NSW GOVERNMENT

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

NEW SOUTH WALES

ANIMAL HEALTH SURVEILLANCE

January–March 2014 » Issue 2014/1

IN THIS ISSUE

Anthrax surveillance: an essential official veterinary service role

Surveillance for anthrax is one of the biggest priorities for NSW DPI and Local Land Services. An outbreak of this disease has the potential to cause trade disruption and market loss.

Anthrax was excluded as the cause of death on 20 occasions between January and March 2014, and there were no positive cases of anthrax during the quarter.

Twelve investigations involved cattle and five involved sheep. One investigation each involved a horse, a wild eastern grey kangaroo and a Barbary sheep in a zoo.

The anthrax ICT (immunochromatographic test) has revolutionised safe surveillance for anthrax. A blood sample can be tested in the field. If it is negative, the vet can safely go ahead with a post-mortem examination. Twelve such tests were conducted in the field in the last quarter.

In one case in early March, a ram in good condition in a group of 30 rams died suddenly overnight. Another ram was sick and lying on its side. The flock was in a 50-hectare dry lucerne paddock. The paddock was very bare and the sheep were being fed supplementary oats trailed out on the ground.

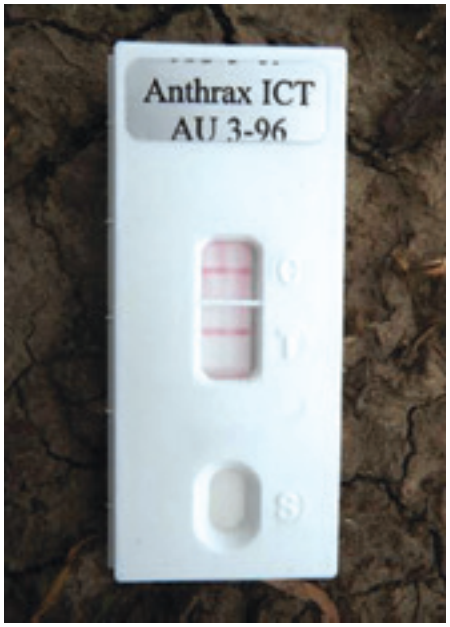
The dead ram was flat out on its right side, and there were signs of struggle in the red dirt where it lay. The oral mucous membranes were dark blue. A small amount of frothy fluid and blood was exuding from the nose. The eyes were sunken and the skin tented when raised, pointing to severe dehydration. Anthrax was considered a possibility; the District Vet conducted an ICT with negative results and was able to safely go ahead with a post-mortem examination.

The dead ram had severe pneumonia. The bottom rearward half of the left lung was stuck to the chest wall. A cheesy-green film coated the lung's surface and the affected tissue was dark purple-black and firm to the touch. The remaining lung tissue was red-purple, with blood oozing out of the cut surface.

At necropsy, the left lung had approximately 10 nodules ranging in size from 5 to 20 millimetres scattered across the surface and extending down into the tissue. The nodules were firm to touch and contained green pus.

The final diagnosis confirmed at the laboratory was pulmonary pneumonia and septicaemia with abscesses due to the bacterium *Pasteurella multocida.*

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



Anthrax ICT. In the immunochromatographic test, two red lines indicate a positive result. Photo S Slattery

New test solves the bluetongue disease surveillance problem

Australia does not have *clinical* bluetongue disease in sheep or cattle, but we do have some bluetongue virus strains that could cause disease if they were to enter our sheep-raising areas. Moreover, new strains could enter Australia in biting midges (*Culicoides* spp.) carried on the wind from South East Asia, because exotic viruses and vectors continue to enter northern Australia in association with severe weather events.

With the increasing weather pattern volatility—and particularly rises in temperatures—southern extension of the distribution of vectors is likely, threatening large sheep populations and rendering large numbers of sheep and cattle ineligible for export.

