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NEW SOUTH WALES

ANIMAL HEALTH SURVEILLANCE

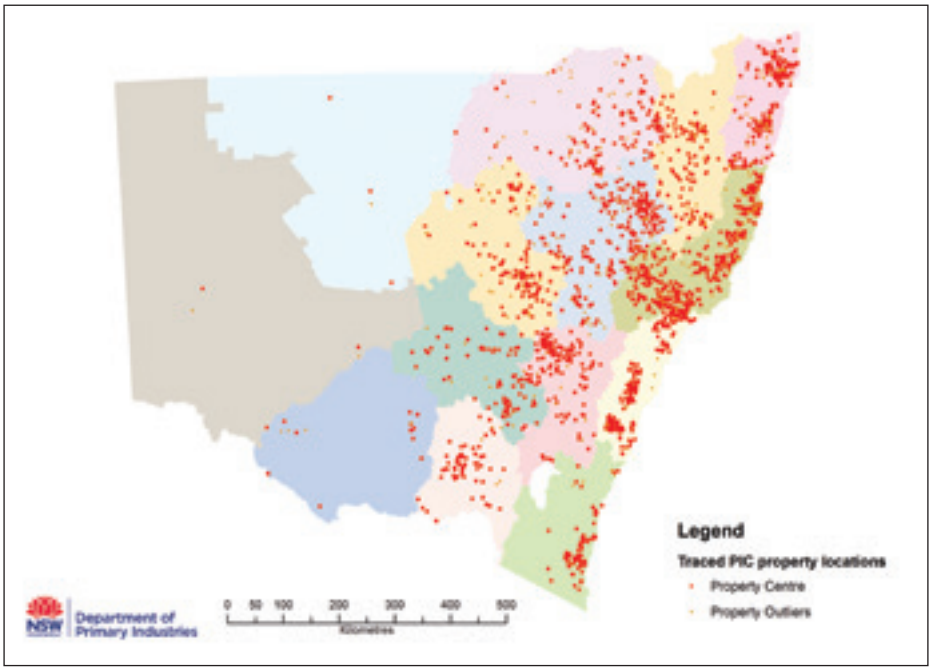
Information contributed by staff of the Livestock Health and Pest Authorities and the Department of Primary Industries

Tuckerbox musters sheep and cattle

In April 2012, Biosecurity NSW ran a simulated foot and mouth disease (FMD) desktop sheep tracing exercise, code-named Exercise Tuckerbox. The exercise used the following scenario:

A suspected case of FMD is detected on a sheep property in the Gundagai district and reported to the Chief Veterinary Officer (CVO), who activates the alert phase of an emergency response. The CVO requires tracing of all risk movements over the previous month to be completed by the following morning in preparation for a meeting of the national CCEAD (Consultative Committee on Emergency Animal Diseases). The tracing data will help to determine the size of restricted and control areas. They will also help in working out the nature and scope of, and resource requirements for, an emergency response if the disease is confirmed.

These requirements are consistent with the National Livestock Traceability Performance Standards (NLTPS), which require all tracing of stock movements on and off suspect premises during the previous 30 days to be completed within 24 hours. Exercise Tuckerbox used confidential data about real properties and livestock movements already recorded in the National Livestock Identification System (NLIS) database. This information was collated in spreadsheets and analysed.



Simulated locations of properties where cattle were identified as at risk of FMD in Exercise Tuckerbox. These cattle traces were mapped from property identification codes taken from NLIS records.

In this issue!

From the Exercise Tuckerbox simulation we learned the following:

- About 2300 properties and 438 000 cattle, sheep and goats were successfully traced.
- Traced premises were located throughout NSW, as well as in Victoria, Queensland, South Australia, the ACT and Tasmania.
- About one-third of all NSW saleyards and most abattoirs were detected in the tracing.
- Mob-based or summary database reports were the fastest way of completing tracing for both sheep and cattle.
- The equivalent of 7 staff completed all tracing in less than 12 hours' working time.
- The likely presence of pigs, deer and other FMD-susceptible species on traced properties was confirmed by using LHPA (Livestock Health and Pest Authorities) annual land and stock return data, but no movements of these animals could be traced, as movements are not recorded on national databases.

Exercise Tuckerbox confirmed that the current mob-based system for NLIS Sheep and Goats and the individual electronic identification system for NLIS Cattle are equally effective in tracing for FMD.

