

NEW SOUTH WALES ANIMAL HEALTH SURVEILLANCE

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

April - June 2009 • Number 2009/2

Theileriosis in cattle: update

The previous issue of *Animal Health Surveillance* (see http://www.dpi.nsw.gov.au/__data/assets/pdf_file/0006/278844/ahs-2009-1.pdf) provided information on theileriosis in NSW. We have since established a project called 'Better understanding of theileriosis in NSW cattle' to encourage veterinarians to submit samples to Elizabeth Macarthur Agricultural Institute (EMAI) in cases where theileriosis is considered among the differential diagnoses. The project provides funding for laboratory charges for theileriosis testing; in most cases this involves packed cell volume (PCV) testing and smear examination.

During the last quarter, 21 submissions were received at NSW DPI veterinary laboratories where *Theileria* testing was performed. Most submissions came from coastal properties, with a few from the tablelands. In six of the submissions, veterinarians indicated that animals had been introduced to the farm from other districts. *Theileria* was detected in 14 of the submissions, and benign theileriosis was diagnosed in all 10 submissions in which the animals had clinical signs related to anaemia. Tick fever was not diagnosed in any of the submissions, although veterinarians should continue to include it as a differential diagnosis where cattle have anaemia. If tick fever is a

possibility, capillary blood (see the Vet Lab Submission Manual at http://www.dpi.nsw.gov.au/agriculture/vetmanual/specimens-by-disease-syndrome/diseases_of_livestock/tick_fever_of_cattle), as well as smears of tail or jugular vein blood and blood in EDTA, should be submitted.

The latest reports indicate that pyrexia is commonly seen in early cases. Although blood has occasionally been detected in the urine on urinary dipsticks, 'redwater' (haemoglobinuria) is not a feature of the disease. Typically, at the time the property owner seeks veterinary help, one or more affected animals has a PCV of less than 15.

NSW DPI is convening a workshop in September 2009 to bring together interested scientists and cattle industry representatives to review theileriosis and its potential impacts on production and market access. It is expected that the workshop will help to identify and prioritise the actions required to manage the disease.

**For further information contact
Graham Bailey, NSW DPI Orange,
on (02) 6391 3870.**

Lameness and sudden death in cattle

Histophilus somni was confirmed as the cause of sudden death in a yearling steer in the Bylong Valley, near Mudgee, in an incident in which four others died with lameness. The

In this issue!

Theileriosis in cattle: update	1
Lameness and sudden death in cattle	1
Cattle with coughing and ill thrift	2
Suspected nitrate poisoning in cattle	3
Bat positive for lyssavirus	3
Atypical chorioretinitis in cattle	3
Polioencephalomalacia in Merino ewes	4
Not phosphorus deficiency	4
Pneumonia in sheep	4
Poison peach toxicity in goats	5
Vitamin A deficiency in North Coast poultry	5
Plant toxicities in southern NSW	5
Bovine herpes virus 1 balanoposthitis survey	6
Metabolic disorders in southern NSW	7
Anthrax exclusions	7
Cape tulip poisoning of cattle	7
Amazing progress in footrot control	7



NSW DEPARTMENT OF
PRIMARY INDUSTRIES

bacterium was isolated from the liver, joints and spinal fluid of the dead steer. The steer was observed lame in the near hind leg the night before and found dead the next morning. Gross pathology included extensive haemorrhages, especially in the anterior of the carcass, pale patches in the liver (especially in the caudal lobes), injection and inflammation of the joint synovial membranes, and necrosis of the caudal brain.

The laboratory findings were suppurative encephalitis and septicaemia caused by *Histophilus somni*.



Pale areas in a liver infected with Histophilus somni. Photo courtesy D Gardner.

One possible predisposing contributing factor identified by the district veterinarian was rock fern poisoning. Laboratory blood tests showed a low white blood cell count of $2.8 \times 10^9/L$ (normal 5 to $10 \times 10^9/L$) and low platelet count of $64 \times 10^9/L$ (normal 100 to $800 \times 10^9/L$); rock fern was present in the paddock.

For further information contact David Gardner, DV Central North Livestock Health and Pest Authority (LHPA), on (02) 6372 1866.