Past testing methods used for surveillance for bluetongue are too slow to enable sensible response

plans to be developed should there be a clinical case of bluetongue. By the time insects are collected in light traps and any *Culicoides* are identified by entomologists, the *Culicoides* could have moved hundreds, if not thousands, of kilometres. Virus culture techniques are then required to see if the *Culicoides* are actually carrying the virus. By the time all the surveillance testing is completed, the information is way out of date.

To solve the problem, real-time PCR (polymerase chain reaction) assays have been developed that allow us to rapidly detect and identify both exotic vectors and any bluetongue viruses that they are carrying. Results can be obtained for several hundred samples in a day.

These assays are also extremely sensitive and have the potential to detect a single exotic midge in a collection containing more than 20,000 other insects.

For virus identification, at present, a single test can identify a maximum of five serotypes from the 10 serotypes potentially present in Australia or the 26 that have been identified globally.

However, a very sensitive prototype assay platform has now been developed on the basis of magnetic bead technology that will theoretically allow us to detect all known serotypes. Collectively, these assays have the potential to revolutionise both insect and virus detection and identification. It is especially important that the insects are not destroyed by the tests, because identification of the insects by entomologists is still useful for confirming the test results.

In practical terms, when a case of clinical bluetongue is confirmed, a surveillance grid can be immediately set up and samples collected and analysed within days. Response staff and property owners will be able to sample midges (in light traps and off cattle) and send them to the laboratory for immediate testing. We will be able to rapidly decide which areas are already infected, which are clean, where the disease is likely to appear next and whether vaccination may be helpful, thus saving enormous amounts of money and potentially preserving the markets for exports from new zones declared bluetongue free.

Biting midges (*Culicoides*) transmit bluetongue virus. Photo DPI image library

For further information contact Dr Peter Kirkland, Senior Principal Research Scientist, Elizabeth Macarthur Agricultural Institute, DPI Menangle, on (02) 4640 6331.

New national standards for private vets working in emergency animal disease responses

What happens if we have a foot and mouth disease outbreak? Governments will need private vets to help.

The equine influenza outbreak highlighted inconsistencies in the conditions under which private vets were employed by the states in such emergencies and in their rates of pay.

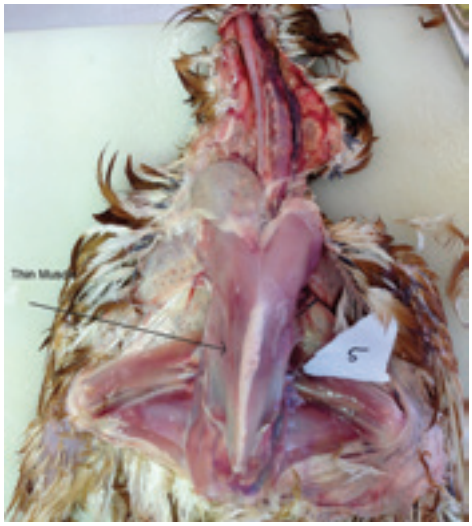
The great news is there are new contracts for the employment of private vets in EAD (emergency animal disease) responses. A working group consisting of the Australian Veterinary Association and government and private vets has developed employment conditions and a remuneration policy that are nationally consistent. There are now clear guidelines on what determines a vet to be an employee or contractor (see *www.dpi.nsw.gov.au/biosecurity/animal/info-vets*). Issues such as professional indemnity and insurance are also covered in the policy.

Vets can be employed directly by the state government as temporary or casual employees, or as contractors. Employees will be paid at one of three rates according to their experience and training. The current rates are between $86,247 and $109,584 a year. For contractors, the current rate is $180 per hour.

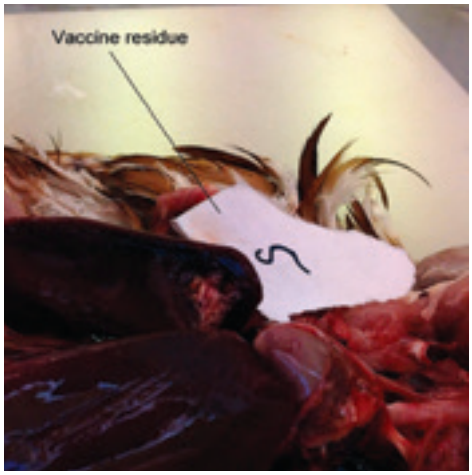
Rates of pay will be reviewed annually and increased according to changes in Commonwealth pay rates.