For further information contact Lisa Davison, Senior Policy Officer Biosecurity Traceability Systems, NSW DPI Orange, on (02) 6391 3212.

Expansion of property identification code system in NSW

The property identification code (PIC) system has been expanded in NSW. From 1 September 2012, anyone who keeps livestock will be required to obtain a PIC for the land on which the livestock are kept.

The change in PIC rules in NSW is covered under clause 37 of the Stock Diseases Regulation 2009, which states that all livestock owners, managers, and occupiers of land that carries one or more cattle, sheep, goats, pigs, bison, buffalo, equines, deer, camelids and poultry must ensure the property has a PIC. This clause was subject to an exemption from 1 September 2009, but the exemption expired on 31 August 2012.

Equines include donkeys, mules, asses and zebras. Poultry include 100 or more chickens, turkeys, guinea fowl, ducks, geese, quails, pigeons, pheasants, partridges, and 10 or more emus and/or ostriches. The threshold for 100 poultry includes properties keeping 100 mixed species of poultry.

Some properties will be exempt from needing a PIC, namely those that have fewer than 100 poultry or 10 emus or ostriches, veterinary practices where stock do not stay overnight, laboratories, and animal exhibits licensed under the *Exhibited Animals Protection Act 1986*.

PICs allow NSW DPI and the LHPA to quickly identify the location of a property and its associated livestock, as well as its owner or occupier's contact details and its boundary details (Lot and DP) in the event of an animal disease outbreak.

PICs are already in use and are integral to the operation of the NLIS (National Livestock Identification System) to trace the movement of cattle, sheep, goats and pigs between properties and to sales or slaughter.

All states and territories have already put in place PIC systems so that they can identify properties with livestock. The expansion of the PIC system in NSW will provide similar information and is a critical component of our Biosecurity strategy.

The key message is that every livestock owner or producer in NSW should have a PIC for the land on which they keep their livestock.

For more information on PICs, or to apply for a PIC, see the dedicated PIC page on the LHPA website (www.lhpa.org.au) or contact an LHPA office.

Unusual pulpy kidney in lambs

Three hundred and fifty out of 1500 three- to four-week-old Merino lambs on a property north-west of Cobar were suspected to have died from an unusual case of pulpy kidney (clostridial enterotoxaemia).

The lambs were dying between 1 and 4 weeks of age, but mainly at age 3 or 4 weeks. Big, strong, healthy lambs were found dead, with their mothers standing beside them. The unvaccinated Merino ewes were in great condition—typically 3 or 4 (out of 5) score—and were grazing on native pasture with no supplementary feed. The feed was a mix of tall dry grasses with green pick coming through.

One 4-week-old lamb was necropsied after being stored in the coolroom for 24 hours. There were no remarkable external lesions. Some haemorrhages were seen throughout the lungs. The heart appeared normal,

apart from some calcification of the auricles. There was a large amount of clotted milk in the abomasum and green matter in the rumen. The intestines were mainly empty, apart from a build-up of gas. The kidneys had adequate fat cover but were soft and hard to cut into sections.

Histopathological examination showed accelerated renal autolysis (self-digestion); this supported the diagnosis of pulpy kidney.

Pulpy kidney is generally associated with a rapid change of diet, either on lush, rapidly growing pasture or with grain feeding. In this case, the change of diet that had allowed the *Clostridium perfringens* bacteria to proliferate was the increasing amount of milk that was being supplied by the strong, healthy ewes to these lambs, as well as the rise in pasture consumption typical of such rapidly growing lambs.

For further information contact Charlotte Cavanagh, Veterinary Officer, NSW DPI Bourke, on (02) 6830 0003.

Cluster of (non-exotic) poultry diseases

In mid-August 2012 three poultry farmers contacted the North Coast LHPA because of disease in their flocks. The first producer was the owner of 1000 free-range chickens near Lismore. He reported three birds with neurological signs such as head tilt, followed by weight loss and death. The second producer was a poultry fancier from near Murwillumbah. He found respiratory disease in 15 of his 40 birds, and a few had died. The third producer was also a poultry fancier but was located closer to Lismore. He reported respiratory signs in 15 of his 50 birds. Some of the birds had shown mild illness and recovered, whereas the signs had been so severe in other birds that he had needed to have them euthanased. The disease began about 2 weeks after he had brought some of his birds home from a local poultry show. Both of the poultry fanciers had sought help from their local private veterinarian, but antibiotics had not improved the illnesses. The first and third producers each gave the North Coast LHPA a sick live bird, which was examined, euthanased and necropsied.

