NSW GOVERNMENT

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

NEW SOUTH WALES

ANIMAL HEALTH SURVEILLANCE

April – September 2021 » Issue 2021/2

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Background to the NSW DPI-Local Land Services animal disease and pest surveillance program

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

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

POMS proof of freedom in south east NSW

Surveillance validates pre-import protocol and demonstrates freedom from Pacific Oyster Mortality Syndrome (POMS) in the Clyde and Shoalhaven/Crookhaven rivers in south east NSW.

In 2016, NSW DPI restricted importation of oysters from Tasmania to POMS free areas of NSW, following an outbreak of POMS in Tasmania. This resulted in a shortfall of available triploid Pacific Oyster hatchery spat (juvenile oysters) for cultivation the Clyde and Shoalhaven/Crookhaven rivers on the NSW south coast. In 2018, NSW DPI initiated a highly collaborative project involving the oyster industry and an independent consultant to address the shortfall. Following Tasmanian hatchery biosecurity upgrades, a strict shellfish hatchery protocol was developed to support importation of Tasmanian spat to POMS free areas of NSW. As a precaution, a three-year temporary suspension on outward oyster movements was placed on receiving NSW estuaries. Under this protocol, oysters were first imported to NSW on 2 August 2018.

Surveillance was completed in the Clyde and Shoalhaven/Crookhaven rivers in March 2020 and March 2021. Surveillance was competed after Food Agility CRC water quality sensors confirmed that water temperatures had reached and sustained a minimum 21°C, when POMS is known to express if present. Each year, Polymerase Chain Reaction on 160 oysters from each estuary system was completed at NSW DPI Elizabeth Macarthur Agricultural Institute. Testing 160 oysters provided 95% confidence of detecting the virus that causes POMS at 2% prevalence in each estuary. A total of 640 oysters were analysed with no evidence of the virus that causes POMS detected.

NSW DPI now considers the protocol to be validated and that biosecurity in the Tasmanian hatchery is meeting the level of protection required for importation to POMS free areas of NSW. The temporary outward restrictions from the Clyde and Shoalhaven/Crookhaven rivers have now been removed. This project and surveillance program demonstrates best practise in management of a high-risk oyster pathogen, underpinned by robust science. Since resumption of supply in 2018, NSW DPI Aquaculture Production Reports are already showing a return to triploid Pacific Oyster production levels in these estuaries similar to those reported prior to the restrictions in 2016.

The edible oyster industry in NSW is dominated by cultivation of the native Sydney Rock Oyster (*Saccostrea glomerata*). The Pacific Oyster (*Crassostrea gigas*, also known as *Magallana gigas*) is a diversification option for the edible oyster industry in some NSW estuaries, comprising 4.8% of total oyster production in NSW (2019-2020). The Pacific Oyster is a non-native species to NSW, with the potential to proliferate and cover cultivated oysters, infrastructure and estuarine foreshore areas. Given this, cultivation of the species in NSW is mostly comprised of hatchery produced triploid (functionally sterile) Pacific Oysters. The NSW industry has a strong preference to source these oysters from Tasmanian hatcheries.

The Pacific Oyster is susceptible to POMS, a seasonal disease caused by the highly virulent Ostreid Herpesvirus microvariant-1. POMS can cause acute mortality of up to 100% in cultivated crops in areas where it is known to occur in NSW. Viral replication and clinical disease in NSW require sustained water temperatures of at least 21°C. Juvenile three to six-month-old oysters are most susceptible to infection.

For further information, contact Ben Rampano, Aquatic Policy & Projects Officer, Animal Biosecurity, NSW DPI, Port Stephens on 0409 321 813.

Anthrax exclusions April 2021 to September 2021

There were no anthrax incidents during the two quarters April to September.

During the period there were 106 investigations of mortalities where anthrax was excluded as the cause of death. Of these, a total of 64 investigations were in cattle where alternative diagnoses included bloat, enteritis, metabolic disease, plant and lead poisonings, clostridial infections, septicaemia and internal parasitism. There were 37 investigations in sheep where alternative diagnoses included lactic acidosis, hypocalcaemia, internal parasitism, pneumonia and polioencephalomalacia. Two investigations in alpaca diagnosed *Clostridium botulinum* encephalomyocarditis respectively, and two investigations in goats, one of which was diagnosed as Haemonchosis. No alternate diagnosis was found for a single investigation in a horse.

