NSW DPI

NEW SOUTH WALES

# ANIMAL HEALTH SURVEILLANCE

Information contributed by staff of the Rural Lands Protection Boards and the NSW Department of Primary Industries

July - December 2007 • Numbers 2007/3 and 4

## Equine Influenza

Equine Influenza was first suspected at Eastern Creek Quarantine Station (Sydney NSW) in August 2007. Horses in central Sydney showing symptoms of equine influenza (EI) were reported to the Hotline on 24th August 2007. These horses were stabled at Centennial Park. At approximately 11.00pm on Friday 24th a positive diagnosis for Influenza A antigen was confirmed by virologists at Elizabeth Macarthur Agricultural Institute (EMAI). The First Response Team was placed on standby in the early afternoon of 24th of August. The State Disease Control Head Quarters (SDCHQ) had been established at around 4.30pm on Friday 24th in case this diagnosis was confirmed. Following the positive diagnosis a full-scale emergency animal disease response was activated in accordance with the *AUSVETPLAN Disease strategy for equine influenza*. A 72 hour nationwide horse movement standstill was initiated.

H3 equine influenza virus was later confirmed at both Eastern Creek Quarantine Station and Centennial Park stables by viral sequencing tests, undertaken by the CSIRO Australian Animal Health Laboratory (CSIRO AAHL), Geelong Victoria. Sequence analysis of the Matrix gene undertaken at the CSIRO AAHL confirmed that the Australian EI isolate was almost identical to the Matrix gene sequence of the Wisconsin strain of EI (A/equine/Wisconsin/1/03(H3N8).

In late September a vaccination strategy commenced to help control and eradicate EI. Buffer and ring vaccination zones were used to contain the infection and protect unaffected regions in NSW and QLD. The vaccine used is a canary pox vaccine called ProteqFlu® and ProteqFlu TE®. (ProteqFlu TE® includes tetanus toxoid).

By the end of September, about 25 000 horses had been exposed on more than 2800 infected premises in the State. In the first few weeks of October about 1000 new cases occurred. Spread continued to occur, mainly through close contact and aerosol spread over short distances. However, outbreaks at Forbes, Wellington and Grenfell were attributed to inadvertent human assistance in moving the virus over longer distances. New cases declined substantially from mid-October, to about 50 per week during November, and declined further to fewer than 5 per week during early December. The last new case was reported on 22 December 2007; however this property was probably infected much earlier than this. A total of about 6100 NSW premises were declared as infected during the outbreak.

Extensive surveillance occurred during October and November to confirm that previously infected or suspect infected areas were free from EI. This resulted in zone changes, with much of the amber zone (a buffer area with low risk of infection, located between where infection was and the disease free area) upgraded to green zone status (area free from EI) and outlying red zones (areas previously with active infection) upgraded to amber and green zones. By the end of December, about 35 000 polymerase chain reaction (PCR) tests had been done, of which 3900 were positive, while 15 000 out of 42 000 enzyme-linked immunosorbent assay (ELISA) tests were positive. The high number of ELISA positive tests was due to extensive testing of known infected properties to demonstrate that all horses had become infected and were immune.

## *In this issue!*



Rural Lands Protection Boards

NSW DEPARTMENT OF PRIMARY INDUSTRIES

Critical components of the response strategy included:

- Immediate implementation of a complete standstill on horse movements following confirmation of the first case. This allowed restriction of infection to northern and eastern New South Wales and southern Queensland. If this standstill had been delayed, infection would have been much more widespread and possibly have extended into other States.
- Implementation of a State-wide zoning strategy to allow the management of movements and risk in different parts of the State. Zoning was important in allowing some movements to continue in areas where this was appropriate, while prohibiting or controlling movements in other areas, fostering cooperation from the horse industry.
- Introduction of vaccination as a means of control.
- Ongoing support of the various horse industry sectors for the program and their compliance with movement and biosecurity requirements.

## QUARTERLY HIGHLIGHTS

### JULY TO SEPTEMBER 2007

### Swill feeding workshops

Throughout the state, stock inspectors from RLPBs and DPI attended workshops on swill feeding. Swill feeding is the traditional name for the feeding of food scraps and other waste material to pigs. This practice has caused foot-and-mouth disease outbreaks overseas, including the catastrophic epidemic in the United Kingdom in 2001. Swill feeding, which was common in Australia in the 1950s, is now banned in all states, including New South Wales. Some food wastes have been categorised as 'prohibited substances', and it is against the law to feed them to pigs. Meat, meat products, or anything that has been in contact with meat cannot be fed to pigs.