The first bird was a mature hen in poor body condition. It could walk around but was weak and held its head upside down with a turn to the left. It occasionally would attempt to eat but was usually unsuccessful.

The third bird was in moderate body condition, with a high temperature, mild breathing difficulty and a watery discharge from the nostrils. The comb and wattles were very red. There was an obvious noise when the bird breathed.

Apart from weight loss, no gross abnormalities were detected in the necropsy of the first bird. The second bird was sent to the State Diagnostic Laboratory for necropsy and testing. The bird had enlarged, dark, rounded kidneys. Necropsy of the third bird by the North Coast LHPA revealed that the pharynx was more red than usual and the nostrils were filled with fluid.

The second and third birds were tested for avian influenza, infectious laryngotracheitis and Newcastle disease. Both birds were negative for all these diseases on PCR (polymerase chain reaction) testing.

Histopathology of the bird with a head tilt (the first bird) showed severe otitis media (middle ear infection) consistent with a bacterial infection. *Pasteurella multocida* was suggested as a likely cause. The second bird had moderate, chronic non-suppurative rhinitis, pharyngitis, tracheitis and bronchitis. The third bird had ulcerative tracheitis and pneumonia and a substantial parasite burden in the intestine. Bacterial culture of the lungs and trachea of the second bird yielded a profuse growth of *Pseudomonas aeroginosa*.

It was a coincidence that three significant poultry outbreaks were reported within 4 days and within a relatively small region. Exotic disease was not the cause of these outbreaks. The neurological condition in the first bird was the result of a sporadic endemic disease. *Pseudomonas* was the likely cause of disease in the second bird. The farm had experienced very wet conditions, which would have favoured the survival of these bacteria. We assumed that the outbreak on the third producer's farm was caused by an endemic viral or bacterial respiratory disease. Further testing was not done to determine a cause, but infectious bronchitis, mycoplasma and infectious coryza are possibilities.

For further information contact Matt Ball, Senior District Veterinarian, North Coast Livestock Health and Pest Authority, Lismore, on (02) 6621 2317.

Low pathogenic avian influenza H4N6 in ducks

Low pathogenic avian influenza subtype H4N6 virus was found in ducks of several age groups on a multi-age farm of 2400 ducks located on the North Coast of NSW. The virus was identified during investigations of respiratory disease and ongoing deaths in several batches of ducks. Increased mortality was observed, along with respiratory signs such as dyspnoea (laboured breathing), sneezing, facial swelling and conjunctivitis (discharge from the eyes). Sickness rates varied from batch to batch. About 300 out of 5440 ducks died in 11 batches over a 5-month period. At least 50% of the batches experienced higher than normal death rates (as high as 13%). Mortality in these batches peaked at 8 to 10 ducks a day at age 16 days.

Serology testing showed that the respiratory signs and deaths were not consistently associated with infection with avian influenza. *Mannheimia haemolytica* (previously *Pasteurella haemolytica*) was consistently isolated from the ducks submitted to the laboratory. This organism has often been reported to be associated with a variety of clinical signs and variable death rates, especially in young chickens and turkeys. Its role in ducks has not been elucidated, but it is generally regarded as a secondary pathogen.

The ducks were brooded in a shed until age 28 days and then moved outside until 8 or 9 weeks of age, when they were sent to a small local poultry abattoir for processing. Infection was evident mainly in the ducks inside the shed. No obvious sickness or deaths were observed in chickens or geese housed 10 to 20 m away from the ducks, and infection with H4N6 was not detected in these species.

The source of H4N6 could not be identified, but there is a dam frequented by wild waterfowl on the property; the wild waterfowl could have been the source.

H4N6 has not been reported to cause clinical signs in humans, and none was detected in the farm staff or the processing plant workers.

For further information contact George Arzey, Technical Specialist Poultry, NSW DPI Menangle on (02) 4640 6402 or Phillip Kemsley, District Veterinarian, North Coast Livestock Health and Pest Authority, Casino on (02) 6662 3166.