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

Table 1: Number of investigations with alternate diagnoses for mortality investigations where anthrax was excluded

Species	Diagnosis	No. Investigations		
		ICT Used		
		No	Yes	Total
Cattle	Bloat		1	1
	Bloat, peritonitis, pneumonia		1	1
	Clostridial infection		1	1
	Clostridial infection - *Cl. chauvoei*		1	1
	Clostridial infection - *Cl. perfringens*		2	2
	Coccidiosis, septicaemia; blood poisoning; bacteraemia		1	1
	Enteritis		2	2
	Enteritis, calf diphtheria - *Fusobacterium necrophorum*, internal parasitism - Oesophagostomum, pneumonia		1	1
	Female foetus: Interstitial nephritis, non-suppurative, chronic, multifocal, mild Leptospira seropositive	1		1
	Hepatitis, clostridial infection - *Cl. perfringens*	1		1
	Hepatopathy, peritonitis, toxicity - fungus - aflatoxin - Aspergillus flavus - aflatoxicosis, toxicity - pyrrolizidine alkaloid - *Heliotropium* spp		1	1
	Hepatopathy, toxicity - diterpenoid glycoside - *Cestrum parqui*		1	1
	Hypocalcaemia		1	1
	Hypomagnesaemia		3	3
	Malnutrition, arthritis, hypomagnesaemia, internal parasitism - *Fasciolia*, internal parasitism - general		1	1
	Mannheimia haemolytica, pneumonia		1	1
	Pestivirus (BVDV type 1) - Persistently infected, Bovine virus diarrhoea/mucosal disease (BVDV type 1)		1	1
	Pneumonia		1	1
	Salmonellosis	1		1
	Sporadic bovine encephalomyelitis		1	1
	Toxicity - cardiac glycoside - *Bryophyllum* spp		1	1
	Toxicity - diterpenoid glycoside - *Cestrum parqui*		1	1
	Toxicity - lead		1	1
	Toxicity - ptaquiloside - *Cheilanthes sieberi*		1	1
	Trauma		1	1
	Yersinia pseudotuberculosis	1		1
		4	30	34
Cattle Total		8	56	64
Sheep	Bloat		1	1
	Bloat, Chlamydiosis - *Chlamydophila pecorum*		1	1
	Calf diphtheria - *Fusobacterium necrophorum*, Coccidiosis		1	1
	Clostridial infection - *Cl. perfringens*, hypocalcaemia		1	1
	Clostridial infection - *Cl. perfringens*, internal parasitism - general		1	1
	Enteritis		1	1
	Hepatitis, calf diphtheria - *Fusobacterium necrophorum*		1	1
	Histophilus somnus, internal parasitism - Haemonchus, internal parasitism - Trichostrongylus		1	1
	Hypocalcaemia		2	2
	Hypocalcaemia, hypomagnesaemia		1	1

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Species	Diagnosis	No. Investigations		
		ICT Used		
		No	Yes	Total
	Hypocalcaemia, ketosis, internal parasitism - general		1	1
	Internal parasitism - Haemonchus		2	2
	Lactic acidosis		2	2
	Mannheimia haemolytica		1	1
	Mycoplasma ovis		2	2
	Pasteurella multocida, pneumonia, abscess - *Trueperella pyogenes*	1		1
	Pneumonia, abscess - *Trueperella pyogenes*	1		1
	Polioencephalomalacia	1		1
	Red gut		1	1
	Salmonellosis		2	2
	Septicaemia; blood poisoning; bacteraemia		1	1
		2	9	11
Sheep Total		**5**	**32**	**37**
Grand Total		**16**	**91**	**107**
Camelid	*Clostridium botulinum*	1		1
	Encephalomyocarditis		1	1
Camelid Total		**1**	**1**	**2**
Goat	Internal parasitism - Haemonchus		1	1
	Unknown		1	1
Goat Total			**2**	**2**
Horse		1		1
Horse Total		**1**		**1**

Lead Toxicity Exclusion in Weaner Sheep

During August 2021, 30 from 500 lambs suddenly developed ataxia, convulsions progressing to mortalities when being mustered for drenching on a property near Inverell in Northern NSW.

The case involved a single mob of cross-bred weaners that had been grazing a paddock of lush oats. They had also been receiving a grain supplement, but this had run out the week prior to this incident. The weaners had received 2 doses of 5 in 1 with the second dose about 4 weeks prior.

Weaners presented in several ways. Some were walking around with a swaying gait and ataxia. A number of these appeared blind or at least with central nervous system depression. Others were reluctant to stand and remained in sternal recumbency even when encouraged to stand. The worst affected were in lateral recumbency, with rigid limbs and paddling. Two of the worst clinically affected weaners were taken aside for blood sampling and post mortem. No gross pathology was noted, and a selection of samples were collected for anthrax exclusion, lead levels, biochemistry, and histopathology.