The workshops provided inspectors with an overview of prohibited feed substances and reminded them of their responsibilities in ensuring compliance with the feed ban by pig owners. Further information on swill feeding is contained in the Primefact 'Swill feeding', available at: http://www.dpi.nsw.gov.au/agriculture/livestock/pigs/health/swill-feeding

### Photosensitisation in Hereford cows on a wheat crop

Hereford cows in a herd of Angus and Hereford cattle in the Walgett RLPB district developed photosensitisation after grazing a new wheat crop.

Wheat plants are a recognised, although rare, cause of photosensitisation. An unknown toxin in the wheat plant causes damage to the liver. General liver damage results in interference with excretion of phylloerythrin into bile. Phylloerythrin is the product of microbial metabolism of chlorophyll, the green photosynthesising pigment in plants. This interference to phylloerythrin excretion can occur in the absence of clinically-detectable jaundice. Phylloerythrin then enters the general blood circulation and travels to capillary walls in the skin and on exposure to sunlight (not just UV), the phylloerythrin creates free radicles which initiate inflammation. Unpigmented skin with a sparse hair coat is the prime site for lesions. The underside of the tongue is also commonly affected in cattle as they lick their noses and expose this area to sunlight.

*NSW DPI would like to acknowledge the assistance of Richard Hernando, former DV Walgett and advice from Dr Ross MacKenzie in the investigation of this case.*

*Hereford cow with photosensitisation evident on the nasal plane. Photo by Bruce Kirk, AQIS*

*Hereford cow with photosensitisation evident on the teats. Photo by Bruce Kirk, AQIS*

## Photosensitisation in cattle on pasture

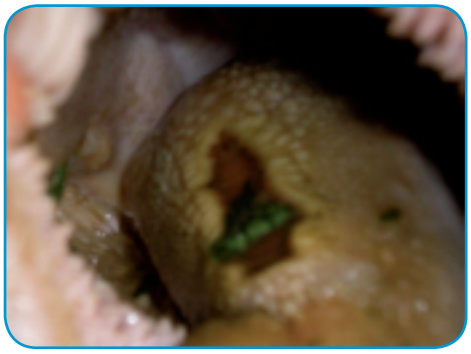
The owner of a specialist pasture based steer fattening enterprise in the Wagga Wagga RLPB observed his cattle staying in tree lines. The steers were in a paddock of improved pasture that was growing vigorously and had sustained high weight gains. The owner became concerned when he noticed lameness in the cattle and lesions around their eyes and nose. Approximately 40 out of 240 steers were affected.

DV Wagga examined the cattle and noticed lesions were only present on white-haired and bare skin parts of the animal, with Herefords predominantly affected. This led him to suspect the steers were suffering from photosensitisation. A blood sample was collected from an affected steer. Infectious bovine rhinotracheitis was excluded on serology. Liver enzymes were within the normal range, suggesting that a primary photosensitisation had occurred. The cause of primary photosensitisation on pasture remains unknown.

**For further information contact Tony Morton, DV Wagga Wagga RLPB on (02) 6923 0900.**

## Oral lesions in sheep

Weight loss and difficulty chewing and swallowing was noticed in 30 out of 300 Poll Dorset sheep on a property in the Narrandera RLPB district. Ulcerative lesions varying in size from 8mm$^2$ to 40 x 12 mm were seen on the dorsal surface of the tongues of affected sheep. Initially it was suspected that the diet, which included wheaten hay with large awns, had caused mechanical damage to the sheep's tongues; however, a mob of merino ewes fed the same hay were not affected. Additionally, the hay had been fed for several months, but hay feeding had ceased seven weeks prior to the investigation and the affected sheep mob was fed pellets and put onto a Yarran oat crop, making it less likely that the hay was the cause. A blood sample from one affected sheep tested positive for malignant catarrhal fever. Sheep are normally carriers of Ovine herpesvirus-2 (OHV2), and it is unusual to see clinical signs in sheep infected with OHV2. Further investigation confirmed that this was a coincidental finding: the farmer examined the leaves of the Yarran oats and found that the edges had very sharp serrations. It is most likely that the awned wheat hay started the damage and then the serrated edges of the green oats prevented the tongue lesions from healing.