Low pathogenic avian influenza H9N2 in turkeys

In April 2012, H9N2 low pathogenic avian influenza virus was confirmed on a turkey farm housing about 26 500 turkeys in three sheds near the Hunter Valley. The birds ranged in age from 27 to 43 days. Increased death rates were evident in one shed, and coughing, sneezing and gasping were observed in a second shed. At necropsy, sinusitis, fibrinous pericarditis (inflammation of the heart sac), perihepatitis (inflammation of the surface of the liver), enlarged liver and spleen, inflammation of the air sacs and swelling around the eyes and beak were evident.

Exclusion testing by PCR (polymerase chain reaction) at the State Veterinary Laboratory found that the samples were positive for avian influenza virus. The virus was later identified by the AAHL (Australian Animal Health Laboratory) as H9N2. This avian influenza virus subtype is common in poultry in other countries, but this is the first time that it has been reported in poultry in Australia. It is also the first reported case of any avian influenza in turkeys in Australia.

The company chose to depopulate the farm. Dead birds and other organic matter were composted on the farm, and the temperature of the composting piles was monitored to ensure it was hot enough to destroy any pathogens.

There had been no recent movements of turkeys from the farm, and there were no other commercial poultry farms within a 10-km radius. Surveillance was done on the company farms visited by those personnel or feed trucks that had visited the infected farm. On one of these farms the same virus was detected in a healthy flock of turkeys. When this farm was tested 2 weeks later no avian influenza virus was detected on PCR, but samples were positive for avian influenza antibody, indicating that the infection was no longer active.

It is unclear whether the virus was transmitted somehow between the two farms or was acquired independently by each farm. Although the second farm was visited by a person who had visited the first farm, the visit to the second farm took place 2.5 days after the first and the person didn't enter the sheds. The second farm was in a wetland area with plenty of opportunities for transmission of the infection directly or indirectly via wild ducks.

Although the first farm's water supply came from a large dam near the sheds, the drinking water was chlorinated at concentrations aimed to kill this and other viruses. The virus could have entered the sheds on boots or equipment, or in the drinking water if it had been treated sub-optimally.

Routes of avian influenza infection on farms have rarely been identified in outbreaks of avian influenza in Australia, but generally wild waterfowl are believed to be the main source of infection. In May 2012 an H9 avian influenza virus subtype was identified in samples collected from magpie geese and wild ducks in the Hexham wetland area, and in previous years H9N2 was detected in wild ducks on the Morpeth sewage ponds. Both areas are within a 40-km radius of the affected farms.

For further information contact George Arzey, Technical Specialist Poultry, NSW DPI Menangle on (02) 4640 6402.

Swine brucellosis and leptospirosis in feral pigs in northern NSW

Five out of a sample of 10 feral pigs in northern NSW were recently found to be serologically positive for *Brucella suis* on Rose Bengal and complement fixation testing. These same five pigs were also positive for *Leptospira pomona* in microscopic agglutination testing. The pigs were sampled as part of a longer-term study looking at the prevalences of *B. suis*, *L. pomona* and *Leptospira hardjo* in feral pigs across northern NSW. This study is due for completion in early 2013.

Brucellosis in pigs is caused by the *Brucella suis* bacterium. Clinical signs of infection include orchitis (inflammation of the testes) and reproductive failure. Swine brucellosis has been previously reported as an infection of feral pigs in northern Australia, but this is the first time that *B. suis* antibodies have been detected in the NSW feral pig population. To confirm this infection, more serology samples from feral pigs in northern NSW will be collected. We will also try to recover *B. suis* organisms from the tissues of test-positive pigs.

Brucella suis infection has also recently been found in pig-hunting dogs in NSW. In two separate cases in March and July 2012, four pig-hunting dogs tested serologically positive for *B. suis*. Two of these dogs presented to private vets with orchitis, but they were otherwise bright and alert.

Brucella suis is a zoonotic disease (i.e. it can be transmitted between animals and humans), and since 2008 there have been 16 human cases diagnosed in NSW. Infection in humans can cause serious disease and spontaneous abortion in women. Clinical signs include intermittent fever, sweating, lethargy, loss of appetite, headaches, and back pain. If left untreated, infection is fatal in about 2% of people.

Brucella suis is a notifiable disease in NSW, and any suspect cases in animals should be reported to a NSW DPI inspector or LHPA inspector.

Leptospirosis can adversely affect pig and cattle health and production. Some animals are asymptomatic carriers and are a source of infection to other animals. In humans leptospirosis can cause a serious flu-like illness that can be fatal without treatment. Since 2008 there have been 112 human cases of leptospirosis infection in NSW.