For further information contact Sarah Britton, Veterinary Officer, DPI Orange, on (02) 6391 3717.

Sudden death in chickens after vaccination



Thinness of the breast muscle may increase the risk of vaccination errors. Photo A Lee



Vaccine residue on the underside of the liver. Photo A Lee

Sudden death occurred in 11-week-old Lohmann Brown pullets following vaccination with an inactivated oil-emulsified combined Newcastle disease and egg drop syndrome vaccine.

The vaccine was administered by experienced vaccinators using a 20-gauge × 12-millimetre needle. On the day of vaccination, the ambient temperature was 30 to 32 °C.

The 2000 birds were vaccinated with one batch of vaccine with no problems. However, when the second batch was used, 70 out of 7000 birds died within 1 hour of vaccination. Alarm bells were raised because of the possibility that the deaths were caused by the vaccine. However, the vaccine manufacturer confirmed that this particular batch of vaccine had been used by three other customers without any similar issues.

The consulting poultry vet considered that accidental injection of vaccine into internal organs was the most likely cause of death. Five birds were subsequently frozen and submitted to the State Veterinary Diagnostic laboratory at EMAI (Elizabeth Macarthur Agricultural Institute). Freezing creates many artefacts (e.g. bruises in the muscle or fluid in cavities) and is not recommended if histological examination is required. However, in this case, because of the distance from the laboratory, the owner of the birds could not guarantee that he could supply fresh dead birds by the next day.

At necropsy the findings were the same in all of the birds: there was vaccine in the main body cavity and in the liver.

The lateral and distal breast muscle is less than 10 millimetres thick in birds. Particular care must be taken during intramuscular injection of vaccines with a 12-millimetre-long needle to ensure that the thick part of the breast muscle is the site of injection. Aligning the needle parallel to the breast muscle rather than perpendicular to the breast muscle will help prevent injection into the body cavity. Vaccinating the pullets at 11 weeks of age instead of the normal 12 to 13 weeks may have resulted in less breast muscle being available for administration of the vaccine. In addition, the pullets appeared to be underweight at necropsy.

Subsequently, the same batch of vaccine was used in other flocks without a problem. In this case, the deaths were attributed to shock as a result of aberrant vaccine administration.

The owner indicated that he would retrain his vaccinators so that the problem did not recur.

For further information contact Amanda Lee, Senior Veterinary Officer, Animal Biosecurity Branch, EMAI, on (02) 4640 6308.

Eastern grey kangaroo mass mortality event, NSW

Three hundred and fifty eastern grey kangaroos (*Macropus giganteus*) died over a 3-month period in the grounds of a hospital on the shores of Lake Macquarie, on the Central Coast of New South Wales.

The grass around the hospital, which was surrounded by bushland, had been browsed low to the ground by resident macropod populations. The site had a high population of insect parasites: ticks, biting midges and mosquitoes.

The kangaroos were the most common species of macropod on the site; however, red-necked wallabies (*Macropus rufogriseus*) and swamp wallabies (*Wallabia bicolor*) were present in low numbers. The area was described as overpopulated with kangaroos.

Despite signs telling visitors not feed the wildlife, international tourists, the local community and other visitors were hand-feeding the kangaroos.

Both adult and juvenile kangaroos were found dead; small numbers were also found profoundly weak but died soon after. Clinical signs also included bleeding from the nose (epistaxis) and pale mucous membranes, as well as neurological signs such as a 'wide-based stance', drooling, seizures and staggering. There were no reports of red-necked wallaby or swamp wallaby deaths at the site.

