepatopathy, pneumonia, traumatic reticulopericarditis, and bloat. Four investigations involved sheep where alternative diagnoses included Clostridial infection and Salmonella enteritis.

One investigation involved a kangaroo with no alternative diagnosis and one horse where the alternative diagnosis was positive for Hendra virus.

The immunochromatographic test (ICT), to diagnose Anthrax was conducted in forty-two cattle investigations with negative results. Eleven of these were confirmed negative by laboratory testing. The ICT was used in two sheep investigations with negative results and confirmed negative by laboratory testing.

The remaining two sheep and eleven cattle exclusions were based on alternate diagnoses or clinical grounds. The kangaroo was excluded by laboratory testing. The horse was excluded based on the positive Hendra virus result.

For further information contact Barbara Moloney, Technical Specialist Disease Surveillance, NSW DPI, Orange, on (02) 6391 3687.

Salmonella Enteritidis

The NSW egg and poultry industries are currently engaged in an effort to stop the spread of *Salmonella* Enteritidis (SE) and manage its impacts after it was detected in NSW for the first time in September last year. The detection followed confirmation of twenty-three human cases of SE gastroenteritis in the Sydney area. Investigations by the NSW Food Authority found that several of those who had fallen ill had consumed eggs from one commercial layer farm in South West Sydney.

Since the initial detection, NSW Department of Primary Industries (DPI), NSW Food Authority, NSW Health and Local Land Services (LLS) have increased monitoring and surveillance to limit the impact of SE on producers and consumers. As a result, thirteen infected properties have been identified since SE was first discovered in NSW. All of these identified properties are interconnected in that people, eggs or equipment have moved between them.

The approach taken by NSW DPI to manage the infected properties in NSW has remained consistent with the principles outlined in the industry SE response plan published by Australian Eggs Pty Limited. Infected properties have been issued with biosecurity directions to prevent movement of birds, eggs, manure, soil and materials. Depopulation of birds, disposal and decontamination programs have then been implemented. Surveillance at properties where SE has been detected is ongoing.

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The investigations have reinforced the ongoing need to continually review biosecurity measures to ensure best-practice arrangements are in place for the safety of consumers and industry. That is why NSW DPI continues to work with industry and affected producers to lift biosecurity standards and ensure a sustainable egg industry.

SE is the most common serotype of *Salmonella* isolated from human cases of foodborne gastroenteritis around the world. In young chickens, less than two weeks of age, infection with SE can result in symptoms of gastroenteritis, including poor growth, weakness, diarrhoea, and dehydration. In contrast, adult hens that become colonised with SE typically remain asymptomatic with intermittent faecal shedding. This form of *Salmonella* has the specialised ability to colonise the avian reproductive tract and contaminate the internal contents of eggs.

Several resources have been developed since September 2018 and have been made available to assist producers to protect their poultry farms from SE. Current information about SE in NSW can be accessed using the dedicated NSW DPI SE webpage **www.dpi.nsw.gov.au/SE**.

For further information contact Jenna Fraser, Veterinary Policy & Project Officer, NSW DPI Animal Biosecurity, Paterson NSW on (02) 4939 8940.

Figure 1: Map of Infected Premises. Tableau.

Figure 2: Layer hens housed in a barn system with automatic feeders and drinkers. Since September 2018, SE has been detected on commercial farms that house chickens in cage, barn and free-range facilities. Image by J. Fraser.

Figure 3: There have been a number of egg recalls associated with detections of SE in NSW. Image by J. Fraser.

Figure 4: Staff from the Greater Sydney LLS region and the NSW DPI Animal Biosecurity unit collected whole blood and cloacal swab samples from layer chickens on the first infected property in September 2018. Image by J. Fraser.

Lowood Goose Herpes virus infection

On 9 January 2019 a poultry owner on a property in the Tweed Valley, north coast NSW, reported mortalities in geese. The owner had fourteen Brown Chinese geese that were free range and had access to a creek which had dried to a series of waterholes. The geese were all three months old, and both sexes were affected. Five had died over the preceding sixteen days. The sixth, a gander, was presented sick to a local veterinary practice with a special interest in avian medicine on 9 January. Deaths occurred on 23, 24 and 26 December 2018 and another two mortalities reported on 5 and 6 January 2019. In contact poultry were not affected. These were approximately twenty Blue Aylesbury ducks, thirty Black Orpington ducks and over twenty mixed age chickens.

All the geese were found by the owner with similar clinical signs progressing to death within 2-3 hours or were found dead. The gander was found sick on the morning of 9 January. The initial sign was quiet demeanour; the bird was usually aggressive. When presented to the clinic, it was sitting, not eating and had polyuria and polydipsia. During the following six hours, the bird deteriorated. It stopped drinking, and the faeces become watery, with brown urates and some blood. The bird then developed coarse respiratory rales. It did not have the typical flaccid neck paralysis of botulism. The practitioner notified the District Veterinarian (DV) on suspicion of Lowood Goose Herpes virus infection. In consultation with the DV and the owner, a decision was made to euthanise the bird late that evening. The practitioner requested the DV to undertake an autopsy and laboratory workup.

On autopsy, the goose was in good condition and well grown. Gross changes were confined to the liver and small intestine. The liver had well circumscribed, depressed, focal necrotic haemorrhages on the surface, of 1–2 mm diameter (Figure 1). The small intestine had scattered pale, focal, raised lesions on the mucosa, 1–2 mm diameter (Figure 2). The lymphoid tissue of the small intestine had multiple, fine, petechial haemorrhages.

Brain, heart, liver, lung, kidney, spleen, pancreas, oesophagus, gizzard, proventriculus, small intestine, large intestine and caecum were sampled fixed for histopathology and fresh. Small and large intestine were submitted for *Salmonella* culture and intestinal smears for coccidia. Gizzard content was sampled for toxicology if required. Cloacal and tracheal swabs in Phosphate Buffered Glycerol Saline (PBGS) were submitted for Avian Influenza and Newcastle Disease virus exclusion.

Herpes virus was isolated in chicken embryo fibroblast (CEF) cells from both the cloacal swab and fresh liver. Herpes virus was also confirmed on electron microscopy. Histopathology of the liver had suspicious herpes virus changes - multifocal, moderate, acute, hepatic necrosis with intranuclear inclusion bodies. There was marked multifocal to coalescing, acute splenic necrosis. The lungs had marked congestion and infiltrate of mixed leucocytes. There was evidence of multifocal, mild, acute necrotising oesophagitis. The proventriculus and elsewhere in the gastrointestinal tract had increased lymphocyte and plasma cells, but changes were masked by autolysis. Despite being refrigerated immediately after euthanasia, being a waterfowl, geese have remarkable insulation afforded by the feathers and subcutaneous fat. The bird's natural insulation slowed chilling to internal organs, allowing a degree of autolysis in the twelve hours between death and autopsy). Histopathology of heart and kidney was unremarkable.

Salmonella, coccidia, Avian Influenza and Newcastle disease were all negative. Tests for toxicology and botulism were not conducted.