While awaiting results several actions were taken. As clostridial disease and overwhelming of vaccination protection resulting in Focal Symmetrical Encephalomalacia was a possibility, all weaner age sheep received a 5 in 1 booster. Calcium and magnesium supplements along with hay were made available in the paddock where the weaners were returned.

Initial laboratory results excluded both anthrax and lead toxicity. Blood calcium levels in the 2 weaners sampled was 1.23 and 1.21 mmol/L. The normal low end of normal is 2.12 mmol/L. Histopathology did not identify any changes in the tissues examined.

Based on serum biochemistry, a diagnosis of hypocalcaemia was made.

Factors contributing to the severity of the clinical signs included the high digestibility and moisture content of the oats, previous grain supplementation without added calcium, and exhaustion of limited available calcium in circulation.

This investigation provided the owner of the sheep with a potential cause of the losses and actions to be taken to avoid future cases. It was also an opportunity to sample clinically affected animals to exclude lead toxicity and anthrax.

For more information contact Andrew Biddle, Northern Tablelands Local Land Services, Inverell on 0427 825 725.

Skin lesions in a border disease positive lamb

The owner of a flock of self-replacing composite ewes with a history of Border disease reported that one lamb had skin lesions suggestive of scabby mouth (contagious ecthyma, orf). This lamb was from the tail of mob of 450 September 2020 drop composite lambs that were treated with moxidectin, vaccinated with clostridial 5:1 and weaned in March 2021. The lambs were weaned into ryegrass, clover pastures with some saffron thistles. The lambs were not vaccinated against orf. The owner noticed the skin lesions on the nose and lower limbs on 26 June when the lamb died. It was kept in cool storage for two days prior to examination.

The carcase was emaciated, weighing approximately 20kg with a hairy fleece. There were several pink hairless 1-2cm diameter ulcers from the coronary band to the hock on all legs. One ulcer on the coronary band was full skin thickness, while the others were flat and superficial. There were also shallow ulcers on lips and strip of raw ulcerated skin 5 by 3 cm down the bridge of the nose (Image 1).

Swabs of the lip and coronary band ulcers were submitted in phosphate buffered gelatin saline (PBGS). Thoracic fluid, tissues in formalin, chilled liver and lung along with samples for histopathology. While foot and mouth disease (FMD) and vesicular stomatitis (VS) exclusions were requested this case was regarded as a low risk FMD exclusion. There were no recent sheep introductions and the owners and visitors had not travelled overseas recently. There was virtually no access to feral pigs. The alternative and most likely diagnoses were Border disease and/or orf.

Samples of thoracic fluid and swabs from the lip and feet lesions tested negative for FMD (NSW DPI EMAI Laboratories and Australian Centre for Disease Preparedness (ACDP)) and VS (ACDP) by real-time PCR. These samples were also negative for FMD and VS(ACDP) by virus isolation. Swabs from the feet lesions were negative for orf virus by electron microscopy. The thoracic fluid tested negative for pestivirus antibodies by agar gel immunodiffusion (AGID) but was positive for pestivirus antigen by pestivirus antigen capture ELISA (PACE).

These results confirm that the lamb was congenitally infected with Border disease virus and was immunotolerant to the virus.

Border disease is caused by a pestivirus (Flaviviridae) infection of the foetus in early pregnancy (16-85 days of gestation). Infection of naïve, pregnant ewes can lead to placentitis, foetal death and abortion. Affected lambs are undersized with a hairy coat and may exhibit a variety of signs including ulcerations, neurological symptoms and skeletal defects. Surviving hairy shaker sheep remain infected and excrete the virus throughout their lives. Most, but not all, die in the first six months of life.

For more information, please contact Bruce Watt, Senior District Veterinarian, Central Tablelands Local Land Services on 0428 935 559.

Figure 2: Closer view of ulcerated, crusty nose in Border disease positive lamb.

Figure 1: Ulcerated, crusty nose in Border disease positive lamb.

Figure 3: Ulcerated skin from coronary band to fetlock right front leg.

Mother of millions toxicity in heifer and anthrax exclusion

In late June a farmer from South west Sydney contacted the District Veterinarian to report the sudden death of a yearling heifer. The heifer had been moved from a nearby property with 11 others just two weeks prior to the incident. Two months prior to move, the animals' owner excavated a small dam on the property to facilitate water for the livestock. Cattle were grazing mixed native pasture, including Danthonia, Microaena, and Red grass. Annual vaccination and drenching status were up to date.

On arrival at the property the carcass appeared bloated with a bloody discharge oozing from the anus and nostrils. Anthrax was considered unlikely as the property is not located in an anthrax area, however, an anthrax Immunochromatographic test (ICT) was performed, which was negative. A necropsy examination was performed and samples (fresh and fixed) for laboratory examination were collected from internal organs. The major post mortem findings were severe ulceration on the abomasal wall with signs of internal bleeding and epicardial haemorrhages.