*Sheep with a large ulcerative lesion on the base of the tongue. Photo by Gabe Morrice.*

**For further information contact Gabe Morrice, DV Narrandera RLPB on (02) 6959 2322.**

## Hepatic encephalopathy in goats

A flock of 200 Angora goats in the Murray RLPB had a 12 month history of emaciation and weakness with progression to death, affecting approximately 20% of the flock. All age groups from 6 months to 6 years old were affected. Affected animals were weak and their mucous membranes were pale and slightly jaundiced.

Worm counts and coccidial counts ruled out internal parasites as the cause.

A post mortem examination was performed on two of the older affected goats. Jaundice and a fibrotic liver were noticed grossly in one animal and a smaller than normal liver was noticed grossly in the other animal. Organs, including the liver and the brain, were submitted to Menangle RVL for histopathology and TSE exclusion.

Hepatic cirrhosis and hepatic encephalopathy was diagnosed on histopathology.

**For further information contact Harry Suddes, DV Murray on (03) 5886 1203.**

## Equine herpesvirus-1

From mid July to late August 2007 a total of eleven histopathologically confirmed abortions due to EHV-1, and one histologically confirmed perinatal death due to EHV-1, that occurred on a number of thoroughbred studs in the Hunter Valley NSW, were reported. The introduction of EHV1-carrier mares and increased movement of mares in late gestation to stud is suspected to have led to the high incidence of EHV1 abortions in the Hunter region this foaling season.

Exposure to EHV-1 is very common in the horse population. A study in the Hunter Valley in 1995 showed that approximately 20 to 30% of all mares on a large thoroughbred stud had been exposed to EHV1 and more than 99% of mares had been exposed to equine herpesvirus type 4 (EHV4)[1].

A recent study in Kentucky USA found that 54% of thoroughbred broodmares had been exposed to EHV1[2].

Once infected with EHV-1 the horse will usually be latently infected for life. The virus lies dormant in lymph nodes or nervous system tissue and if the horse is stressed the virus can be reactivated. If a pregnant mare becomes infected with EHV-1, or if she is latently infected with EHV-1 and is stressed while pregnant so EHV-1 is reactivated, the virus can cross the placenta and cause the foal to be aborted. Sometimes an infected full term foal may be born alive but it will usually die within a few days of birth.

When a mare aborts due to EHV-1 she may have reactivated a previously latent infection, or another horse that she has been in contact with may be the source of infection. Since EHV-1 is very common in the horse population, mare owners should treat all pregnant mares as if they are at risk of EHV-1 abortion. Avoiding stress in pregnant mares is most important in preventing abortion due to EHV-1. Pregnant mares should be kept in their established group and their social structure should not be disturbed by introducing a new horse to the group or moving a pregnant mare to a new group of horses. Mares in late gestation (within 2 months of foaling) should not be transported.

The Australian Equine Veterinarians Association guide to the management of EHV-1 abortion contains sound recommendations and should be referred to

1 Gilkerson J.R et al (1999) Epidemiology of EHV-1 and EHV-4 in the mare and foal populations on a Hunter Valley stud farm: are mares the source of EHV-1 for unweaned foals *Veterinary Microbiology* 68: 27-34.

2 Allen GP et al (2008) Prevalence of latent, neuropathogenic equine herpesvirus-1 in the Thoroughbred broodmare population of central Kentucky. *Equine Vet J* 40(2): 105-110.

for more information on the prevention and management of EHV-1 abortion.

EHV-1 abortion is a notifiable disease in NSW. Any case of abortion where EHV-1 is diagnosed as the cause must be reported to an inspector under the Stock Diseases Act (i.e. District Veterinarian or a Ranger employed by the Rural Lands Protection Board or a NSW Department of Primary Industries veterinarian).

**For further information contact Sarah Robinson, NSW DPI on (02) 6938 1967.**

## Regional surveillance project: cattle fertility

Farmers in the NSW Southern Slopes region were urged by their district veterinarian to participate in a collaborative project aimed at determining the prevalence of pestivirus, leptospirosis and vibriosis in beef cattle in the region. All three diseases have potential to impact on reproductive performance. Up-to-date knowledge on the prevalence of these diseases is currently not available, so a project was designed find out the prevalence of these diseases and to develop advisory material and identify opportunities for future research.

Testing of cattle from a total of 75 randomly selected herds in the Riverina, Murray, Hume, Wagga Wagga, Gundagai, Cooma, Bombala, Braidwood, South Coast, Hay, and Narrandera Rural Lands Protection Boards was carried out during the quarter. Laboratory testing fees are being funded by NSW DPI, Mc Garvie Smith Institute, Schering Plough and Pfizer.