This is understood to be the second time that deaths from Lowood Goose Herpes virus has been recorded in NSW, with a previous incident in Muscovies about eight years ago on a property in the Tweed Valley, approximately 20 to 30 km from this site. The first recorded outbreak was at Lowood Queensland in 1989. Free-flying wild ducks are suspected to be the wildlife reservoir with virus spill-over occurring when wild ducks have contact with domestic geese. In this event, the owner observed that Pacific Black ducks and Whistling ducks frequented the waterway.

Investigations into multiple deaths of domestic and free-flying birds are critical, not only to confirm unusual diseases but to exclude important notifiable diseases such as Avian Influenza and Newcastle disease.

For further information, contact Phillip Kemsley District Veterinarian Lismore, North Coast Local Land Services on 0427 896 822.

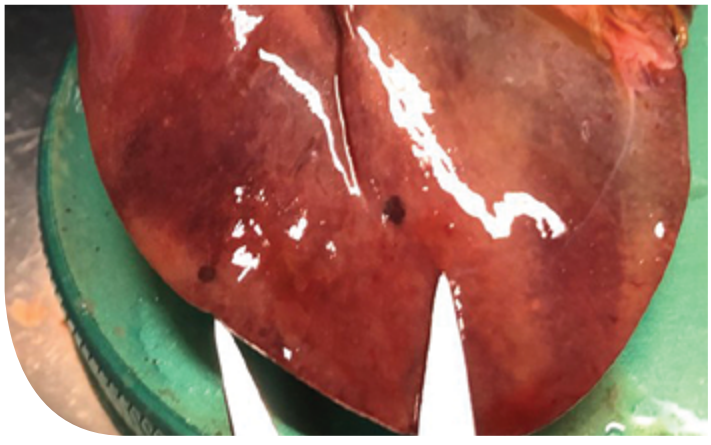

Figure 1: Changes observed in the surface of the liver; depressed, focal haemorrhagic necrosis. Image by P. Kemsley.

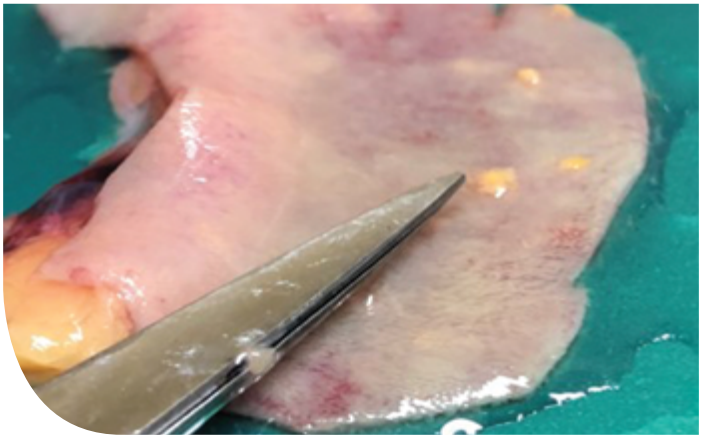

Figure 2: Changes observed in the small intestinal mucosa; raised nodules and multiple petechial haemorrhages of intestinal lymphoid tissue. Image by P. Kemsley.

Transmissible Spongiform Encephalopathy (TSE) excluded with a diagnosis of cerebellar abiotrophy in an ataxic adult merino ewe

In January 2019 a superfine merino wool producer in the South East region informed their District Veterinarian (DV) of a three-year-old, home-bred ewe that was showing signs of ataxia. Other ewes and wethers had been similarly affected in the past, having progressed to recumbency and death. Examination revealed pronounced ataxia, mostly in the hindlimbs, and a mild head tremor. The ewe stumbled and fell when under stress, then struggled to rise, and occasionally adopted a "dog-sitting" posture (i.e. tarsal flexion). She appeared to have normal mentation and vision and was otherwise unremarkable on physical examination.

The ewe was sedated with 1ml ACP 10mg/ml given IM and euthanased using a captive bolt device on the poll in vertical alignment to provide a prompt, safe and humane euthanasia while avoiding damage to the hindbrain. No gross abnormalities were identified during necropsy.

The primary differential diagnoses were Scrapie, copper deficiency, phalaris toxicosis and genetic disorders, including ovine segmental axonopathy and cerebellar abiotrophy. Vitamin A deficiency was considered possible but unlikely given access to green pasture.

Scrapie is a Transmissible Spongiform Encephalopathy (TSE) that occurs in both classical and atypical forms, which differ in their neuroanatomical distribution of neuropil vacuolation. The classical form is exotic to Australia, while there have been sporadic outbreaks of the atypical form. The introduction of classical Scrapie to Australia could result in the loss of export markets at an estimated cost of between AUD$35 million to $2.2 billion. Scrapie typically occurs in sheep of either sex between two and five years of age. Scrapie manifests clinically in animals under stress (e.g. handling), and is associated with locomotor incoordination, head tremor, and behavioural change, all of which was observed in this case.

The entire brain, a segment of the cervical spinal cord and a range of other samples were submitted to Laboratory Services at the Elizabeth Macarthur Agricultural Institute (EMAI), through the National Transmissible Spongiform Encephalopathy Surveillance Project (NTSESP), for exclusion of Scrapie and investigation of alternative diagnoses.

There were two striking histopathological lesions: within the cerebellum, Purkinje cells (neurons) were diffusely lost, degenerate or necrotic, with some replacement by Bergmann-type glial cells, and within the spinal cord there was bilaterally symmetrical Wallerian degeneration (axonopathy). Both lesions were consistent with cerebellar abiotrophy (CA), also known as cerebellar cortical degeneration, or "Yass Ataxia". Additionally, there was minor, scattered neuropil and myelinic vacuolation within the cerebellum and brainstem, and an individual intraneuronal vacuole within a dorsal vagal nucleus. These findings prompted prion protein testing for exclusion of Scrapie, which was negative. Copper deficiency, which can also cause Wallerian degeneration in the spinal cord, was excluded by measurement of liver copper. Vitamin A deficiency, for which there was no histopathological evidence, was also excluded by measurement of plasma Vitamin A levels.

Cerebellar Abiotrophy, is an inherited syndrome causing late-onset cerebellar cortical degeneration. It was first recorded in 1974 in the Yass region. Sheep with this condition will fall over due to equilibrium disturbances and incoordination when hurried or handled, followed by vigorous and often futile attempts to stand again (Bourke, 1995). The disorder is reported in fine wool merinos aged between one and six years of age. The specific genetic cause of CA is currently unknown, and consequently, there is no pre-mortem testing available.

For further information contact Lou Baskind, District Veterinarian, South East Local Land Services, Braidwood, on (02) 4842 2594.