Multiple tissue sets were collected from 17 dead kangaroos and samples sent to a number of laboratories for toxicology, bacteriology, virology and entomology. In addition, a number of live, clinically affected animals were examined, a site environmental assessment was conducted, and a daily mortality log was collated.

The live animals had mild subcutaneous oedema and pinpoint haemorrhages on their mucosal surfaces. The dead animals had extensive, multisystemic haemorrhage and oedema, including pulmonary congestion, oedema and haemorrhage.

Preliminary laboratory analysis detected a *Babesia*-like parasite in blood films and brain and kidney impression smears. The epidemiology of the event, however, is not characteristic of the macropod babesiosis caused by the protozoan *Babesia macropus*.

Babesia macropus has been identified only in kangaroos less than 19 months of age, and mainly in hand-raised animals. It normally causes chronic anaemia and ill thrift. It has not been previously associated with disease in adults, or with acute mortality, multisystemic haemorrhagic disease, or mass mortality.

Further molecular and microscopic characterization of the *Babesia*-like parasite will help us to determine whether this protozoan parasite is similar to the *Babesia* sp. previously described in Australian macropods. Further research is required to determine its significance and pathology.

Because of the unusual presentation of this event, investigations are ongoing and will aim to determine the possible presence of co-infection. Similar cases should be reported to your District Vet or the Australian Wildlife Health Network.

For further information contact Tiggy Grillo, National Coordinator, Australian Wildlife Health Network, on (02) 9978 4788.

Pacific oyster deaths in Port Stephens: further investigation through experimental transmission trials at EMAI

Animal Health Surveillance 4/2013 reported on major deaths of farmed Pacific Oysters in the Port Stephens estuary from January to June 2013 and then again from November 2013 to January 2014.

Despite thorough diagnostic investigations involving 54 laboratory submissions to NSW DPI's Elizabeth Macarthur Agricultural Institute (EMAI) and a structured sampling program run in January 2014, there has been no clear evidence to implicate any single infectious agent in the oyster deaths.

To further determine whether a directly transmissible agent is involved in these deaths, an experimental transmission trial was started at EMAI in February 2014. The trial is using stored affected samples that were collected during the structured sampling program in January 2014.

Groups of oysters are directly inoculated with unfiltered or filtered homogenates derived from the stored affected samples or with a pool of bacterial isolates that have the potential to cause disease in oysters. The trial also incorporates various controls to make sure that the effects of injection of naïve oysters with the tissue homogenates can be differentiated from the effects of true infectious agents. The trial is expected to conclude in April 2014. The findings will be provided to key stakeholders, including the NSW oyster industry.

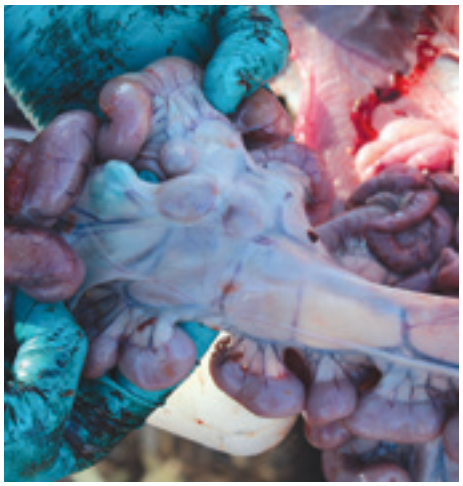
Laboratory staff trying to find the cause of death in Pacific oysters. Photo J Go

At the time of publication, the disease had not yet been replicated in the laboratory.

There have been no further reports of deaths in farmed Pacific oysters in the Port Stephens area since mid-January 2014.

For further information contact Melissa Walker, Strategy Leader, Aquatic Biosecurity Unit, DPI, on (02) 4982 1232.