The results will be analysed and published in 2008. Combining resources across such a vast area will provide producers in this region with invaluable information to improve cattle fertility.

**For further information contact Sarah Robinson, NSW DPI on (02) 6938 1967.**

## BJD Testing

Kempsey RLPB is currently a BJD protected zone. The impending introduction of the new Dairy Assurance Score (DAS) provided an impetus for non assessed dairy herds in Kempsey RLPB to utilise Cattle Council funding to undertake a check test. The results to date are very encouraging with 14 dairy herds completing check tests and only a single ELISA positive animal out of the 700 animals tested. These results support the belief of local veterinarians that the true prevalence of BJD in the dairy herds in the BJD protected zones is low and nothing like the 20% herd prevalence seen in the control zones. If further testing supports this contention then an education campaign focusing on the biosecurity needed to keep BJD out of dairy herds will be very important.

In a northern NSW Rural Lands Protection Board area two CattleMAP beef herds were confirmed as infected during maintenance testing. In one herd the infected animal was a 10 year old cow introduced as a 2 year old. This cow was ELISA negative on two previous tests and clinically normal when confirmed as infected. The other cattle MAP herd had an infected homebred 3 year old heifer with clinical signs of BJD. This herd had two previous negative herd tests in 2002 and 2004.

Routine maintenance testing in a CattleMAP (MN3) beef herd in southern NSW revealed four positive animals on serological testing. Follow up testing of these reactors has been done and two animals were positive on faecal culture and PCR tests. The two BJD infected animals were two out of two MN3 dairy-cross house cows belonging to the neighbour of the owner of the MN3 beef herd. The two seropositive animals from the MN3 beef herd were retested with two faecal cultures 4 months apart and were negative for BJD.

The two BJD infected cows were twins whose dam was a purchased Friesian cow. The Friesian cow's previous progeny had all been slaughtered. The Friesian cow was found dead the morning after giving birth to the twins. The twin calves were reared on milk replacer and it is unknown if they ever suckled the Friesian cow. It is presumed that the Freisian cow was the source of infection for her twin progeny. The dairy cross cows were MAP tested with the neighbouring beef herd, as a bull from the beef herd is lent to join the house cows.

A detailed risk assessment was conducted for the MN3 beef herd, with every situation of potential exposure of the beef herd to BJD infection from the small neighbouring herd assessed. The beef herd was found not to be at risk of BJD infection and retained CattleMAP MN3 status.

## Salmonella infection in lambs results in heavy losses

Approximately 150 out of 588 lambs (25%) died due to *Salmonella typhimurium* enteritis on a Wagga RLPB property. The sheep left Tasmania nine days prior to the first mortalities and had been spelled for four days before being sold

through two NSW saleyards prior to arriving at the Wagga Wagga property. Transport stress and repeated handling through yards (without access to feed) is likely to have been the trigger for the salmonella infection. The heavy losses in this mob highlight how important it is to minimise stress and provide adequate food and water to stock during transport.

### *Campylobacter* abortions in ewes

*Campylobacter* caused an abortion storm on two properties in the Hume RLPB. On one property two mobs of 400 mixed age cross bred ewes had been kept in an intensive feedlot situation (up until 4 weeks before lambing in the case of mob A and 7 weeks before lambing in the case of mob B). In mob A approximately 65 out of 100 lambs born during the first week of lambing were either aborted late term or stillborn. At this point the ewes were moved to another paddock and the practice of promptly disposing of all dead lambs was instituted. Although the rate of lamb loss subsided over the following weeks, the final lamb loss figure in the mob was approximately 25%. Ewes in mob B were vaccinated with 'Ovilis' vaccine about 2 to 5 weeks prior to lambing. Approximately 10 ewes aborted just prior to and during the first week of lambing but final losses were only of the order of 20 out of 400 in this mob (5%).

Characteristic autopsy findings in the aborted or still born lambs included dropsical abdominal swelling (about 50% of affected lambs showed this finding); enlarged livers; and necrotic hepatic foci. A pure growth of *Campylobacter fetus* subsp. *fetus* cultured from the affected lambs confirmed the diagnosis.

On the second property a mob of 320 maiden Merino ewes that had also been run under lot feeding conditions suffered a similar fate. Approximately 100 out of 320 ewes aborted or had still born lambs. *C. fetus* subsp. *fetus* was also isolated from these affected lambs.