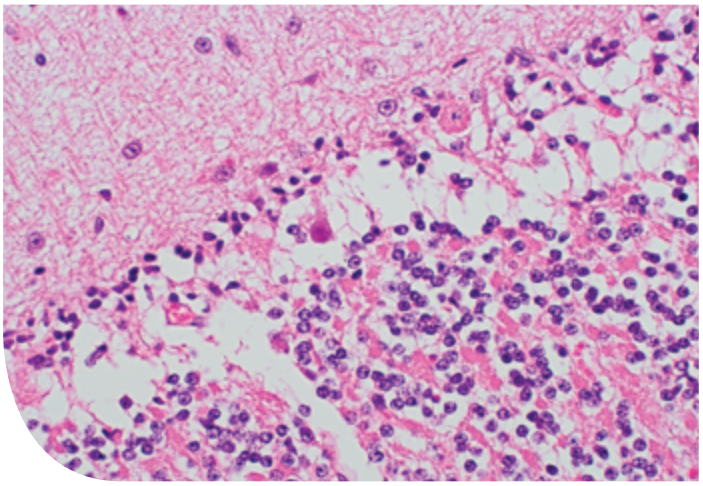

Figure 1: Cerebellum. Between the granule cell layer (bottom) and molecular layer (top), there is a linear clearing, in which Purkinje cells are either absent, creating 'empty baskets', or necrotic (centred). There are increased numbers of glial cells clustered at the interface between the Purkinje cell and molecular layers (Bergmann gliosis). Image from SIU SOM.

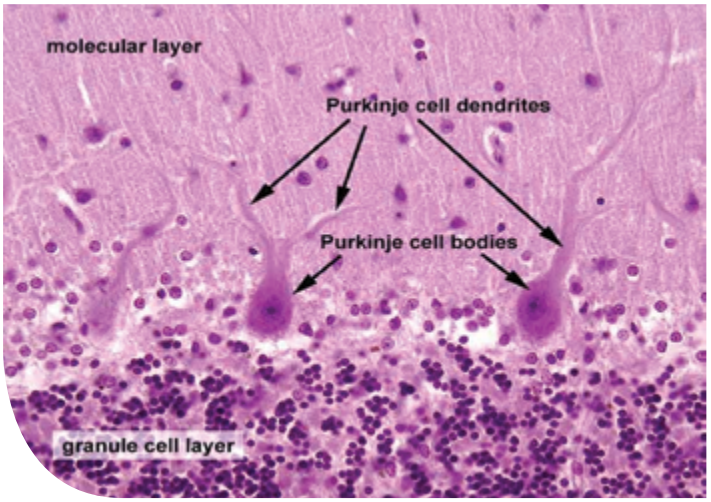

Figure 2: Normal cerebellum for reference. Image from SIU SOM.

Bluetongue virus excluded from lambs with face and ear lesions

A sheep producer in the NSW Central Tablelands noticed eight dead and five sick lambs in a group of 650 second-cross lambs. The June 2018 drop lambs had been weaned onto grazing brassica plus pellets in a self-feeder and in early January, the producer moved the group to paddocks with summer grasses. The owner noticed the sick and dead lambs two weeks later.

Three lambs were temporarily moved to a yard for examination and blood testing by a government veterinarian. The affected lambs were bright and alert but had raw bleeding ears, swollen faces, and hair loss from the face and ears (Figure 1). One lamb had slightly icteric mucous membranes. All lambs scratched and rubbed their ears and faces, especially when standing in the sunlight.

The most likely diagnosis was hepatogenous photosensitisation. Clinical bluetongue virus (BTV) infection was considered a differential diagnosis. Clinical signs of bluetongue disease can include depression; swelling of lips, tongue, gums and face; cyanotic (blue) tongue; lameness; unwillingness or inability to stand; pneumonia and laboured breathing. Clinical bluetongue disease is not observed in Australia, although BTVs confined to northern Australia are known to be potentially pathogenic in sheep. In overseas cases, sheep are the most severely affected of ruminant species.

The blood tests revealed elevated glutamate dehydrogenase levels, indicating hepatocellular damage, and elevated gamma-glutamyltransferase levels consistent with biliary damage. One lamb had an elevated bilirubin level. The samples tested negative for BTV by polymerase chain reaction (PCR) assay.

Although grazing brassicas can cause photosensitisation in sheep, it is usually primary photosensitisation, without mortality. In this case, the lambs developed clinical signs after grazing in the summer grass paddock for two weeks. The owner had ploughed the paddock, but sweet panic (*Panicum gilvum*) was identified where the plough had not turned the soil. On the Central Tablelands in the past summer, there were several cases of hepatogenous photosensitisation caused by *P. gilvum*, often with substantial morbidity and mortality.

The owner removed the lambs from the suspect paddock, and no further deaths occurred. The three affected lambs were nursed in the shade until they recovered completely.

For further information contact Bruce Watt, District Veterinarian, Central Tablelands Local Land Services, Bathurst on (02) 6333 2303.

Figure 1: Photosensitised lambs in yard with summer grasses, including *Panicum gilvum*. Image by B. Watts.

Nitrate/nitrite poisoning in the Northern Tablelands region

Nitrite poisoning was the cause of two mass, sudden death events affecting cattle in the Northern Tablelands Local Land Services region.

In late February, cattle were being fed untested, home-grown sorghum hay which had been utilised with no issues a week prior. Hay was fed in the late afternoon and overnight 75 of 336 Pregnancy Tested in Calf (PTIC) Angus cows, and 10 of 340 PTIC Angus heifers were found dead. Externally there was evidence that some cows had struggled before death, and a small number of carcasses had blood from the nostrils and anus. An anthrax immunochromatographic test (ICT) test was negative, and no significant findings were observed on post-mortem. Aqueous humour analysis confirmed nitrate/nitrite poisoning. Four feed samples submitted found nitrate levels up to 30,600 ppm (safe range < 3,080 ppm) and were also positive for cyanide.

In early March, another event occurred on a different property where 22 of 70 mixed breed, six-month-old weaner calves died. They were fed millet hay and soon started to show signs of discomfort, became recumbent and died over the following five hours until access to the hay was removed. No further losses were incurred following a switch to lucerne hay. Aqueous humour analysis confirmed nitrate/nitrite poisoning and a feed test found nitrate at 25,000 ppm.

In both cases, other bales of hay from the same cut had been fed without incident, highlighting the variability of nitrate content within the feed and the impact of individual animal factors on susceptibility. As drought conditions persist and cattle are being maintained on supplementary feed, this issue continues to pose a threat to livestock production in the region.

For further information contact Amanda Walker, District Veterinarian, Northern Tablelands Land Services on 0439 836 673.

Gypsum toxicity in crossbred ewes causing severe renal disease

In February 2019, a producer reported twelve crossbred ewes dead and thirty sick in a herd of 1,000. They were dry ewes running on a stubble paddock for the two weeks leading up to the event, with a pile of gypsum in the paddock ready for spreading. They had been supplemented with canola hay, pasture silage and two days before losses were observed, the herd had been fed approximately 0.5kg/head of wheat. Clinical signs of affected ewes were depression with low head carriage, scouring and mucoid nasal discharge. Affected ewes would go into sternal and lateral recumbency before dying 24 hours after initial onset of clinical signs. No ewes appeared blind. By the fourth day, approximately 100 ewes had died and twenty sick. Lactic acidosis was considered to be the main differential.