Given the post mortem lesions, cardiac glycoside toxicity was added to the differentials. A paddock walk revealed many Mother of Million plants next to the dam, with clear signs of grazing.

Histopathology revealed a subacute cardiomyopathy, acute periacinar hepatic necrosis and severe abomasal mucosal haemorrhages. Heart muscle damage and gastrointestinal irritation are primary effects of cardiac glycosides, with periacinar hepatic necrosis occurring secondary to heart failure related hypoxia. Following a discussion with the pathologist, a diagnosis of cardiac glycoside toxicity was made. The source of the cardiac glycoside was flowering *Bryophyllum* sp (Mother of Millions).

Bryophyllum sp was introduced to Australia from Africa as a garden plant and has become a serious weed on the coast and the northwest slopes and plains of NSW. The plant flowers between May and October (depending on species). Most livestock poisoning occurs as the flower heads are more toxic than the leaves and stems. Bryophyllum spp contain cardiac glycosides of the bufadienolide type. These toxins cause heart arrhythmias leading to ventricular fibrillation and arrest.Gastrointestinal tract ulcers occur, especially in the abomasum and on the leaves of the omasum. Ulcers on the omasal pillars into the abomasum are pathognomonic. There is usually a mucoid, black, moderate diarrhoea as a result of ulceration. Affected cattle are often staggering. Sudden death on moving, due to a cardiac episode is commonly reported. Acutely affected cattle may die within 5 days or up to 3 weeks post removal, due to the rumen becoming atonic and then functioning again as toxin wears off.

Naïve cattle are attracted to the flowers, but not the plant. Pastures that have been overgrazed or overstocked can predispose livestock to eating anything they can, especially if they are hungry. Several factors can lead to livestock becoming poisoned from eating toxic plants. Producers should evaluate their pastures to determine if adequate forage exists before introduce livestock on to the paddock. If there is a lack of adequate forage, livestock may be more likely to eat poisonous plants.

For further information, contact Aziz Chowdhury, District Veterinarian, Greater Sydney Local Land Services on 0427 284 797.

Figure 2: Yearling heifers.

Figure 3: Haemorrhages in the abomasal mucosa.

Figure 1: *Bryophyllum* sp. (Mother of Millions) plants.

Figure 4: Epicardial haemorrhage.

Low Pathogenic H10N7 Avian influenza confirmed in backyard poultry

Diagnostic testing for a disease investigation into Avian Influenza (AI) in a flock of non-commercial backyard chickens returned a positive result and a team of emergency response, laboratory and operational staff took rapid steps to identify if this was an EAD incident.

In July 2021, a private veterinarian in the southern tablelands of NSW directed a poultry owner to contact their District Veterinarian (DV) after they reported mild respiratory symptoms of 1- to 2-weeks duration and associated mortalities. The flock consisted of 120 to 150 heritage breed chickens confined to a penned area. There were 60 – 80 guinea fowl that had a large free flight range, and access to chicken pen. Two pet geese also ranged the property. Several management issues were noted relating to stocking density, manure management and biosecurity practices. The water source for the domestic poultry was a large dam which was frequented by many wild ducks. Wild ducks and other wild birds comingled with the chickens in the pen, congregating around feed and watering points. The property is a small holding and the poultry are kept for heritage and rare colour variant breeding and home consumption of eggs. A mid-size commercial free-range egg farm is located 7km away.

Roughly 10% of the chickens were showing clinical signs including coughing, sneezing, rattly breathing sounds, watery eyes, ruffled feathers, huddling, lethargy or diarrhea. The owner reported decreased feed consumption and egg production. The mortality rate of the chickens was about 8%. The guinea fowl and geese had no clinical signs and no mortalities.

An autopsy was performed on a rooster which revealed massively swollen kidneys and visceral urate deposition. Fresh kidney and spleen, and formalin fixed kidney, liver and heart were collected from the deceased bird. Oropharyngeal and cloacal swabs were collected from the deceased rooster and three live, clinically affected chickens transferred to phosphate buffered gelatin saline (PBGS) viral transport media. . The property visit was on a Friday, and the samples were sent via courier on the Sunday, arriving at the Elizabeth Macarthur Agricultural Institute (EMAI) on the Monday morning.

Initial PCR screening excluded Infectious Laryngotracheitis and Newcastle Disease and was positive for Influenza A on three of the four swabs submitted. The NSW Chief Veterinary Officer (CVO) was notified. The CVO and her team at NSW Department of Primary Industries (NSW DPI) directed the DV to promptly collect further samples. In parallel, the Virology Laboratory at EMAI worked to further characterise the Influenza A and was able to exclude subtypes H5 and H7 by PCR later that same day.