**For further information contact Steve Whittaker, DV Hume RLPB on (02) 6040 4210.**

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## QUARTERLY HIGHLIGHTS

## OCTOBER TO DECEMBER 2007

### Equine Fescue Oedema

During the period late September to early December 2007 a new toxicosis occurred in horses in Australia, a toxicosis which has not been reported before in any country.

Dr Chris Bourke, Principal Research Scientist at Orange Agricultural Institute, will shortly publish a report of this new toxicosis that he identified in horses.

Farms with affected horses were located at Scone, Cootamundra, and Albury in NSW and at Kangarilla in South Australia. The Cootamundra farm had seen the same syndrome, for the first time, in some of its horses in the spring of 2006. Affected horses on all farms were grazing mixed pastures but the common factor was a particular type of perennial tall fescue grass (*Festuca arundinacea*), known as a Mediterranean, winter active, tall fescue type. Until about 2005 almost all tall fescue pastures were established using the Continental, summer active, tall fescue type. The Mediterranean varieties that are now in commercial use are called Fraydo, Flecha, Origin, Prosper, and Resolute. All other commercial fescue varieties belong to the Continental group.

The fescue content of toxic pastures varied between < 10% up to 95%. In pastures of lower fescue content there was a preference for grazing fescue because it was green and leafy when other pasture grasses (eg ryegrass) had started to hay off. The risk period was late September to early December, ie full leaf to early seed head development stage. All toxic pastures involved endophyte infected fescue and preliminary investigations would suggest that this is a *Neotyphodium coenophialum* endophyte. Mediterranean-type winter active fescues are not the natural host for *Neotyphodium coenophialum;* consequently it is feared that the combination of this particular endophyte and a Mediterranean-type fescue may have resulted in the production of a new toxin to which horses are particularly susceptible. The four outbreaks involved two different fescue varieties. On two of these farms the seed purchased for sowing had been presumed to be endophyte free, but this turned out not to be the case. At this stage all Mediterranean fescue varieties sold as endophyte containing, together with all of those sold without a certified nil endophyte status, should be regarded as potentially toxic to horses. This includes Mediterranean-type fescue seed that has been incorporated into one of the many special pasture blend mixes that are offered for sale to horse owners.

The majority of affected horses have been brood mares in pasture paddocks with foals at foot. Out of the 49 adult horses that grazed toxic pastures on the four farms 41 became intoxicated and 2 subsequently died. On some farms affected suckling foals were also reported. Clinical cases first appear about 3 to 6 days after horses start grazing the pasture. If they are immediately moved off then most affected animals will return to normal within another 3 to 7 days, but some may require supportive therapy in the form of plasma administration, and some may die despite this treatment. Some affected horses may take up to 28 days to recover.

Affected horses become lethargic, depressed, and inappetant and are inclined to be recumbent. Most develop mild to moderate swelling of the head and neck as a result of gravity-dependent subcutaneous oedema. Concurrently they develop oedematous thickening of the wall of the right dorsal colon. This is visible on ultrasound scan images of the abdomen. They go on to develop oedema of the chest and belly (ventral oedema), including accumulation of fluid within the abdominal cavity. Whereas the head and neck oedema quickly resolves, the ventral oedema can persist for several weeks. Protracted weight loss and associated anoestrous are risks for affected lactating mares. Secondary inflammatory changes such as peritonitis are possible in some animals.

The outstanding and consistent finding is a drop in total plasma proteins, with values as low as 27 g/L (normal range 52-72 g/L). This is due to an albumin decline. The PCV is elevated, eg 56 to 70 (normal range 36-48). There is a variable drop in calcium, sodium and chloride values and a variable elevation in the white blood cell count and fibrinogen value. If the albumin drop is too severe the horse will die before plasma administration can restore it. During the recovery phase plasma proteins may not return to normal for several weeks. In some affected horses these biochemical and haematological changes can develop quite suddenly and return to normal quite quickly once they are moved off the pasture.

The most likely pathogenesis for this toxicosis is a primary vasculopathy resulting in effusion and oedema. In the horse this type of injury is characteristically expressed in the colon and the subcutis. In some affected mares it also appears to have been expressed in the reproductive tract.

This toxicosis shares some clinical features in common with phenylbutazone poisoning, equine-type arsenic poisoning, Oak poisoning,

Walnut poisoning, Yellow Wood poisoning (nephrosis form) and African Horse Sickness.