3D syndrome investigation in the Western Division: *Salmonella* isolated



The affected calf had an inflamed small intestine and enlarged jejunal lymph nodes. Photo G Bailey

The oesophageal mucosa of the affected bull calf; note the light brown-red areas that could be the precursor of the inflammation of the oesophagus seen in 3D cases. Photo G Bailey and N Gillan

A sporadic syndrome with the key signs of drooling and diarrhoea leading to death ('3D syndrome') has been under investigation on extensively grazed properties in Western NSW by DPI and LLS (Local Land Services) disease surveillance specialists. Cases were first investigated in 2006, and again in 2009 and 2013. One of the earliest properties investigated was reported in *Animal Health Surveillance 2007/1* under the title 'Suspected salmonellosis in cattle'.

Although cattle of all ages have been affected, calves have had higher attack rates. Index cases were seen in November in each of three years on most, but not all, affected properties. Samples have been tested for exotic viruses, but all tests have been negative. A variety of endemic infectious agents have been found, but none consistently. At this stage the cause of the syndrome has not been identified.

This case report provides details of an investigation conducted as part of the 3D syndrome investigations.

Twenty-six cows with calves aged about 6 to 12 weeks were moved to a different paddock about 2.5 weeks before the first observed cases of diarrhoea, lethargy, and drooling in calves. No adult cattle were affected. Both paddocks had similar pasture. In the original paddock, water was provided by a bore pumped to a concrete trough. Watering in the paddock in which the calves became affected was by a ground tank; another, leaking, tank provided a shallow pool of water as well.

Five calves either died or were euthanased, and NSW DPI and LLS staff attended the property.

A 6-to 8-week-old heifer in good body condition was found lying in the shade and separated from the mob. Its rectal temperature was 40.7 °C (the ambient temperature was 37 °C) and it was not dehydrated. It had pale, watery diarrhoea staining around the perineum. At necropsy it had inflamed small intestines; this was particularly obvious in the jejunum. The jejunal lymph nodes were enlarged and the cut surface was reddened. The external surface of the caecum and colon appeared normal but the mucosa was congested, with a few small areas of haemorrhage. Histopathology later showed that there was substantial damage to the large intestine, with necrosis of the mucosal epithelial lining and severe crypt-cell damage.

A 10-to 12-week-old affected bull calf in good body condition was also found in the shade with other cows and calves. It was euthanased. Its rectal temperature was 40.5 °C but, as with the other calf, it was not dehydrated. The necropsy revealed that the oesophagus contained dark red spots of blood or mucus that could be removed by gently washing the mucosa, suggesting that the blood had been swallowed during euthanasia. However, it also had small, slightly raised dark red-brown areas on the mucosal surface. These were about 2 to 4 millimetres across and had irregular borders. They were most obvious in the thoracic section of the oesophagus, near the base of the heart.

The caecum and colon contained blood, and their mucosal surfaces were severely inflamed.

Laboratory histopathology later showed that the mucosal lining of the oesophagus had multifocal, full-thickness necrosis, with bacterial colonies and exudates of fibrin. The large intestine was similarly affected, with loss of surface epithelium, inflammation, cell damage and haemorrhage consistent with a severe bacterial infection.

Salmonella enterica serovar Give was isolated from the intestinal contents and liver of the heifer, and *Salmonella enterica* serovar Anatum was isolated from the intestinal contents of the bull calf. Both serovars have been isolated from cattle with diarrhoea, and the intestinal pathology is consistent

with salmonellosis. However, the oesophageal lesions in the bull calf are not a feature of salmonellosis but have been observed in calves and adult cattle with 3D syndrome.

To further investigate the *Salmonella* isolates, PFGE (pulsed-field gel electrophoresis; see *Animal Health Surveillance 2013/4*) was performed. Routine and selective culture was repeated on all the available samples from both calves. All 21 isolates from the heifer with *S. enterica* serovar Give had the same pulsotype, indicating that the strain was the predominant, if not the sole, *Salmonella* strain in that calf. Similar testing was performed on the bull calf with three isolates from selective culture of the pooled caecal and colon contents. All three isolates were of the same pulsotype as the isolate that was serotyped as *S. enterica* Anatum. This indicated that each calf had a distinct *Salmonella* strain, probably acquired from separate carrier animals.