The DV, with a supporting biosecurity officer, collected tracheal swabs, cloacal swabs and blood samples from a further 14 chickens (a mix of symptomatic and asymptomatic) and 2 guinea fowl (both asymptomatic). A tracheal swab and cloacal swab were also collected from one goose.

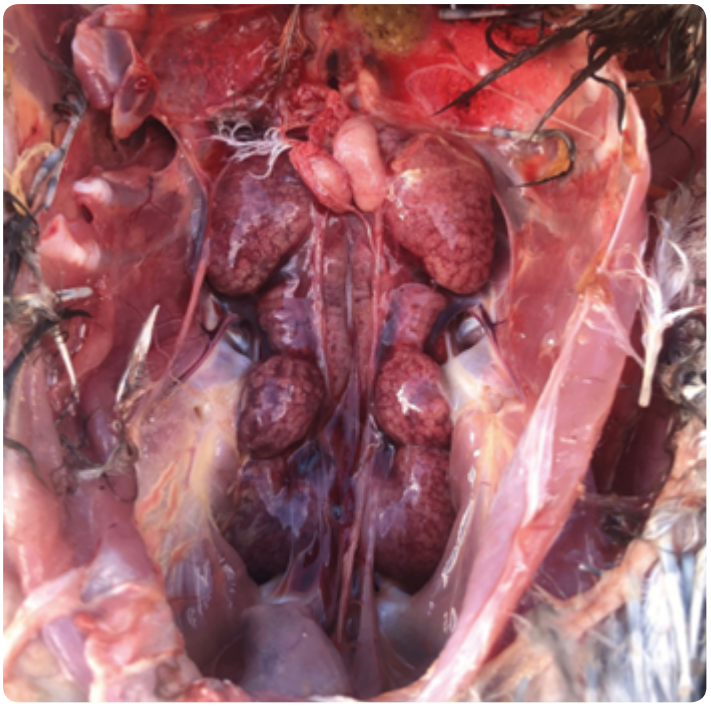

Figure 1: Massively enlarged kidneys in a deceased rooster that tested positive for Avian Influenza.

AI Antibody ELISA showed that all the chickens tested, and the two-guinea fowl tested, had been exposed to AI. Curiously none of the 34 swabs collected on the second site visit resulted in a positive Influenza A matrix PCR result.

Within 48 hours of the initial positive, EMAI had been able to sequence the virus as H10N7 and this was subsequently confirmed at the Australian Centre for Disease Preparedness (ACDP) as Low Pathogenic H10N7. This meant that the incident would not be treated as an emergency animal disease outbreak.

The DV returned to the property again to complete an AI field investigation questionnaire and gather more epidemiological information. In consultation with NSW DPI a risk assessment determined that a response was not necessary and identified the need for education to improve biosecurity practices. NSW Health was notified and spoke with the poultry owner regarding the human health risks of zoonotic H10N7. The DV prepared a report for the poultry owner. Improvements in biosecurity practices, the exclusion of wild birds, and reduction of potentially immunosuppressive stress factors were recommended.

H10N7 has been detected in Australian before. An outbreak in March 2010 resulted in conjunctivitis and minor upper respiratory tract symptoms in abattoir workers processing birds from the affected farm. In that outbreak respiratory signs were absent in the flock, but the mortality rate increased, and egg production decreased, for a period of 8 – 14 days.

Australia remains free of HPAI in commercial poultry.

For further information, contact Lou Baskind, District Veterinarian, South East Local Land Services, Braidwood on 02 4842 3800.

Suspected Kangaroo Gait in composite ewes following a TSE exclusion

Transmissible spongiform encephalopathy (TSE) was excluded from a mob of composite ewes displaying neurological signs at a property in the western Murray region in July 2021.

The producer initially noticed an abnormal lameness in around 5-10 ewes out of a mob of 500 ewes with lambs at foot as they were brought up to the yards for lamb marking. They engaged their local District Veterinarian to come and inspect the ewes.

The ewes were of mixed age and had been grazing mixed grass pastures containing predominately barley grass, some rye grass, and other natural grasses.

On clinical examination the affected ewes were observed to be ataxic with knuckling at the fetlocks in the forelimbs. Proprioception of the hind feet appeared abnormal due to a hoppy hindlimb gait. The clinical signs worsened as more pressure was placed on the ewe to move, resulting in the affected ewes regularly falling in the forelimbs. The ewes were able to weight bear on all feet when stationary but with a tucked-up and crouched hindlimb stance. No swelling or abnormalities were seen on examination of the joints and feet. The cranial nerve exam of affected ewes appeared normal.

