

NEW SOUTH WALES

# ANIMAL HEALTH SURVEILLANCE

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## Note on congenital hypotrichosis

Although the case reported in *Animal Health Surveillance Newsletter* 2014, Issue 3, titled ‘Congenital hypotrichosis in White Suffolk sheep’, did indeed involve that breed, note that this condition has been reported historically in other breeds. The Australian White Suffolk Association has taken a proactive role in testing for the gene within their flocks and identifying its prevalence. It is moving towards eliminating it from the breed.

## Surveillance statistics

Animal disease surveillance in New South Wales comprises both active field and laboratory investigations and the examination of passive data from non-government sources.

During the quarter, NSW Government vets targeted about 600 significant disease incidents for investigation to detect emergency diseases as early as possible and to provide assurance of disease status and prevalence on a district basis.

About 40 government vets, who are located on a geographical and enterprise risk basis around the state, defined the disease status of their districts by conducting on-property disease investigations, visiting and inspecting high-risk enterprises, meeting with intensive industry vets, collaborating with private vets

and analysing data generated from laboratory reports.

They targeted syndromes characterised by sudden death, high death rates, high sickness rates or signs that raised suspicion of a notifiable or emergency disease.

The cases reported later in this report illustrate the range of issues that the government vets address on a district basis.

During the same period, the NSW Government State Veterinary Diagnostic Laboratory processed samples from about 850 disease incidents. Of these, 629 (75%) were targeted by the government for laboratory testing to exclude exotic, emergency or otherwise notifiable diseases. The most common tests were for avian influenza, bluetongue, bovine

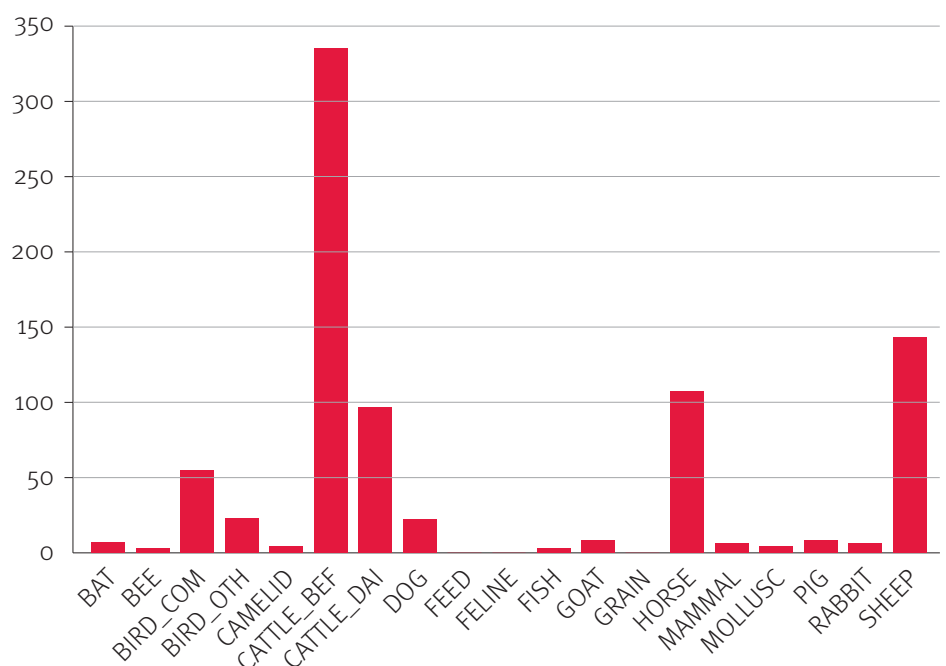
brucellosis, Hendra virus, lyssavirus, Johnes’s disease, Newcastle disease, pigeon paramyxovirus, transmissible spongiform encephalopathy (TSE), rabies and tick fever. No emergency animal diseases were detected.

The most common syndromes that were examined at the laboratory were weakness/anorexia/depression and malaise (14%), sudden death (7%), nervous signs (10%), ill thrift and weight loss (10%), animals being found dead (6%) and abortion/still birth (6%).

As well as the ‘traditional’ domestic livestock classes, a number of other species and samples were tested (see Table 1).

For further information contact Rory Arthur, Manager Animal Disease Surveillance and Research, NSW DPI, Orange, on (02) 6391 3608.

Chart 1 NSW State Veterinary Diagnostic Laboratory samples, by class of livestock, species or feedstuff, October–December 2014.





## Cattle deaths from salt poisoning

In November 2014, a cattle producer and their private vet called the district vet in southern central New South Wales to investigate a case of sudden death on a property east of Jerilderie.

Seven days earlier, 75 mixed-sex, 12- to 30-month-old crossbred cattle had been moved from a 400-ha paddock of mixed mature annual grasses and lucerne to an adjacent paddock of similar grasses without lucerne. The cattle were checked 1 day after being moved to ensure they had found water. When they were next checked 6 days later, several cattle were dead and all of the live animals appeared clinically ill.