Report written by Dr Chris Bourke, Principal Research Scientist (Poisonous Plants) and Specialist Veterinary Toxicologist, Orange Agricultural Institute, Forest Rd Orange NSW 2800.

**For further information contact Chris Bourke on (02) 6391 3867.**

## Anthrax

Anthrax was diagnosed on 14 properties during November and December 2007 (some near Scone occurred in early Jan 2008). Three of these were sporadic incidents in the usual anthrax-endemic districts of Condobolin (two properties) and Forbes (one property). In Condobolin in November, one case involved the death of 38 out of 620 sheep, and a second case involved the death of 2 out of a herd of 94 cattle. The third property, in the Forbes district, lost 3 out of 220 cattle in December.

In late December, an incident occurred in the Hunter district. This involved 11 properties and the loss of 54 cattle and 1 horse by the end of the incident. These cases were outside the usual anthrax-endemic area, but there were anecdotal reports of anthrax occurring in the 1940s and documented reports in the 1870s. It is thought that heavy rains and flooding in the area in the middle of the year may have exposed anthrax spores.

All cases have been managed according to the New South Wales Department of Primary Industries anthrax policy. Properties were placed in quarantine, carcases burnt and sites disinfected, and all at-risk stock were vaccinated. In the case of the Hunter incident, there was also widespread vaccination on neighbouring properties. Several exposed personnel were advised to seek medical attention, but no cases of human infection were confirmed on culture.

**For further information contact Barbara Moloney, NSW DPI on (02) 6391 3687.**

## *Nosema ceranae* in bees

*Nosema ceranae* has been detected in adult bee samples from beekeepers based in Queensland, NSW and Victoria. *N. ceranae* is a newly recognised pathogen of honeybees in Australia and the full effects of infection have not been determined in this country. However, overseas it has caused gradual depopulation, higher autumn–winter colony deaths and low honey production. It is considered to be more pathogenic than the endemic *N. apis*, which has been long recognised worldwide.

The Rural Industries Research and Development Corporation has funded the Elizabeth Macarthur Agricultural Institute to determine the extent of infection in beehives in Australia. To date, beekeepers have submitted samples as follows: New South Wales 44 samples (3 positive), Queensland 45 samples (27 positive) and Victoria 44 samples (2 positive). *N. ceranae* has not been detected in 24 samples from Western Australia, 19 samples from Tasmania and 8 samples from South Australia. The survey is due to be completed by June 2008.

**For further information contact Michael Hornitzky, NSW DPI on (02) 4640 6311.**

## Botulism in cattle

A herd of Angus cattle on extensive rangeland (native grasses, forbs and shrubs) along the Murrumbidgee River in the Riverina RLPB district were affected by a syndrome characterised by limb weakness, flaccid paralysis and death within 3 days.

One paddock had a history of occasional cattle deaths without a cause being identified, and the owner had earlier in the year placed phosphorus blocks in the paddock. In October 2007, 15 of 200 adult cows in this paddock and 5 of 200 heifers in an adjoining paddock were affected. Affected animals were in sternal recumbency and alert, and most had kinked necks. With encouragement, they would attempt to stand but appeared too weak. One attempted to charge. There was variable flaccid paralysis of the tongue; some had reasonable muscle tone but others were completely flaccid. Temperatures were within the normal range. Necropsy showed no gross or microscopic abnormalities. Serum phosphorus was within the normal range. No *Clostridium botulinum* toxin was detected from serum, liver or ruminal contents. Botulism was

*Affected animal in sternal recumbency. Photo taken by Dan Salmon*

*laccid paralysis of the tongue of an affected animal. Photo taken by Dan Salmon*

*An unusual number of flies were observed on affected animals. This may be due to loss of the ability to twitch their tail or skin. Photo taken by Dan Salmon*

diagnosed on the basis of the history and the flaccid paralysis. The entire herd was vaccinated against *C. botulinum* types C and D toxin. Two more animals were affected in the week following the initial vaccination, but no more cases were seen in the month after that.