Postmortems were performed on four affected ewes, revealing rumens largely full of roughage, some wheat and a pH between 6-7, inflamed or ulcerated abomasums containing large amounts (up to 2 cups worth) of sand-like substance, presumed to be gypsum (Figure 1), dark, friable livers; and abnormal kidneys, ranging from pallor with striations evident in the cortex and medulla and bulging on the cut surface (Figure 2), to atypically dark.

Samples were collected and sent to Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI). Aqueous and serum results showed normal D-lactate and extremely elevated sulphate of 9.9 and 12.6 mmol/L respectively (ref. 0.7-2.0 mmol/L), ruling out the possibility of lactic acidosis. Histopathology revealed ruminitis, abomasitis and severe changes in the kidneys with acute tubular necrosis and degeneration. Blood and urine samples from sick ewes also demonstrated severe renal damage and failure.

Fluoride may also result in similar lesions and therefore, samples were sent to the National Measurement Institute, to measure the fluoride on blood samples collected from sick ewes. No fluoride was detected. Sulphur toxicity was considered the primary diagnosis, given its ability to cause polioencephalomalacia (PEM), can cause direct toxicity to the rumen and abomasum and can result in nephropathies (as sulphur is excreted in the urinary system). This form of sulphur toxicity is rarely reported. Other nephrotoxic agents suggested by EMAI included toxic plants (such as redroot pigweed, oak and lesser loosestrife) mycotoxins (such as ochratoxin) and heavy metals (such as lead, arsenic, mercury, cadmium). However, these were all excluded, being unavailable to the ewes.

Oral fluid replacement therapy was initiated for affected ewes; however, they were unresponsive to supportive treatment and mortalities continued over a two-week period, resulting in 190 deaths (19% of the mob).

Many producers run sheep in paddocks where gypsum has been dumped before spreading; however, they have rarely resulted in mortalities and is most likely associated with the volume ingested - perhaps out of hunger or due to mineral deficiencies. Cases of sudden death often present with clinical signs suggestive of a new, emerging or exotic animal disease. As such, these cases form an important part of notifiable disease surveillance in NSW and emergency animal diseases should always be considered as differential diagnoses.

For further information contact Belinda Edmonstone, Nik Cronin and Hanna Thomas, District Veterinarians, Central West Local Land Services on (02) 6841 6500.

Figure 1: Substance (most likely gypsum) coated the rumen wall. Image by N. Cronin.

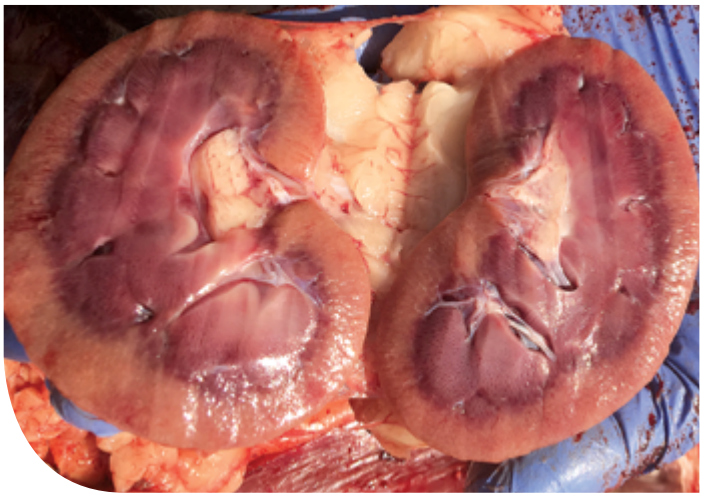

Figure 2: Bulging cut surface of pale kidney. Image by N. Cronin.

Nitrate toxicity in beef cattle fed millet hay

A Deniliquin District Veterinarian (DV) was contacted by a Private Veterinarian (PVP) in February 2019 to assist with a mass mortality event in a beef cattle herd in the Murray region. The herd consisted of around 120 cows and calves and 200 weaners. The producer initially estimated seventy weaners and thirty cows died overnight. All cattle were considered healthy the previous evening when they were fed millet hay. The affected cattle were found dead the next morning, and there was no sign of illness in the herd.

Nitrate/nitrite poisoning was the primary differential diagnosis however, the PVP first ruled out anthrax, another possible differential. An anthrax Immunochromatographic Test (ICT) was performed on-farm and returned a negative result. Following this, the PVP performed multiple post mortem examinations. There were no significant gross findings in any of the carcasses examined. A range of tissues were collected for laboratory examination, including aqueous humour. Five aqueous humour samples were tested, three of which returned a nitrate level of 50 ppm and the other two showed 25 ppm (where normal levels should be zero). All five were positive for nitrite, confirming the diagnosis of nitrate/nitrite toxicity. Nitrate/nitrite poisoning occurs commonly in cattle as rumen microbes convert nitrate in feed to the more toxic nitrite. Nitrite, when absorbed into the bloodstream, alters the oxygen-carrying haemoglobin so that it can no longer carry oxygen. Death occurs when more than 80% of the haemoglobin is altered so that it cannot carry oxygen.

Three samples of the hay that had been fed the previous evening were tested. All had a nitrate level of 50,000 ppm and were negative for nitrite and cyanide, confirming the hay as the source of nitrate. While individual animal factors and weather conditions can alter susceptibility to nitrate toxicity, usually levels of more than 5,000 ppm of nitrate in feed are thought to be potentially toxic to ruminants.

The cattle had been fed a diet of predominantly millet hay for around ten days. The owners grew their own irrigated Shirohie millet in a paddock on the farm. Some of the millet had been continually grazed by the mob of cows and calves, and some had been cut for hay and bailed. Hay from the first bay had been fed out without incident, but the mortality occurred when hay from the second bay was fed out for the first time, highlighting the variability of the nitrate content within the feed. Seven samples of millet were taken from across the whole paddock and ranged in nitrate levels from around 1,500 ppm to 30,000 ppm. There were no risk factors identified that were likely to have contributed to the high nitrate levels or the variation across the paddock, for instance, the millet did not appear stressed at any stage, and no fertiliser had been applied since sowing. This is unusual as traditionally the risk of nitrate/nitrite poisoning is thought to be highest in grazing crops such as oats, sorghum and sudan grass and weeds such as capeweed and not millet. The producer removed and destroyed the most toxic hay and fed the remaining millet carefully by restricting access. No further deaths occurred after the initial event, and a total of 140 cattle were estimated to have died.

Deniliquin, in the Riverina region, lies within the classic 'anthrax belt' and as such anthrax should always be on the list of differential diagnoses in cases of sudden death, particularly after heavy rain in denuded pasture conditions.

For further information contact Scott Ison, District Veterinarian, Murray Local Land Services, Deniliquin, on (03) 5881 9900.