Cattle with coughing and ill thrift

In the Merriwa district, a Wagyu cow and calf were found dead in the paddock during mustering for calf marking. Most of the cows and calves were coughing, and some calves were scouring.

Temperatures ranged from 37.7 to 40°C in the eight head examined and sampled from the mob. Condition scores ranged from 1 to 3, with all calves and many cows clinically showing ill thrift and some showing evidence of bottle jaw. The cough was mild and there was scant nasal discharge.

Laboratory testing confirmed very low selenium status of the cattle, with levels of glutathione peroxidase from 4 (in calves) to 17 (in cows) U/g Hb (normal 40 to 300 U/g Hb). The cattle were running on known selenium-deficient country; supplementation this year had been omitted as a cost-cutting exercise following a very disappointing annual stud sale. Anthrax, benign theileriosis and liver fluke were excluded as possible causes of the deaths and ill thrift.

The coughing syndrome spread to two adjoining mobs, totalling 161 head, running along a creek. A sick, scouring, calf with ill thrift was sacrificed to further investigate the cause and to exclude fog fever. (Note that *Animal Health Surveillance 2007/2* reported a case of fog fever, an acute respiratory distress syndrome caused by brassica consumption and associated with increased tryptophan levels in the rumen.) On laboratory examination the bronchial epithelium contained minimal infiltrations of lymphocytes and neutrophils. The submucosa had moderate perivascular and interstitial accumulations of lymphocytes and plasma cells, but there was no evidence of interstitial oedema, which is characteristic of fog fever.

The cattle responded to supplementation with selenium and a vitamin complex of A, D and E. Rotavirus was isolated as a possible cause of the scour; this is unusual in calves of weaning age, but they were no doubt immunocompromised by the low selenium and the low protein levels, as

evidenced by the bottle jaw. None of the remaining 700 cattle in the herd kept in isolation showed signs of coughing. Further investigations are under way to determine the cause of the respiratory disease, with parainfluenza 3 and/or bovine syncytial virus the chief suspects.

For further information contact
David Gardner, DV Central North LHPA,
on (02) 6372 1866.

Suspected nitrate poisoning in cattle

Nitrate poisoning was suspected in a mixed mob of female cattle grazing a fallow cultivation paddock containing a plentiful growth of wilting *Sonchus oleraceus* (sow thistle/milk thistle) near Warialda.

Within 24 hours of being placed on the sow thistle paddock, one 5-year-old cow died and a 5-month-old heifer was found in sternal recumbency showing severe dyspnoea. She died within a few hours. The aqueous humour was negative for nitrites on a field dipstick test. However, as sampling occurred more than 12 hours after death this result was not considered accurate. Anthrax was excluded by the laboratory.

The identification of *Sonchus oleraceus* was confirmed by the Royal Botanic Gardens, Sydney. It was the only palatable feed in the paddock and was in a wilted state, having been sprayed with a glyphosate mix several days before the entry of the cattle.



Sow Thistle. Photo courtesy John Gasparotto, DPI Image Library

Laboratory testing found a nitrate concentration of 1000 ppm in submitted samples of the plant. Although this concentration is marginally less than the potentially toxic level of 1200 ppm, variations in concentration of nitrates can occur in differing parts of a paddock and individual susceptibility to toxic nitrate levels vary. A review of available literature revealed no references to *Sonchus oleraceus* causing nitrate/nitrite poisoning, although it was suspected in this case.

For further information contact Ted Irwin,
Shaun Slattery and Libby Read, Northwest
LHPA, on (02) 6729 1528.

Bat positive for lyssavirus

A grey-headed flying fox was found hanging low in a tree at Padstow in Sydney and was 'rescued' without anybody being bitten or scratched. It was placid when being caught by the WIRES volunteer but became highly aggressive immediately afterwards, biting at the bars of the cage, vocalising every 30 seconds, and showing agitation when approached. The animal pierced its upper right lip with its canine tooth and continued to bite its lip while trying to bite the handler. It showed other behaviours indicative of neuropathology, appearing unable to decide whether it wanted to eat, urinate, groom its wings or attack, although attack appeared to be the strongest drive.

The bat was euthanased and sent to EMAI. Histopathology of the brain stem showed non-suppurative encephalitis. Brain samples were sent to AAHL where lyssavirus was isolated.