**For further information contact Dan Salmon, DV Riverina RLPB on (03) 5881 1055.**

## Calf scours and mortalities

High rates of calf mortality associated with severe calf scours occurred in the Hume Rural Lands Protection Board. In five cases, losses ranged from 7% to 30%. Heaviest losses were in calves less than 7 days old but deaths were also common in calves as old as 2–3 weeks. Pathogenic *Escherichia coli* was diagnosed as the most likely cause in 2–3-day-old cases, but rotavirus and cryptosporidia were also isolated from affected calves over 1 week old. Stress due to unseasonably hot weather appears to have been the initiating trigger, and a very heavy fly level appears to have been the transmission factor. Early fluid intervention was helpful in most outbreaks, but antimicrobial therapies were of little value.

**For further information contact Steve Whittaker, DV Hume RLPB on (02) 6040 4210.**

## Possible haemonchosis in grazing camels

In December 2007, camels in the north-east of the State had difficulty gaining weight, and showed recurrent diarrhoea and oedema of the pedestal and limbs, as well as dirt eating, difficulty rising from a crouched position and pale mucous membranes. Skin problems were common, and there was a history of swollen heads similar to bighead in horses, and shifting leg lameness.

Faecal eggs counts for four camels were 400, 1600, 2360 and 6420 eggs per gram, and a larval differentiation on pooled samples revealed 42% *Haemonchus* and 58% *Trichostrongylus*. No stomach or liver fluke eggs were seen. Fenbendazole and ivermectin anthelmintics were used for internal parasite control, with ivermectin greatly reducing faecal egg counts.

All four camels were anaemic, with packed cell volumes of 8, 14, 14 and 25%. *Trypanosoma evansi* was excluded as a cause of the anaemia. There was a concurrent copper poisoning due to excessive supplementation with copper sulfate.

**For further information contact Buster Neilson, DV Tweed-Lismore RLPB on (02) 6621 2317.**

## Johne's disease

The number of cattle herds in NSW infected with Bovine Johne's disease (BJD) as at 30 September 2007 is shown in Table 1.

The number of flocks or herds enrolled in Johne's Disease Market Assurance Programs (JD MAP) as at 30 September 2007 is shown in Table 2.

***Table 1: Bovine Johne's disease (BJD)-infected herds in NSW as at 30 September 2007***

| RLPB | Dairy | Beef | Total |
|---|---|---|---|
| Casino | 19 | 13 | 32 |
| Grafton | 1 | 0 | 1 |
| Gundagai | 0 | 1 | 1 |
| Hume | 5 | 2 | 7 |
| Kempsey | 4 | 0 | 4 |
| Maitland | 2 | 1 | 3 |
| Molong | 0 | 1 | 1 |
| Moss Vale | 2 | 0 | 2 |
| Murray | 13 | 1 | 14 |
| Riverina | 7 | 1 | 8 |
| South Coast | 13 | 0 | 13 |
| Tweed Lismore | 16 | 9 | 25 |
| **Total** | **82** | **29** | **111** |

***Table 2: Flocks and herds enrolled in JD MAP as at 30 September 2007***

| | Herd type | Results of status testing at each stage (number of herds) | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | MN1 | MN1-V | MN2 | MN2-V | MN3 | MN3-V | TOTAL |
| **AlpacaMAP** | Stud | 4 | | 23 | | 80 | | 107 |
| | Other | 1 | | 0 | | 7 | | 8 |
| | **Total** | **5** | | **22** | | **89** | | **115** |
| **CattleMAP** | Stud | 52 | | 118 | | 207 | | 377 |
| | Other | 44 | | 44 | | 37 | | 125 |
| | **Total** | **126** | | **191** | | **255** | | **502** |
| **GoatMAP** | Stud | 7 | | 17 | | 4 | | 28 |
| | Other | 2 | | 1 | | 1 | | 4 |
| | **Total** | **12** | | **17** | | **6** | | **32** |
| **SheepMAP** | Stud | 9 | 23 | 21 | 30 | 136 | 38 | 257 |
| | Other | 11 | 10 | 10 | 3 | 33 | 1 | 68 |
| | **Total** | **20** | **33** | **31** | **33** | **169** | **39** | **325** |

MNn = Monitored negative (n = minimum number times sample tested with negative result)
MNn-V = Flocks being vaccinated against OJD (sheep only)

## Getting Information on Animal Diseases

This surveillance report can convey only a very limited amount of information about the occurence and distribution of livestock diseases in New South Wales. If you would like more specific information about diseases occurring in your part of the State, contact your local Rural Lands Protection Board District Veterinarian, Departmental Senior Regional Animal Health Manager, Regional Health Leader, or Regional Veterinary Laboratory.