Kikuyu toxicity in the North Coast region

In early March 2019, a Tyringham beef producer contacted the local District Veterinarian (DV) when several animals were found dead or sick, after being let in to graze a corn crop three days prior. The crop had been planted in December and at the time received a nitrogenous fertilizer application. In the coming months, the crop showed signs of stress while enduring a drier than average summer. Approximately a week before the cattle were let in to graze, an army worm infestation was treated and the grazing withholding period was adhered to. No issues were observed in the mob during the first two days of grazing the crop. When it was discovered that cattle had become sick, they were removed from the crop immediately and put back on to a kikuyu paddock on which they had been set stocked prior to grazing the crop. This paddock had no recent fertilizer history.

On examination of the crop, it was noted that the corn plants had been stripped of all cobs and leaves and that the cattle had begun grazing a significant understory of kikuyu.

On physical examination, affected survivors were severely depressed with some recumbent, some standing with low head carriage and some standing in a 'sawhorse' stance. All affected cattle exhibited marked drooling, mild to moderate hind limb ataxia and severely sunken eyes. While nitrate toxicity was considered, it was unlikely given that mucous membrane colour was normal in all animals. Anthrax was also considered as a possible differential diagnosis, however, was negative on two immunochromatographic (ICT) tests performed on two deceased animals and was less likely given that the property resided external to the 'anthrax belt'. Postmortem of a freshly deceased animal revealed an extremely large volume (20-40L) of opaque, bright green rumen fluid with a pH of 7.5. Solid rumen contents were largely made up of leaf matter with a very small amount of corn. Aqueous and vitreous humours were negative for nitrate and nitrite on test strips. There was a small area of multifocal epicardial haemorrhages on the left atrium, and rumen contents were evident in the airways of the cranioventral lung lobes. Postmortem of another deceased animal revealed similar findings.

Antemortem and postmortem samples were submitted to the Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) for haematology, biochemistry and histopathology. Haematology and biochemistry from multiple animals revealed marked haemoconcentration (PCV up to 59%, ref. 23-44%), azotaemia (urea up to

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23.9mmol/L, ref. 2.1-10.7mmol/L, creatinine up to 649umol/L, ref. 0-186umol/L), hyperphosphataemia (phosphate up to 5.01mmol/L, ref. 0.8-2.8mmol/L) and hyperkalaemia (potassium up to 13.1mmol/L, ref. 3.9-5.8mmol/L). Analysis of a postmortem aqueous humour sample revealed a marked elevation in urea (14.3mmol/L, ref. 2.1-10.7mmol/L) with normal nitrate, nitrite, D-lactate, calcium, magnesium and beta-hydroxybutyrate. Histopathology of the rumen of one animal revealed multifocal ulceration of the mucosa with intervening areas of moderate epithelial hypertrophy and hyperkeratosis. The lamina propria was expanded by neutrophils, lymphocytes, plasma cells and colonies of bacteria located under the ulcerated epithelium and around blood vessels. The inflammatory infiltrates, with some bacteria, extended into the submucosa and muscularis with areas of oedema and congestion. Histopathology of the omasum showed similar lesions.

Deaths continued for a further three days with a total of fifteen deaths and three affected animals recovering without treatment out of a total of approximately eighty head.

Kikuyu toxicity was diagnosed through clinical and postmortem examinations. A media release was produced by North Coast Local Land Services warning local producers of the risks associated with grazing stressed crops and pastures following recent rain. No other outbreaks were reported in the local area in the weeks that followed.

To date, kikuyu toxicity as a disease entity is poorly understood. Poisonings have occurred in association with army worm infestations, prolonged dry periods followed by rain and conditions that favour fungal growth. Proposed mechanisms of toxicity include toxin production within the plant and fungal toxicity.

For further information contact Sarah Bolton, District Veterinarian, North Coast Local Land Services, Grafton, on (02) 6604 1100.

Kikuyu toxicity in the Hunter region

On 17 March 2019, a producer from Quorrobolong in the Lower Hunter region noticed that two Angus cows were showing clinical signs of excessive drooling, staggering, muscle twitching, anorexia, occasional groaning and recumbency. They were separated from the herd and kept in the yards to monitor more closely. They were fed lucerne hay but were not interested in eating. One of the cows died overnight, and the Local Land Services (LLS) was contacted to investigate. When walking through the herd, two other cattle showed clinical signs of ill thrift and drooling.

There were fifteen empty cows in the herd that had been on a four-hectare paddock of 98% Kikuyu (approximately 2% pigweed and couch grass) for five days before the onset of clinical signs. This paddock had been fertilised in 2018 with urea in September and boron and molybdenum in October. 2018 was 200mm below the average rainfall for that area and the driest since 2002. On the 5 March, the paddock was fertilised with potash, manganese sulphate, boron and copper sulphate after soil testing had been done. The cattle were moved into the paddock on the 13 March, and 60mm of rain fell on 16 March. The kikuyu was 100-150mm in height and bright green in colour.

A post mortem was performed, revealing a large volume of green fluid that had come out of the mouth, an excessive amount of fluid in the rumen (approximately three times normal rumen fluid content) and the liver was firm to cut with a slight acinar pattern noticed.

The cattle were removed from the suspect kikuyu paddock and given lucerne hay as an alternative feed source. All other cattle recovered after removal from the toxic paddock.

Given the clinical presentation and some signs consistent with Bovine Spongiform Encephalopathy (BSE), the brain was submitted to the National Transmissible Spongiform Encephalopathy (TSE) program for TSE exclusion. Pasture samples were submitted for analysis, looking at the mineral content (calcium, magnesium and potassium), oxalate and nitrate levels. Samples were also collected for fungal culture at the University of Sydney, where several fungi have been grown including *Fusarium* species, *Nigrospora* and *Bipolaris* species.

Histopathology results from Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) showed severe necrosupporative omasitis with mild inflammatory changes in the rumen and abomasum consistent with kikuyu poisoning. Common signs of kikuyu poisoning include drooling of thick, ropey saliva, sham drinking, dehydration, inappetence, abdominal pain and recumbency. The poisoning affects the fluid balance, resulting in an accumulation of fluid in the rumen, which is not absorbed into the bloodstream. This is presumably what causes the 'sham drinking' - the dehydrated animal feels thirsty but is unable to fit more fluid into its stomach.

Kikuyu poisoning is still a bit of a mystery. Kikuyu poisoning occurs when cattle graze kikuyu that is rapidly growing after rain in late summer/autumn after a prolonged period of summer drought. There is no visible sign on the kikuyu pasture to indicate that it is affected, other than the fact that cattle will often leave it ungrazed if they have the choice of eating something else. Toxicity occurs when all other grazing options have been exhausted, and the toxic grass is all that remains.

Unfortunately, there are still many unanswered questions surrounding the nature of kikuyu poisoning. The Hunter region is compiling all results to better understand causation.

For further information contact Kylie Greentree, District Veterinarian, Hunter Local Land Services on 0428 498 687.