Given that both *B. suis* and *L. pomona* pose important human health risks, individuals exposed to live or recently killed feral pigs should take preventive measures to minimise the risk of infection. This includes wearing protective clothing such as gloves when handling animals, covering any cuts, avoiding contact with animal fluids (e.g. blood, urine) and avoiding cutting into reproductive tissues potentially harbouring the bacteria when gutting pigs and doing necropsies. Consider using face and eye protection, especially when the risk of exposure is high.

This feral pig survey was part of a collaborative project run by NSW DPI, Pfizer Animal Health and Hunter New England Population Health.

There have been no reports of *B. suis* in commercial pig populations in NSW.

For further information contact Camila Ridoutt, Graduate Officer, NSW DPI Menangle, or Amanda Lee, Technical Specialist Pigs, NSW DPI Menangle, on (02) 4640 6308.

Field pea toxicity in two Hereford heifers

In mid-August 2012, a property near Burren Junction in the North West of NSW was investigated for the cause of neurological signs in two 12-month-old homebred Hereford heifers. The animals at risk included 40 heifers that had grazed on a 10-ha paddock of field peas (*Pisum sativum* var. *arvense*) and fodder oats for 14 days. The peas were in the pre-flowering stage of growth when eaten. At the time that the neurological signs were noticed, all the oats had been eaten and the remaining field peas had been grazed to one-third of their original height.

A nearby, similar paddock of field peas was being grazed by cattle that appeared healthy, but the field peas in this paddock had not been grazed as low.

Fourteen days after the heifers had been introduced to the paddock, the property owners noticed that one had isolated herself from the mob and was repeatedly trying to belch. By that afternoon, the heifer was showing progressively more severe nervous signs, including circling, agitation when approached and repeated flank licking.

The affected cows were in this paddock. The fodder oats had been completely grazed and a pre-flowering field pea crop had been grazed to one-third of its original height. Photo by Shaun Slattery

The cows in this paddock were unaffected. The field pea crop had not been grazed as low. Photo by Shaun Slattery

A second heifer was also beginning to show signs. The mob was moved from the paddock.

The next day, one affected heifer developed a grand mal seizure when yarded. During recovery she displayed repeated muscular tics and jaw chomping. Her vision was not affected. Her temperature, heart rate, respiratory rate, lung sounds, urine dipstick results and mucous membranes were all normal.

Blood analysis revealed no evidence of lead poisoning, sporadic bovine encephalomyelitis (caused by *Chlamydophila pecorum*), or metabolic disease. The level of creatine kinase enzyme (a marker of muscle damage) was high, and the level of GLDH (glutamine dehydrogenase enzyme, a marker of liver disease) was very mildly elevated. Urea and creatinine levels (indicators of kidney function) were normal, but the urea to creatinine ratio was elevated.

A previous case report of *Pisum sativum* var. *arvense* toxicity by Reardon and McKenzie (*Australian Veterinary Journal* 2002) suggests that the way the toxin acts is unknown, but there could be abnormal modification of an enzyme from the pea in the rumen, resulting in the formation of a neurotoxin.

A histopathology report on a bull on the property that died after displaying similar neurological signs revealed cerebral oedema (swelling); this is seen with many toxicities, including ammonia toxicity.

Key features of this case included the fact that the affected cattle recovered rapidly after being removed from the field pea crop; they had normal liver enzymes and kidney function, although they had a slightly high urea to creatinine ratio. These features support the idea that it was a protein-related toxicity, possibly similar to the non-protein nitrogen neurotoxicities seen in cattle that have eaten ammoniated feeds (also known as 'bovine bonkers syndrome'). This syndrome is characterised by wildly unusual behaviour (as displayed in this case). It is thought to be caused by the formation of 4-methylimidazole through the action of ammonia on soluble carbohydrates in ammoniated feedstuffs.

'Pea mania' is reported to occur after consumption of plants in the pre-flowering stage of growth, as occurred in these

cattle. However, unlike with pea mania, no environmental factors such as water stress can be attributed to the toxicity in these cattle, although they were grazing field pea plants at high concentration.

It is possible that an enzyme reaction occurred in the rumen, creating an unknown neurotoxin unrelated to ammonia.

Further investigations of cattle with 'bovine bonkers' are needed. These include analyses of ammonia blood levels and rumen pH sampling.

For further information contact Shaun Slattery, Senior District Veterinarian, North West Livestock Health and Pest Authority, Narrabri on (02) 6792 2533.