One heifer that had remained in the previous paddock (she had been lame when the mob was being moved) appeared healthy. Both paddocks carried about 1000 young, dry ewes that did not appear affected.

All cattle in the affected mob had lost a substantial amount of condition after being prime a week earlier. They were hollow in the left flank and depressed. Most animals had diarrhoea or dysentery and were seeking shade. Several had an overstepping gait when driven, and two were aggressive.

Necropsies were mostly unremarkable apart from varying degrees of enteritis. Handling facilities were not available in the affected paddock, but one dying animal was examined. It had a high rectal temperature—above 42°C.

The district vet considered salmonellosis, clostridial enteritis, coccidiosis, flood plain staggers, mycotoxicosis, blue-green algae poisoning, lead poisoning, arsenic poisoning and nitrate poisoning as differential diagnoses, but each of these was eventually excluded on clinical grounds and from laboratory testing. The cattle that were well enough were moved back to the original paddock, where they recovered over the next 2 weeks. A total of 18 cattle died; only one death occurred after the animals had been moved to the original paddock. One severely affected animal that could not be driven to the original paddock for more than a week was much slower to recover. Both mobs of sheep were driven about 5 km to new paddocks with no signs of illness.

Further analysis of the case revealed that brackish drinking water might have been the cause, and a water sample from the dam was tested. The electrical conductivity was measured at 32 000 µS/cm; sodium and chloride were the most significant elements. Industry water standards class electrical conductivities above 7800 µS/cm as risky to beef cattle, and those above 30 000 µS/cm are considered toxic to all classes of domestic livestock. All of the signs observed in the cattle were consistent with hypernatremia (salt toxicity), and this was the final diagnosis.

The dam in question had been filled from a bore 18 months previously. The other dams on the property had been



Cattle affected by salt poisoning.  
Photo S. Ison

filled more recently from a surface water irrigation channel.

The case provided a diagnostic challenge owing to the variety and pattern of the signs observed and the infrequency of saltwater poisoning in the area. The sheep were probably not clinically affected, because the salinity level of the dam would have been safe when they were introduced to it and they would have become accustomed to the salinity as it slowly increased as the dam water evaporated. Reproductive losses have been associated with salt poisoning in other cases, but the ewes were not pregnant or lactating at the time so effects were unlikely to have been observable.

The owner changed management procedures on the property to prevent reoccurrence of the problem.

For further information, contact Scott Ison, District Veterinarian, Murray Local Land Services, on (02) 6040 4210.

## *Macracanthorhynchus hirudinaceus* in feral pigs

This case, although not of major significance in itself, highlights the NSW Government's commitment to wildlife disease surveillance. It also reminds district vets of a parasite they may not have seen since their undergraduate days.

Two mature feral pigs killed in August 2014 as part of a pest-control program north-east of Bathurst were observed at necropsy to have a heavy infestation of 'tape worms'. A worm collected and submitted to the district vet by the property owner was subsequently identified as an acanthocephalan parasite with a long name, *Macracanthorhynchus hirudinaceus* (the giant thorny-headed worm).

Females of this parasite produce about 260 000 highly environmentally resistant eggs per day. The eggs, which can live for several years, hatch when ingested by the larvae of scarabs and other species of beetles. The worm larvae eventually turn into cysts in the grubs and mature beetles and then infect pigs consuming them. Although most pigs are only lightly infected, the parasite inserts its proboscis into the intestinal wall, causing weight loss and, in some cases, death through intestinal perforation. In one instance, the parasite population caused such a high death rate in nuisance feral pigs (in Iranian sugarcane fields) that it was considered to be a biological control agent.

Although a parasite that can cause emaciation and sometimes death in feral pigs is not of major concern, *M. hirudinaceus* could also infect free-range pigs and is a potential zoonosis. The worm seldom matures in humans, but there are numerous reports of its being found in them. In some countries, people consume beetle larvae for medicinal or dietary purposes, exposing them to the risk of infection.

For further information contact Bruce Watt, District Veterinarian and team Leader, Central Tablelands Local Land Services, on 02 6331 6915.

# Acute common heliotrope poisoning in lambs

A district vet was contacted in December 2014 to investigate a case of photosensitisation in 10-month-old White Suffolk – Merino cross lambs.

The lambs had been grazing unimproved pasture near the Murray River east of Albury. Over the previous 2 weeks, the lambs had crash-grazed a new lucerne stand that was heavily contaminated with common heliotrope.

Two severely affected lambs brought into the yards for examination were found to be jaundiced, blind from inflammation of the eyelids, and sunburned on the ears, with swelling in exposed areas.

Blood samples were taken to confirm photosensitisation secondary to hepatitis. At the same time, the owner examined the remainder of the mob and removed a total of 23 affected lambs, which were housed in a shed to recover. The rest of the mob was moved to a new paddock close to the shed for observation.

The district vet returned to the farm 3 days later, and the two lambs originally examined were euthanased because they hadn't improved. All other affected sheep had begun to recover, and no new cases were observed.