Figure 1: Excessive fluid in the rumen. Image by K. Greentree

Botulism in bats

In March 2019, a North Coast wildlife carer reported the sudden onset of paralysis and deaths in flying foxes at a release facility at Alstonville, North Coast of NSW. Within 24 hours all twenty-nine flying foxes were affected. Signs were a protruding tongue, reduced ability to swallow, weakness, progressing to loss of ability to perch and respiratory difficulty. The clinical course was from several hours to five days. The majority (25) were Black Flying Fox (*Pteropus alecto*), and four were Grey-headed Flying Fox (*Pteropus poliocephalus*). Both genders were represented. Twenty-six were juveniles almost ready for release, another was a juvenile still on its mother, and two were adults. Over the next five days, twenty-seven died or were euthanised, with only two recovering.

The flying foxes were sourced from three carers, with the majority (23) from the one carer. Twenty-seven arrived at the release facility on 3 March; 23 from one carer ("Carer A") and four from another. Another two arrived on 10 March from a third carer. One flying fox was found with paralysis on the evening of 11 March (Day 1) and died soon after. Because of previous incidents of this condition at the aviary, the remaining 28 were moved that evening to "Carer A".

On the morning of 12 March (Day 2), seven were found dead, and all twenty-one live flying foxes developed signs to varying degrees. The District Veterinarian (DV) examined these bats, and the twenty-one live bats were transported to a wildlife hospital. A total of fifteen died or were euthanised that day (Day 2). On 13 March (Day 3), six died. Three died on 14 March (Day 4) and two on 16 March (Day 6).

It is important to note that at "Carer A" there were in-contact flying foxes, which had not been to the release facility and all these remained in good health.

On 12 March the DV autopsied and sampled five flying foxes and a further two were sent whole to Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI). Apart from pulmonary congestion in two, there were no gross lesions.

Botulinum C toxin gene quantitative Polymerase Chain Reaction (qPCR) was positive in samples from the gastrointestinal tract of two of the five bats sampled. All seven were negative for Australian Bat Lyssavirus (ABLV). Histopathology on a range of tissues including brain from the five autopsied had no significant pathology, except for lung congestion and oedema; suspected to be due to shock. One had acute severe bacterial pneumonia; unclear as to was pre-existing or secondary.

The condition followed similar, but less severe events at the same release facility in 2014 and 2017. Between 6 March and 17 April 2014 a total of six juvenile flying foxes (both species and gender) presented with the same clinical signs; tongue protruding, inability to swallow and weakness. Two died, and four recovered.

In four days 27 February to 2 March 2017, eight of thirty-three juvenile flying foxes (both species and gender) developed the same signs. Four died, and four recovered.

The DV examined and sampled live and dead bats. Acute allergic reaction, ABLV or botulism were suspected as the possible cause. One of the bats autopsied was ABLV positive, and no samples were tested for botulism.

At the time of the events, there were high blowfly numbers at the release facility. The flying foxes are fed chopped fruit to which a protein supplement is added each afternoon. The water was the town's supply. The flies were attracted to the food. It is suspected that fly egg masses contaminating the feed were the source of the botulism toxin.

It is understood that this is the first time that botulism has been documented in flying foxes. Wildlife and bats, in particular, are considered important sources of new and emerging viral diseases of both domestic species and humans. For this reason, it is critical that unusual disease events in wildlife are reported and investigated.

For further information, contact Phillip Kemsley, District Veterinarian, North Coast Local Land Services on 0427 896 822.

Figure 1: Live Black Flying Foxes with tongues protruding. Image by P. Kemsley.

Figure 2: Dead Grey-headed Flying Fox flying fox with tongue protruding. Image by P. Kemsley.

Foot and Mouth Disease excluded in a saleyard

Following the discovery of a dead cow in a pen in mid-April, a North Coast district saleyard manager contacted the local District Veterinarian (DV) for an assessment. The cow had arrived at the saleyards the previous evening and appeared agitated and aggressive.

An anthrax immunochromatographic test (ICT) was performed and returned a negative result. On further examination, freshly ruptured vesicles with epithelial tags were noted at the ventral mucosal surfaces of each nostril (Figure 1). There was an absence of associated fibrin, and both lesions were approximately 1-2cm in diameter. There were no foot or udder lesions.

Given the differential diagnosis of Foot and Mouth Disease and other vesicular diseases, the DV contacted the Emergency Animal Disease Hotline and discussed the case with Veterinary Officers at the Department of Primary Industries.

A decision was made to examine all other animals from the same consignment with an emphasis on rectal temperature and presence of further lesions as well as drooling, depression or lameness. Of the twelve other animals in the consignment, two other animals exhibited erosions of the nasal mucosa (Figure 2), and interdigital epithelium (Figure 3) and two calves had mild fevers. All animals were bright with no evidence of depression, drooling or lameness.

Swabs and epithelial tissues from lesions along with blood samples were submitted to Laboratory Services, Elizabeth Macarthur Agricultural Institute (EMAI). A sample of aqueous humour from the deceased animal was also submitted. Foot and Mouth Disease was ruled out based on Polymerase Chain Reaction (PCR), antigen Enzyme-linked immunosorbent assay (ELISA) and serology while Vesicular stomatitis was excluded on PCR and Vesiculovirus serology also returned a negative result. Aqueous humour from the deceased cow returned a magnesium level of 0.81mmol/L (ref. 0.61-1.61 mmol/L) which, given the reported antemortem signs and considering that the concentration of magnesium in eye fluid increases with time post mortem, makes transit tetany a likely cause of death.

Following the investigation, discussion with the vendor revealed a history of access to red lantana, which could have been responsible for the lesions observed. Other possible causes include Bovine Viral Diarrhoea and Infectious Bovine Rhinotracheitis.

For further information contact Sarah Bolton, District Veterinarian, North West Local Land Services on (02) 6604 1104.

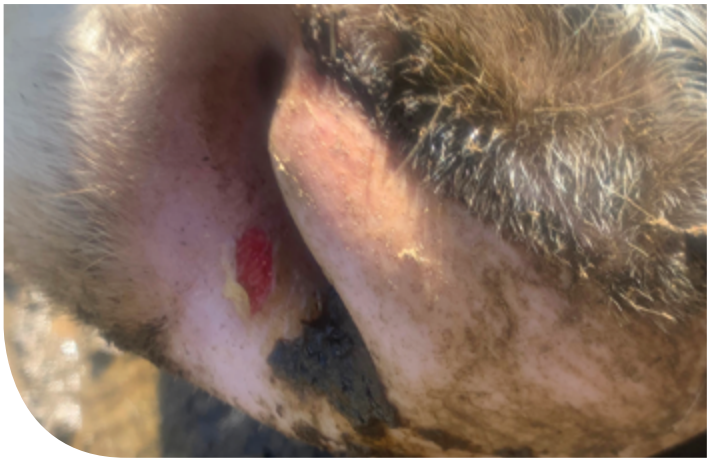

Figure 1: Deceased cow with freshly ruptured vesicle and epithelial tag, nasal mucosa. Image by S. Bolton.