The affected ewes appeared to be in lower body condition (2/5) than the rest of the mob (average 2.5-3/5). No clinical signs were observed in the lambs and they appeared to be in good body condition.

One severely affected ewe was euthanised and a post-mortem was performed. Temperature of the ewe prior to euthanasia was 39.7^{0}C. Gross post-mortem findings appeared normal, except for some thickening of the small intestines and enlargement of the mesenteric lymph nodes. Fresh and fixed samples of the brain, cervical spinal cord, thoracic spinal cord, lumbar spinal cord, and various other tissue samples were collected and submitted to the Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) as part of a TSE exclusion. A video showing the clinical signs was also emailed through to the pathologist.

Biochemistry results from blood collected showed increased AST (253 U/L, ref. 0 – 130 U/L) and CK (1066 U/L, ref. 0 – 300 U/L) enzymes. There were no significant findings on haematology. On histology there were no significant lesions in the neurological tissues examined suggestive of TSE.

Unfortunately, no diagnosis was able to be made from the samples submitted to the laboratory. However, following further discussion with the pathologist, the history of lactating ewes and the clinical signs observed were thought to be consistent with "Kangaroo Gait".

Kangaroo Gait is a peripheral neuropathy affecting the forelimbs of lactating ewes. Affected ewes present with knuckling of the forelimb fetlocks combined with an unusual propulsive hindlimb gait. It is a condition that can occur in older ewes from larger meat breeds. Ewes which are affected show clinical signs 3 to 6 weeks post lambing and are typically feeding multiple lambs. Affected ewes will generally recover once the lambs are weaned.

Laboratory diagnosis of Kangaroo Gait is generally made on histopathology lesions of Wallerian degeneration of the peripheral nerves (particularly the radial nerve), and denervation atrophy of the forelimb muscles (particularly the triceps muscle). However, the aetiology and pathogenesis of the syndrome is still unclear.

Unfortunately, samples of the radial nerve or triceps muscle were not collected in this case. An opportunity to perform a follow up post-mortem for collection of these samples to confirm the provisional diagnosis was also not provided.

Following further discussion with the producer, the producer reported that he drafted off approximately 25 to 30 ewes from the mob showing clinical signs. Weaning occurred at shearing and around five affected ewes died after being shorn. Around five affected ewes were also sold as cast for age ewes. Although actual numbers of those recovered verses those still with clinical signs were not known at the time of writing, the producer reported that he thought some of the ewes had recovered following weaning and some still had the hoppy gait.

Figure 1: Ewe showing tucked up and crouched stance when stationary.

For further information, contact Katelyn Braine, District Veterinarian, Murray Local Land Services, Deniliquin on 03 5881 9900.

African Swine Fever and Classical Swine Fever exclusion for sudden pig mortalities

Sudden deaths and bruising of two out of 12 finisher pigs were investigated on a property in the Gilgandra region in August. The pigs were unvaccinated and had previously been free range until they were housed for sale 1 week prior to the event. No clinical signs were noted by the landholder prior to death. Samples were taken by a private veterinarian and sent to Laboratory Services at Elizabeth Macarthur Agricultural Institute (EMAI) for testing to exclude African swine fever (ASF) and Classical Swine Fever (CSF).

Both carcasses displayed extensive bruising, skin discolouration and swollen submandibular lymph nodes/neck. Petechial haemorrhages of the heart and renal pelvis were noted on post mortem but no gross haemorrhage was present.

The attending veterinarian noted three other pigs in the pen were starting to show signs of malaise and diamond like bruising on their skin during her attendance at the piggery.

PCR testing on blood samples were negative for ASF and CSF. Histological lesions were mild. Changes observed in tissues were an increased number of inflammatory cells inside vessels, indicative of a response to an inflammatory process, and haemorrhage, which is suggestive of shock.

The skin lesions noted on the live pigs were pathognomonic for Erysipelas. They were treated with procaine penicillin and made a full recovery.

For further information, contact Kate Atkinson, District Veterinarian, Central West Local Land Services on 0407 716 816.

QX disease in Port Stephens

QX disease was confirmed by cytology and Polymerase Chain Reaction at low prevalence in Sydney Rock Oysters in Port Stephens for the first time on 27 August 2021.

The edible oyster industry is the largest and oldest aquaculture industry in NSW, valued at $58.2M in 2019-2020. The industry is predominantly based on cultivation of the native Sydney Rock Oyster (*Saccostrea glomerata*), with the species comprising 87% of total production in NSW. Port Stephens is the second largest oyster producing estuary in NSW, comprising estuarine lease cultivation, the largest commercial oyster hatchery in NSW, the dedicated NSW DPI oyster research hatchery and commercial oyster nursery facilities. Port Stephens and Myall Lakes collectively have a water area of approximately 300km^2, making them one of the largest estuarine waterways in NSW. The total catchment of the waterway is some 3700km^2.