Salmonella enterica of various serovars has been isolated from some, but not all, animals investigated for 3D syndrome. Further investigations are required to understand the oesophageal and intestinal pathology in 3D cases and how gut pathogens such as *S. enterica* contribute to the disease.

Given that gastrointestinal pathology is a prominent feature of the disease, the graziers have been advised to take practical measures to reduce the potential load of gut pathogens in their cattle and calves.

On the property on which the cases described above occurred, the leaking concrete tank was repaired to reduce the possibility of cattle drinking faecally contaminated water from the shallow pool that had formed beside the tank.

For further information contact Graham Bailey, Senior Veterinary Officer, Animal Biosecurity Branch, DPI, on (02) 6391 3455.

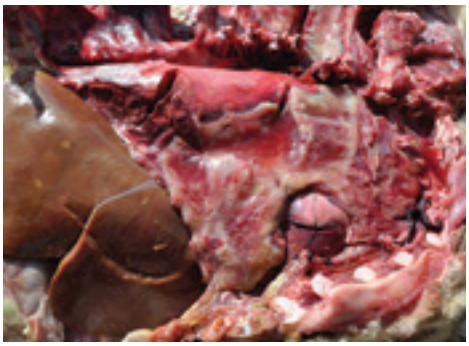
‘Summer pneumonia’ in lambs in an opportunity feedlot

Fifty lambs in an opportunity feedlot died over a 2-week period in mid February 2014 in a drought-hit region of the Central Tablelands of NSW. The feedlot contained nearly 2000 lambs.

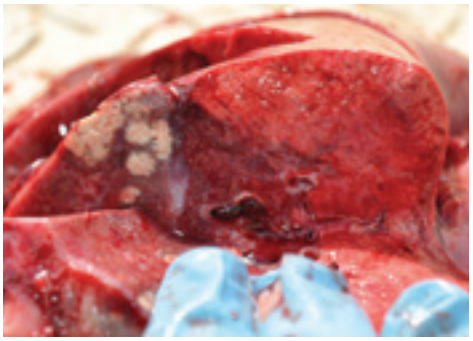
The lambs were fed grain from October 2014 onward and were introduced into the feedlot in early January. They were fed barley and lupins mixed with supplementary wheaten hay.

They presented with either sudden death or weight loss and lethargy. Some lambs showed respiratory distress, exercise intolerance and a nasal discharge.

Three necropsied lambs all showed evidence of a mark fibrinous pleuropneumonia (inflammation of the lungs and their lining) involving at least the bottom half of the lungs. The State Veterinary Diagnostic Laboratory at Menangle cultured the common pneumonia-causing bacteria *Pasteurella trehalosi* and *Mannheimia haemolytica* from the lungs, as well as *Trueperella pyogenes* (formerly *Corynebacterium pyogenes*).



Serious fibrinous pneumonia in a lamb. Photo B Watt



Cut surface of the lung, showing various pneumonia-related changes. Photo B Watt

The bacterial infection was presumably secondary to a viral or *Mycoplasma* infection, although these possibilities were not tested. The *Trueperella* infection probably followed the infection with the other bacteria. Most of the lambs responded to oxytetracycline antibiotic treatment, although 20 lambs that presumably had chronic pneumonia continued to do poorly.

For further information contact Bruce Watt, Manager Biosecurity and Emergencies, Central Tablelands Local Lands Services, on (02) 6331 1377.

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, Departmental Regional Veterinary Officer 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/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*

Disclaimer

The information contained in this publication is based on knowledge and understanding at the time of writing (April 2014). However, because of advances in knowledge, users are reminded of the need to ensure that information upon which they rely is up to date and to check the currency of the information with the appropriate officer of Department of Primary Industries or the user's independent adviser.

The product trade names in this publication are supplied on the understanding that no preference between equivalent products is intended and that the inclusion of a product name does not imply endorsement by the Department of Primary Industries over any equivalent product from another manufacturer.

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

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JN 12790 04–14