Severe bronchopneumonia in weaner cattle

The Hume LHPA was called in to help diagnose a problem in 330 ten-month-old Murray Grey weaners destined for a feedlot. They had been weaned onto a holding paddock about 2 weeks previously and were provided with slightly mouldy silage, hay and water. The weather had been bad about 1 week after the weaning. Over the previous few days, the owner had noted coughing, nasal discharge and the sudden death of two young bulls and had called a local private vet to assess the herd. The private vet noted marked coughing, high temperatures, and difficulty breathing. The worst-affected in the herd were separated and given an injection of long-acting antibiotics (oxytetracycline).

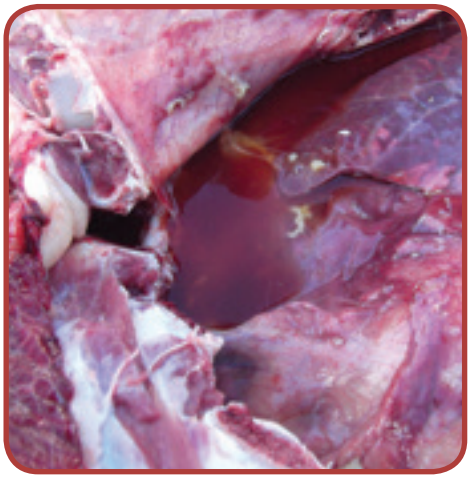
The senior district veterinarian noted that many in the affected mob were depressed and had their heads down and necks extended, with shallow frequent breathing and intermittent coughing. Several animals had a copious seromucous nasal discharge. The sickness rate was estimated to be 50% to 60% but may have been as high as 75%. The most severely affected animals were examined and found to be febrile (average temperature 40°C to 41°C). They had a fast heart rate and breathing. Blood was taken from 10 of the most affected animals for analysis.

One animal from the previously treated animals was selected for necropsy. The heifer appeared dull, had lost condition, and remained lying down until closely approached. The necropsy revealed blood-stained fluid in the chest, with fibrin clots and pleuritic tags throughout the chest cavity. There was severe consolidation of the ventral two-thirds of the lungs, with a sharp line between the grossly affected and unaffected lung tissues. The affected portion of the lungs was dark red to purple and firm; there were pus-filled cavities and fibrosis on the cut surface.

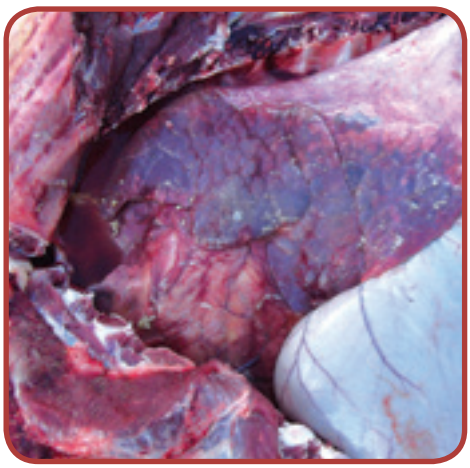
The laboratory confirmed a diagnosis of severe bronchopneumonia and pleurisy, with cuffing pneumonia (a type of lung infection in which cuff-like borders of white blood cells form around small blood vessels); this was suggestive of lymphocytic peribronchiolitis (inflammation of the small airways of the lung, with invasion by white blood cells). The lab isolated a profuse growth of *Mannheimia haemolytica* from the lung tissue. *Mannheimia haemolytica* usually does not initiate disease, i.e. it is a secondary pathogen. Laboratory virology results confirmed an infection with BRSV (bovine respiratory syncytial virus) in three of the weaners. There was IBR (infectious bovine rhinotracheitis) virus in one weaner and pestivirus infection in another.

This situation is likely to have been initiated by stress from the weaning, the close housing, the changes in diet and the adverse environmental conditions. The result was an explosion of bovine respiratory disease complex on this property. The close housing and contamination of feed and water sources allowed rapid transmission between individuals.

All animals in the herd were treated with long-acting oxytetracycline injections and moved from the yards to more suitable pasture. The mouldy silage was replaced with higher quality hay and silage, which was fed in a more dispersed fashion to avoid close contact of animals between feeding. There were no further deaths on the property, and most of the cattle appeared to return to normal after their injection. A small proportion that did not 'look right' were given a second dose of oxytetracycline. Despite the clinical improvement, the lung damage that the animals suffered is likely to cause an ongoing decrease in productivity; the owners were made aware of this.