One of the biggest challenges facing the Sydney Rock Industry is QX disease, caused by the paramyxid parasite *Martelia sydneyi*. QX (Queensland unknown) is a seasonal disease with a complex lifecycle that involves at least one intermediate host, currently understood to be the polychaete worm *Agalophamus* (formerly *Nephtys*) *australiensis*. The parasite enters the oyster through the gills and palp. If it progresses to cause disease, the parasite divides and proliferates. It then migrates to the digestive gland which surrounds the oyster intestine. There it undergoes further development and multiplication to produce spores. Spores damage and block the digestive gland, leading to prolonged starvation and eventual death of the oyster. It is this process of sporulation that results in clinical QX disease. QX infection typically occurs between January to April in areas where it is known to occur in NSW. QX can result in mass mortality, where cultivated stock losses are usually observed from autumn to early winter.

Following extended periods of high rainfall and reduced water quality, some oysters in Port Stephens were observed in August 2021 to be in poor condition, appearing translucent or "watery" and showing signs of slow growth. In some oysters, the digestive gland appeared to be a light tan colour instead of the usual dark brown. On 27 August 2021, laboratory examination at NSW DPI Elizabeth Macarthur Agricultural Institute confirmed the presence of clinical QX sporulating organisms by cytology in 3 of 12 samples collected from around the mouth of the Karuah River. The agent M. sydneyi was confirmed by PCR in 14 of 24 samples. Histopathology was also completed, confirming intracellular protozoa consistent with *M. sydneyi* in 11 of 24 slides. Until this detection, clinical QX has never been confirmed in Port Stephens.

On 9 September 2021, NSW DPI conducted surveillance across Port Stephens oyster growing areas to determine the extent of spread of the outbreak. The port was divided into six zones and oyster samples that appeared to be exhibiting poor growth were targeted. Oysters showing signs of having been held for an extended time in each zone were targeted, to maximise the chances of detecting QX from oysters in each zone rather than those that may have already been infected and moved into the zone from another affected area. 10 oysters were collected from 3 sites within each zone, making a total of 30 oysters per zone and a grand total of 180 oysters collected across the port. QX was confirmed at very low prevalence, with a total of 7 positive oysters found across zones 1, 2 and 3. There was no evidence of QX in zones 4, 5 and 6.

The QX status of Port Stephens remains under investigation. From what is known of QX infection dynamics, these positive results have been

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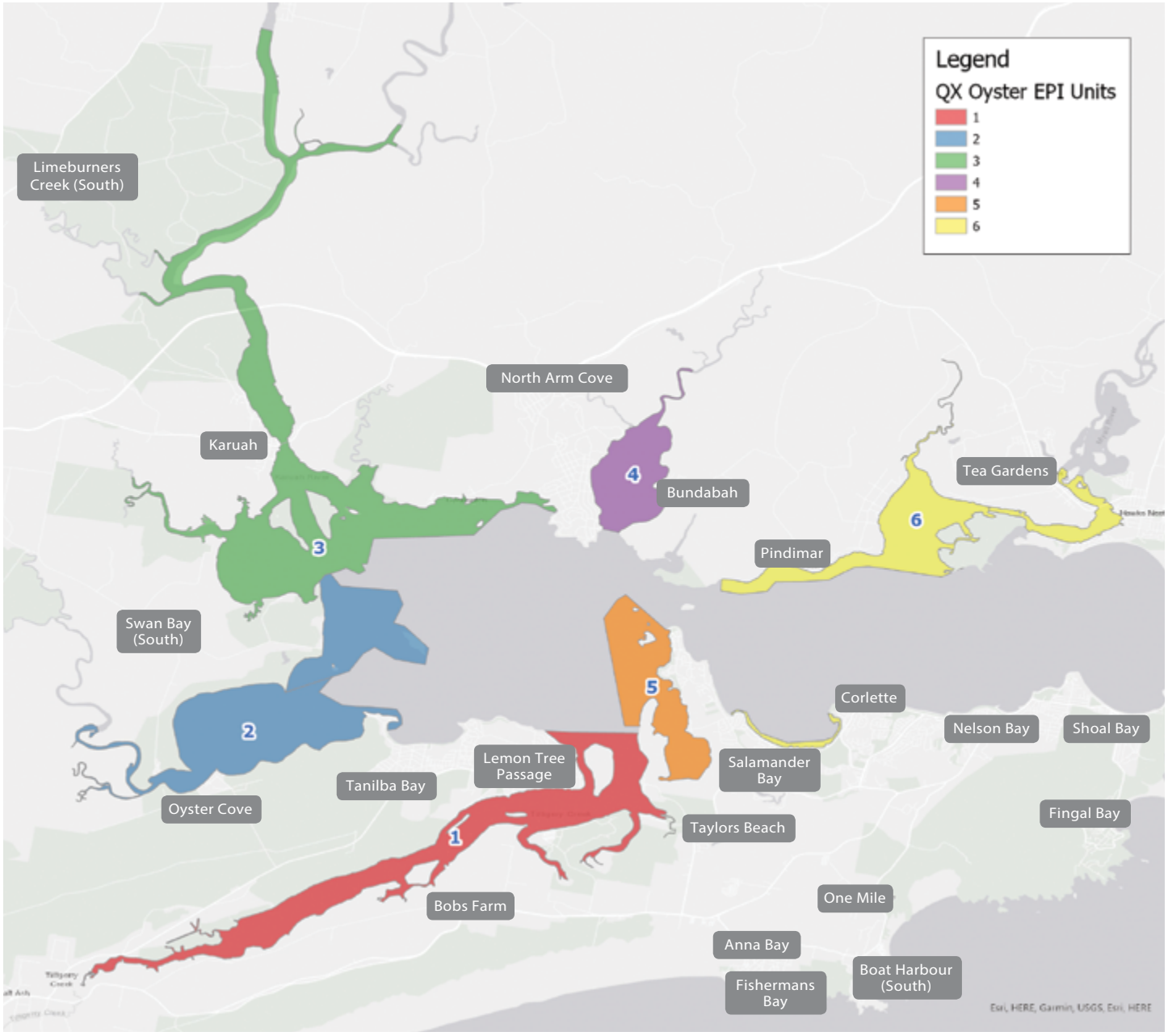