Necropsy of the two euthanased lambs revealed jaundice. Histopathological testing of livers and kidneys in the lab showed subtle megalocytosis



Liver damage in these sheep caused severe jaundice. Photo S. Ison

(cell enlargement) and other changes consistent with acute chemical-driven liver damage.

The naturalised pasture was examined for grass species such as witchgrass and hairy panic that commonly cause secondary photosensitisation in the area at that time of year. Suspect species accounted for only a small percentage of the pasture, and common heliotrope was presumed to have caused the disease.

Acute common heliotrope poisoning is rarely reported. The disease usually occurs after a longer period of exposure or as secondary copper toxicity following several seasons of exposure.

**For further information, contact Scott Ison, District Veterinarian, Murray Local Land Services, on (02) 6040 4210.**

## *Mycotoxigenesis in a Merino flock*

In October 2014, district vets investigated a significant disease incident near Walgett in north-west NSW following an owner report of 20 dead and 50 sick sheep in a mob of 600. The sheep were trembling, having seizures and running into fences before lying down with paddling limbs and dying.

The district vet organised for the remaining sheep to be immediately moved into a different paddock until the cause of the deaths could be determined. A total of 80 sheep became affected and died during the event.

The mixed mob of ewes, lambs and wethers had been grazing native

pasture supplemented with barley hay and grain. The weather had been very hot, with temperatures above 40°C the week before. The property was in drought, with limited pasture feed on offer. Water was clean and readily available in troughs. In preparation for harvest, the landholder had recently cleaned out the silo and spread out the remnant grain in the paddock for the sheep to consume.

On arrival, the district vet ran an anthrax ICT (immunochromatographic test) on a dead ewe to exclude the possibility of an anthrax outbreak. The result was negative for anthrax.

Subsequently, a number of live, affected animals were examined. They all showed evidence of blindness, with varying degrees of muscle tremors and weakness of the legs. Upright animals were running into fences and running in circles. Several sheep were suffering from ongoing seizures; from among these, three ewes were euthanased for necropsy.

All three ewes showed evidence of acute liver damage, with enlarged, pale livers and swollen gall bladders. Two ewes had damage to the heart muscle, with haemorrhages present. All three

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Many sheep died from the effects of the mould toxins. Photo M. Davies

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ewes had kidney damage, with a dark purple discolouration and red stripes throughout the kidney cortex.

Liver, brain, spinal cord and kidney samples were submitted to the State Veterinary Diagnostic Laboratory under the National TSE Surveillance Program. Samples of grain from the silo were also collected for analysis.

The histopathology results from the lab showed no significant brain lesions that could have been responsible for the neurological signs in the ewes. There was no evidence of scrapie, polioencephalomalacia or hepatic encephalopathy.

Histopathology on the livers showed mild fatty liver, suggesting that there had been a recent toxic insult to the liver. Toxicity from moulds (mycotoxicosis) was considered a possible cause, so feed samples were sent for mycotoxin screening. The feed sample contained aflatoxin B1 at 140 ppb (0.14 mg/kg) and deoxynivalenol (DON) at 670 ppb.

Aflatoxin B1 is a mycotoxin produced by *Aspergillus flavus* and *A. parasiticus*, fungi that are common contaminants of grain—particularly grain stored in silos at higher than recommended moisture levels. No animal species are resistant to the acute toxic effects of aflatoxin,

but there is variability in susceptibility among species. Limited data are available for sheep, but potentially harmful levels for cattle are reported to range from 20 to 300 ppb. The level of 140 ppb that we found was therefore significant and was likely the cause of the deaths.

DON (also known as ‘vomitoxin’) is a mycotoxin produced by *Fusarium* spp.—particularly the plant pathogens *Fusarium graminearum* and *F. culmorum*. It primarily causes lack of appetite and vomiting, but recent research has shown that it can potentiate the effects of other mycotoxins when feed is contaminated with multiple fungal species, making a bad situation worse.

Mycotoxicosis is not commonly reported in sheep in Australia. However, when feed supplies are low, livestock owners may be tempted to use feeds they would otherwise avoid. Mycotoxin screening of feedstuffs is available, but its usefulness is limited because there are hundreds of different mycotoxins. The effects of the toxins also vary greatly among species and depend on the portion of the ration that is contaminated, along with the age and immune status of the animals consuming the feed.

There is no specific antidote for mycotoxicity; immediate removal of

the contaminated feed is the single most important step in avoiding further losses. In this case, sheep continued to die in low numbers for 2 days after they had been removed from the affected grain. There have been no subsequent losses.

**For further information contact Megan Davies, District Veterinarian North West Local Land Services, on (02) 6792 2533.**

# 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 Animal and Plant Biosecurity Branch in Orange on (02) 6391 3237 or fax (02) 6361 9976.

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](http://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/status/nahis.cfm](http://www.animalhealthaustralia.com.au/status/nahis.cfm)

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/newsletters/animal-health-surveillance](http://www.dpi.nsw.gov.au/newsletters/animal-health-surveillance)

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

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