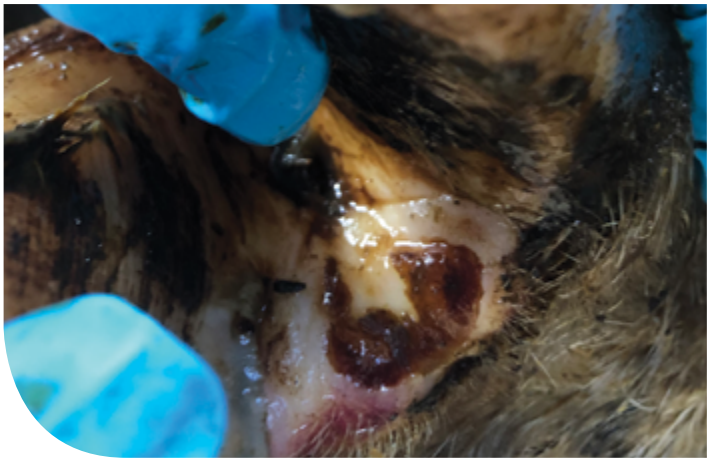

Figure 3: Erosion of the interdigital epithelium in a live, in contact animal. Image by S. Bolton.

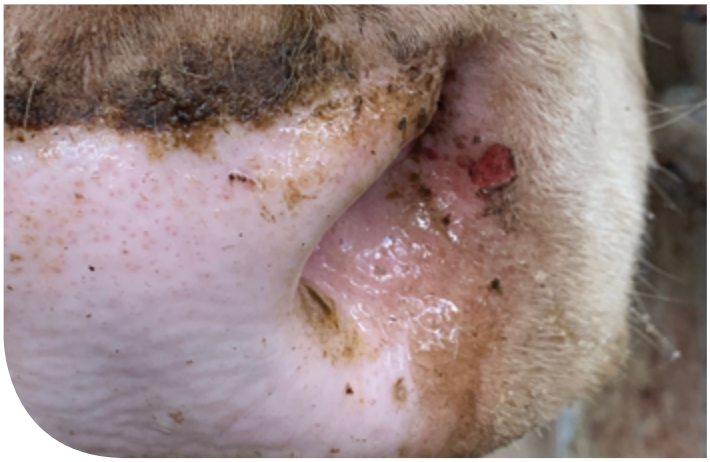

Figure 2: Erosion of the nasal mucosa in a live, in contact animal. Image by S. Bolton.

Figure 4: Ruptured vesicle along oral mucosa. Image by S. Bolton.

Bovine mastitis and concurrent pneumonia caused by *Mannheimia haemolytica*

A mob of 400 travelling stock suffered four deaths in the period of two weeks from mastitis on a Travelling Stock Route (TSR) near Monteagle, NSW in May 2019. The mob was made up of cattle from five different owners and had both adult cows (up to 6-7 years of age) and calves of varying ages. The mob had been on the road for well over twelve months.

The owner drover reported that the animals were noted to be lame on the side of the affected quarter, the quarter was swollen and the animals were dull and anorexic. In all cases, except one, the cows deteriorated and died within 3-4 days. The one surviving animal had a calf at foot who continued to suck from all quarters. In the last case, a clear mucous nasal discharge was noted the day before the cow died. Treatment with oxytetracycline antibiotic intramuscularly was unsuccessful in the animals it was tried in. All four of the affected animals were immediately pre-calving or in the first week of lactation.

There were no new additions to the mob in recent months, however a number of the cows were in late gestation or early lactation with numerous calves in the mob at the time of investigation. They had recently been locked up overnight in the communal lockup of the TSR and were on a slow moving travel permit so the cows had to be moving each day. Weather had been variable with a period of low night and day temperatures followed by a week of moderate day temperatures and then another drop to low night and day temperatures in the previous three weeks before cases developed.

A necropsy was performed on the fourth clinical case to confirm mastitis as the cause of the deaths. The milk in the affected quarter was watery and contained clots. The lungs also displayed signs of pneumonia characterised by consolidation and congestion. Culture of the lung and milk reported profuse pure growth of *Mannheimia haemolytica* sensitive to ampicillin, trimethoprim, tetracycline and cefuroxime.

Mannheimia haemolytica is a common cause of mastitis in sheep but rarely in beef cattle. Also in this case, it is difficult to determine if the pneumonia was the primary cause of disease and the mastitis secondary. Clinically, cases of mastitis alone have indicated progression to gangrenous mastitis in the cow but not a high incidence of deaths. Where as in this case, the udders remained hot, swollen and hard but deaths occurred within 3-4 days regardless. In Canada in 1955, Maplesden and Carter reported on a similar case of *Mannheimia* mastitis with concurrent pneumonia cases occurring in the calves at the time. In this travelling stock mob, no obvious signs of pneumonia were present in the other animals in the herd, nor were the signs obvious in the clinical mastitis cases.

Detection of affected animals and treatment in this case was made more difficult by the fact that the mob was travelling on the stock route, limiting suitable facilities for examination and treatment. All deaths of cattle on TSRs are investigated, so that a diagnosis is determined to ensure the overall disease status of a district is maintained.

At the time of writing, no further cases of mastitis had occurred in the herd.

For further information contact Eliz Braddon, District Veterinarian, Riverina Local Land Services, Young on (02) 6381 4700.

Figure 1: Oedematous, congested piece of lung. Image by E. Braddon.

Figure 2: Affected milk sample. Image by E. Braddon.

Hendra virus in the Hunter Valley region

The first case of Hendra virus infection in the Hunter Valley was confirmed on a property northeast of Scone on 11 June 2019.

The aged unvaccinated stock horse mare was isolated in a small yard on 7 June when she became stiff and lethargic in the morning and during the day became progressively ataxic. The temperature was not recorded. She developed polypnea and dyspnea and had a scant bilateral stable foam. Urinary incontinence was pronounced, and the horse was severely depressed, eventually progressing to lateral recumbency by the evening. Just before 10 pm that day the horse was shot on the property and taken to a burial area.

After considering the clinical history, a small animal veterinarian contacted the Emergency Animal Disease hotline on 9 June to see if Hendra exclusion testing could be done on the dead horse. A District Veterinarian (DV) from Hunter Local Land Services (LLS) visited the property later that day and collected blood and swabs from the dead horse and submitted these to the laboratory for Hendra Virus testing which was confirmed on all samples by Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) on Tuesday 11 June. (Monday was a Public Holiday)

The local Public Health Unit were alerted and identified and interviewed ten people who had varying levels of contact with the dead horse. The contact varied from patting the horse to giving an oral drench and kissing the horses head. NSW Health convened an expert panel, including infectious diseases physicians, to review each of the ten cases using their assessment tool. Monoclonal antibody treatment was not used on any of the ten people, but their health was monitored closely for twenty-one days, and all remained well.

A Biosecurity Direction was placed on the property to manage human, and animal movements and twenty-eight in-contact horses and two dogs were subject to health monitoring for twenty-one days before the Biosecurity Direction was revoked. Tracing identified four horses who had moved off the property in the previous sixteen days, but the DVs assessed all of them as low risk and no action required.