Figure 1: Zones for September 2021 QX surveillance in Port Stephens. Zones 1, 2, and 3 were positive.

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detected late in the season for when QX disease is normally observed. The true infection prevalence for Port Stephens cannot be determined from this sampling alone and will require further surveillance in autumn of 2022. All seafood on the market remains safe to eat. Oysters in the marketplace must comply with stringent food safety standards of the NSW Shellfish Program. Oyster producers who find anything unusual are urged to call the Emergency Animal Disease hotline on 1800 675 888.

For further information, contact Ben Rampano, Aquatic Policy & Projects Officer, Animal Biosecurity, NSW Department of Primary Industries, Port Stephens on 0409 321 813.

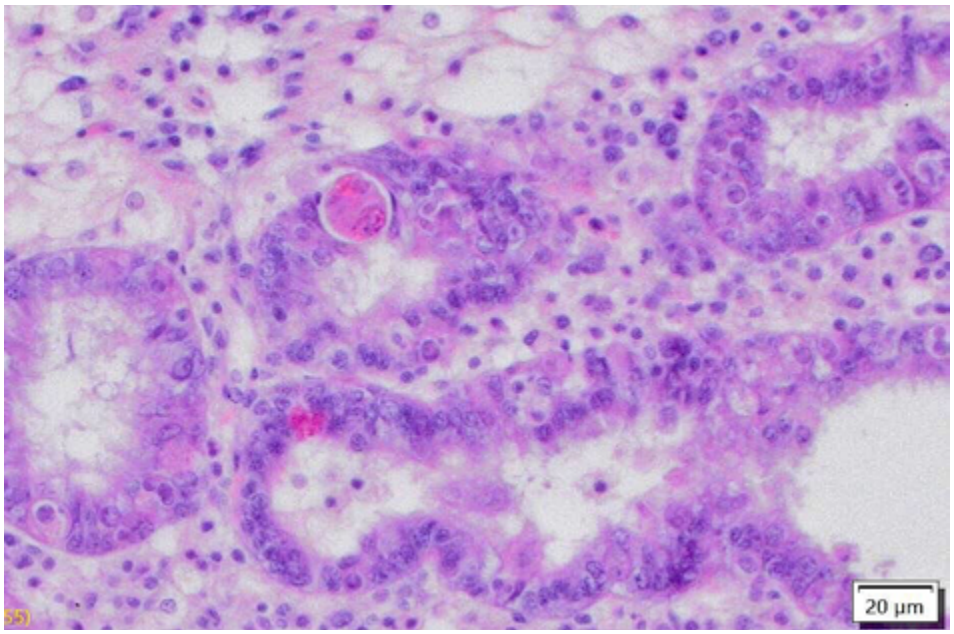

Figure 2: Developing QX sporonts in the digestive gland of a Sydney Rock Oyster" photo courtesy Anne Jordan (NSW DPI EMAI).

Getting information on animal diseases

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

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

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

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

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

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

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

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