Blood-stained hydrothorax with fibrin clots and 'tags' indicating pleurisy (inflammation of the lining of the lungs and chest). Photo by Steve Whittaker



Severe consolidation of the ventral two-thirds of the lung. Photo by Steve Whittaker

Firm, consolidated lung tissue with pus-filled cavities or abscesses and fibrosis on the cut surface. Photo by Steve Whittaker

For more information contact Steve Whittaker, Senior District Veterinarian, Hume Livestock Health and Pest Authority, Albury on (02) 6040 4210.

Yersiniosis in cattle on the North Coast

Cases of yersiniosis (an infection caused by the bacterium *Yersinia*) in cattle are seen each winter on the North Coast. In winter 2012, I investigated eight cases over a 6-week period. A further 14 cases were reported by other veterinarians in the district.

Three of my investigations were done within a 15-day period, from 23 July to 6 August 2012. The properties are located within 3 km of each other in an area known as Greenridge, about 10 km south-east of Casino on the mid Richmond floodplain.

The death rates in the herds were 1 of 19 in herd A, 2 of 42 in herd B and 2 of 52 in herd C.

On 23 July 2012 a Simmental bull from herd A was found dead after 2 days of lethargy, lack of appetite, and acute weight loss and diarrhoea.

In herd B a 6-year-old Murray Grey cow was found dead. Two weeks later, on 30 July, a 4- to 5-year-old Murray Grey cow was found down and unable to rise; for 2 days she had had weight loss and profuse diarrhoea. The cow was near death, with laboured breathing, dehydration and a temperature of 35.5°C. She was euthanased and necropsied.

On 4 August, in herd C, a mature Angus X Hereford cow was found dead with signs of diarrhoea. Two days later, on 6 August, a Brangus cow was found down. The cow was under a fence and unable to rise. She had diarrhoea and was groaning, grinding her teeth and unresponsive. She had a temperature of 38.8°C. The cow was euthanased and necropsied.

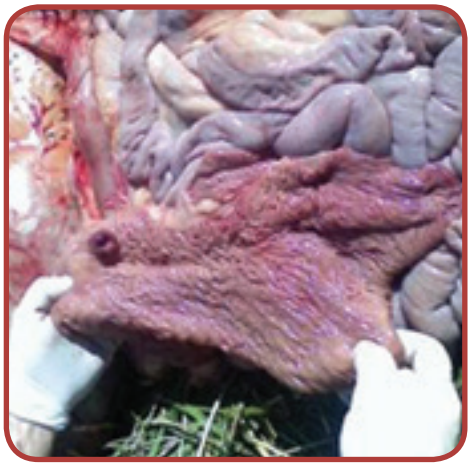
The necropsy findings in all three animals were very similar. There was inflammation of the caecum, colon and ileum. Changes to the mucosa included thickening, ridging, inflammation, a swollen ileocaecal valve and a thick, white catarrhal fluid. There were casts in the colon. There was minimal change in the intestinal lymph nodes. Petechial (small) haemorrhages were found throughout the carcass, including in the omentum, mesentery, pleural surfaces and epicardium (the outer layer of heart tissue).

Samples for laboratory testing were taken only from herd B. *Yersinia pseudotuberculosis* was isolated from the ileum and from the contents of the intestine of the necropsied cow. A faecal sample was taken from the carcass of the first cow that had died; it was negative for *Yersinia*.

Yersiniosis had been confirmed from earlier cases in the district, so because of the characteristic gross pathology we did not try to isolate it from all cases.When yersiniosis is confirmed on a property, the owners should check the cattle at least twice a day for signs of lethargy, lack of appetite or diarrhoea. Any sick cattle should be treated with long-acting oxytetracycline.

As with most conditions, a paddock shift is recommended.

In continuing outbreaks there is a place for preventive administration of antibiotics, for example in situations where stock can't be moved to another paddock.



Thickened mucosa and swollen ileocaecal valve in a cow with yersiniosis. Photo by Phillip Kemsley

For further information contact Phillip Kemsley, District Veterinarian, North Coast Livestock Health and Pest Authority, Casino on (02) 6662 3166.

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. If you would like more specific information about diseases occurring in your part of the state, contact your local Livestock Health and Pest Authorities District Veterinarian or Departmental Regional Veterinary Officer.

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.

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

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

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