***For Statewide information, contact NSW DPI's Animal and Plant Biosecurity Branch in Orange on (02) 6391 3237 or fax (02) 6361 9976.***

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

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***Copies of NSW Animal Health Surveillance reports are available on the internet at:***
***http://www.dpi.nsw.gov.au/reader/ah-surveillance***

### Disclaimer



NSW DEPARTMENT OF
PRIMARY INDUSTRIES

Rural Lands Protection Boards

## Transmissible spongiform encephalopathy (TSE) surveillance

TSE surveillance continued during the quarter. All samples were TSE negative.

The Equine Influenza outbreak has significantly impacted on the number of TSE submissions from District veterinarians, and the number of submissions received from private Veterinarians has not declined. Continuing submissions to the National Transmissible Spongiform Encephalopathy Surveillance Program are encouraged. Cattle over 30 months and less than 9 years and sheep over 18 months which exhibit neurological symptoms are eligible for sampling under the program. Submission numbers are given in Tables 3 & 4.

**For further information on TSE or BJD contact Sally Spence, NSW DPI, on (02) 6391 3630.**

## Enzootic bovine leucosis (EBL)

Table 5 and 6 below shows the EBL status of NSW dairy herds as at the end of July 2007 and December 2007 respectively.

**For further information contact Richard Zelski, NSW DPI, on (02) 4939 8959.**

***Table 3 TSE surveillance notifications by RLPB 1.7.2007 to 30.9.2007***

| RLPB | DV sheep | DV cattle | Abattoir sheep | Abattoir Cattle | Private vet sheep | Private vet cattle | Total sheep | Total cattle |
|---|---|---|---|---|---|---|---|---|
| Cooma | 1 | | | | | | 1 | |
| Grafton | 1 | | | | | | 1 | |
| Gundagai | | 2 | | | | | | 2 |
| Hume | | | | | | 4 | | 4 |
| Kempsey | | 1 | | | | | | 1 |
| Moss Vale | | | | | | 2 | | 2 |
| Mudgee-Merriwa | | 1 | | | 1 | | 1 | 1 |
| Murray | 1 | | | | | | 1 | |
| Northern New England | | 2 | | | | | | 2 |
| South Coast | | | | | 1 | | 1 | |
| Tamworth | | 3 | | | | | | 3 |
| Wagga Wagga | 3 | | | | | | 3 | |
| **Total** | **6** | **9** | **0** | **0** | **2** | **6** | **8** | **15** |

***Table 4 TSE surveillance notifications by RLPB 1.10.2007 to 31.12.2007***

| RLPB | DV sheep | DV cattle | Abattoir sheep | Abattoir Cattle | Private vet sheep | Private vet cattle | Total sheep | Total cattle |
|---|---|---|---|---|---|---|---|---|
| Armidale | | 2 | | | | | | 2 |
| Bourke | 1 | | | | | | 1 | |
| Braidwood | | 1 | | | | | | 1 |
| Central Tablelands | 1 | | | | | | 1 | |
| Cooma | 1 | | | | | | 1 | |
| Gundagai | 1 | | | | | | 1 | |
| Hume | | | | | | 2 | | 2 |
| Maitland | | 1 | | | | | | 1 |
| Moree | | | | | | 1 | | 1 |
| Moss Vale | | | | | | 1 | | 1 |
| Mudgee-Merriwa | 1 | | | | | | 1 | |
| Murray | 1 | | | | | | 1 | |
| Narrandera | | 1 | | | | | | 1 |
| Northern Slopes | 1 | 1 | | | | | 1 | 1 |
| Riverina | 2 | 2 | | | | | 2 | 2 |
| Tamworth | | | | | | 1 | | 1 |
| Wagga Wagga | 2 | | | | 1 | | 3 | |
| **Total** | **11** | **8** | **0** | **0** | **1** | **5** | **12** | **13** |

***Table 5 EBL status of NSW dairy herds as at the end of July 2007***

| Status | Number Herds | Percent |
|---|---|---|
| Monitored free | 838 | 92.9 |
| BMT negative | 27 | 3.0 |
| Not assessed | 37 | 4.1 |
| **Total** | **902** | **100.0** |

***Table 6 EBL status of NSW dairy herds as at the end of December 2007***

| Status | Number Herds | Percent |
|---|---|---|
| Monitored free | 821 | 91.7 |
| BMT negative | 39 | 4.4 |
| Not assessed | 35 | 3.9 |
| **Total** | **895** | **100.0** |



DPI JN 8725