This was the first case of Hendra virus in the Hunter Valley. The previous most southern case had been at Kempsey. A research team including bat ecologists visited the area just after the positive confirmation to gather information about flying fox populations and species present in the region around the property. They visited several roosts and identified that small numbers of Black flying foxes were present in some of these roosts and undertook pooled urine sampling of roosts to gain a better understanding of what might have been happening in the flying fox populations during the time of the spillover.

Because of the high horse population in the Hunter Valley, there has been considerable public interest in the case, and an increase in vaccination uptake has been noted, which is encouraging. Because of their strong local networks and good local knowledge the DVs were able to provide local veterinarians and horse owners with accurate and timely information about the case and general aspects about minimising the risk of Hendra exposure which was much appreciated by everyone.

An After Action Review is planned soon, to capture learnings from this case.

This most recent case confirms that outbreaks of Hendra virus, while sporadic and infrequent, will occur wherever flying foxes and in particular Black flying foxes are present. It is a highly fatal infection for horses and people and the importance of taking appropriate biosecurity protection whenever handling horses cannot be overstated.

Vaccination of horses is the most effective way to help manage Hendra virus disease. Vaccination of horses provides a public health and workplace health and safety benefit by reducing the risk of Hendra virus transmission to humans and other susceptible animals.

For further information contact Jim Kerr, District Veterinarian, Hunter Local Land Services on (02) 4938 4900, Jane Bennett, District Veterinarian, Hunter Local Land Services on (02) 4938 4900, or Paul Freeman, Senior Veterinary Policy & Project Officer, NSW DPI Animal Biosecurity, Orange NSW, on (02) 6626 1214.

Canine leishmaniasis in the Greater Sydney region

In June, a case of *Leishmania infantum* was positively diagnosed in a two-year-old intact male Burnese Mountain Dog in Greater Sydney. The dog arrived from Spain four months earlier and tested negative for leishmaniasis prior to departure.

Leishmaniasis is a protozoal disease affecting humans and animals caused by numerous *Leishmania* species and it is transmitted primarily by phlebotomine sandflies. Most *Leishmania* species are zoonotic. After a variable incubation period (up to seven years in some cases), the disease manifests in a range of forms, including cutaneous and visceral syndromes. Leishmaniasis in humans and dogs is widespread throughout Africa, parts of Asia, southern Europe, and in South and Central America.

Clinical manifestations in the affected dog included epistaxis and shifting lameness. Neoplasia was considered the likely differential diagnosis; however, *Leishmania* was considered a possibility, given that the dog was imported from an endemic country for leishmaniasis. Conjunctival swabs and blood samples (EDTA and clotted) were collected and submitted to Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) and confirmed at the Australian Animal Health Laboratory (AAHL) in Geelong. Serology was used to detect anti-leishmanial antibodies through enzyme-linked immunosorbent assay (ELISA) and reinforced with polymerase chain reaction (PCR) to detect parasite DNA within the samples.

Following confirmation, Greater Sydney Local Land Services (LLS) and Department of Primary Industries conducted a joint investigation to determine the likely source of infection and to identify whether other animals may have been exposed. The acquired source of infection, in this case, is most likely before entry into Australia. NSW Health was consulted for assessing the Public Health risk. The zoonotic risk was deemed low, given that a competent vector has not yet been identified in Australia and no Australian phlebotomine species are recognised pests of humans. The risk to animals was considered a medium risk, given that venereal transmission has been documented and the dog was not castrated. As such, it was advised that the dog be desexed to minimise the risk to other dogs.

The owners agreed to undertake measures to reduce the risk to an acceptable level in order to avoid euthanasia. Drug therapy will be employed to reduce parasitaemia, permanent and on-going external parasite control will be used to inhibit bites from potential biological and mechanical insect vectors, and periodic serological monitoring will be implemented.

Previous introductions of exotic *Leishmania* species. into Australia (via infected travellers/immigrants and infected imported dogs) have not resulted in onward transmission. The only known endemic *Leishmania* species in Australia is *L. australiensis*. It was first isolated in 2001 from the skin lesions of a group of captive red kangaroos (Macropus rufus) in the Northern Territory. To date, it has not been associated with disease in humans or domestic animal species.

For further information contact Ofir Schwarzmann, Veterinary Policy & Project Officer, NSW DPI Animal Biosecurity, Orange NSW on (02) 6391 4612.

Survey – How can we improve the NSW Animal Health Surveillance newsletter?

We're seeking your thoughts on the Animal Health Surveillance newsletter. The information and articles for this newsletter are supplied by NSW Department of Primary Industries (DPI) staff and Local Land Services (LLS) District Veterinarians. We'd like to know what works, what doesn't work, and what we can do better. Accordingly, we would appreciate you taking the time to complete this short survey.

This survey can be completed by hand, then scanned and emailed to **ofir.schwarzmann@dpi.nsw.gov.au**

Alternatively, this survey can be completed electronically at this link **https://www.surveymonkey.com/r/SDP9P9K**

1. **Please rate the quality of the information contained in the NSW Animal Health Surveillance newsletter?**
   ❑ Excellent ❑ Good ❑ Okay
   ❑ Needs improvement ❑ Poor

2. **Having information and case studies written by LLS District Veterinarians who conduct disease investigations on-farm is engaging and the best way to present animal health surveillance information.**
   ❑ Strongly agree ❑ Agree ❑ Neutral ❑ Disagree

3. **I like the current layout and look of the Animal Health Surveillance newsletter.**
   ❑ Strongly agree ❑ Agree ❑ Neutral ❑ Disagree

4. **Photos of diseases and animal health issues are important.**
   ❑ Strongly agree ❑ Agree ❑ Neutral ❑ Disagree

5. **I would like to see more animal health statistics and data presented in the Animal Health Surveillance newsletter.**
   ❑ Strongly agree ❑ Agree ❑ Neutral ❑ Disagree

6. **I would prefer the Animal Health Surveillance newsletter to be delivered:**
   ❑ Electronically via email
   ❑ Electronically via the NSW DPI or LLS website
   ❑ As a paper-based hard copy

7. **What is the one thing you find most valuable about the Animal Health Surveillance newsletter?**

8. **If you could change one thing about the Animal Health Surveillance newsletter, what would it be?**

Getting information on animal diseases

This surveillance report can convey only a very limited amount of information about the occurrence and distribution of livestock diseases in New South Wales.

For statewide information, contact the Department of Primary Industries Biosecurity and Food Safety unit on 1800 684 244.

If you would like more specific information about diseases occurring in your part of the state, contact your Local Land Services District Veterinarian or the Department of Primary Industries Senior Veterinary Officer for your region, or go to: *www.lls.nsw.gov.au*

For more information on national disease status, check the National Animal Health Information System (NAHIS) via the internet at: *www.animalhealthaustralia.com.au*

This is a report under the Animal Disease Surveillance Operational Plan, Project 8, 'Reporting for Animal Disease Status in NSW'.

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Copies of NSW Animal Health Surveillance reports are available on the internet at:

www.dpi.nsw.gov.au/about-us/publications/animal-health-surveillance

Information contributed by staff of NSW Department of Primary Industries and Local Land Service

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