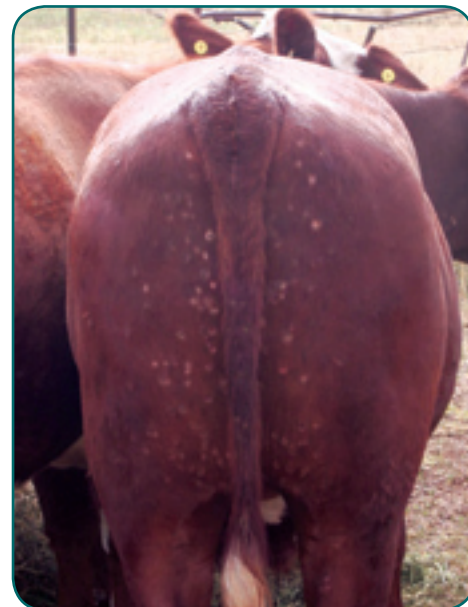
For further information contact Rory Arthur,
NSW DPI Orange, on (02) 6391 3608.

Atypical chorioptic mange in cattle

Chorioptic mange is a skin disease of cattle. It is caused by the mite *Chorioptes bovis* and usually begins on the legs, spreading to the scrotum,

tail and perineal area. It was recently diagnosed in a small herd of 16 adults and two calves near Narrabri. It usually causes intense pruritis (itching) and small skin nodules that exude serum, progressing to form crusts and scabs.

In this case, affected animals developed intense pruritis in association with nodular skin lesions, mainly in the region of the neck, perineum and upper hind legs. The nodules were raised from the surrounding skin and ranged in diameter from 5 to 20 mm. They did not show the characteristic scabbing, but they did show damage attributable to traumatic injury associated with pruritis.



Chorioptic mange. Photo courtesy S. Slattery.

The exotic condition known as lumpy skin disease, which is caused by a capripoxvirus, was excluded, as the cattle were not showing signs of systemic illness and pruritis is not generally a feature of the condition.

Individuals were not examined clinically and skin scrapings were not taken because the facilities were inadequate for these purposes, so treatment with a macrocyclic lactone back-liner was implemented. The cattle ceased rubbing within 2 or 3 days of treatment, and the owner reported that the nodules had disappeared within a week.

For further information contact Libby Read, DV Narrabri/Walgett, and Shaun Slattery, SDV Northwest LHPA, on (02) 6792 2533.

Polioencephalomalacia in Merino ewes

In May, four of 120 sheep on a property located near Dalgety developed nervous symptoms. The ewes were aged 18 to 24 months. They became recumbent in the paddock but were initially able to regain their feet. As the disease developed over 2 or 3 days there was pronounced head turning, flickering of the eyelids and twisting of the legs.

All sheep were in reasonable body condition despite the drought conditions.

One of the recumbent ewes was injected with vitamin B1 (thiamin). After two injections she was able to stand up again after being recumbent. This animal was euthanased and autopsied. The lungs, heart, liver, kidneys and small intestine were normal on histopathology. In the occipital cortex of the brain the changes were diagnostic of polioencephalomalacia (PEM). The meninges had moderate perivascular and interstitial accumulations of activated macrophages. Similar accumulations were noted in the adjacent grey matter.

PEM is known to be associated with thiamin deficiency as a result of thiaminase activity in the rumen. All sheep were moved into another paddock and no further cases were reported.

For further information contact Brian Hodge, DV South Coast LHPA, on (02) 6452 1122.

Not phosphorus deficiency

Recently, in the Braidwood area of the South East Livestock Health and Pest Authority, many cases of phosphorus deficiency have been diagnosed on properties with native pastures

that have never been treated with superphosphate.

One beef producer thought that his property was phosphorus deficient because his cows were showing ill thrift similar to that seen in clinical phosphorus deficiency; the producer called the district veterinarian to confirm his diagnosis. The district veterinarian took blood samples to determine the phosphorus, copper and selenium levels of the cattle. A liver fluke ELISA was also requested for all samples. At the laboratory, all the mineral levels were within the normal range. However, every animal had a positive test result for liver fluke.

Once drenched for liver fluke, the herd showed a marked improvement in condition.

For further information contact Bob Templeton, DV South East Livestock Health and Pest Authority, on 02 4842 2536.

Pneumonia in sheep

Two pregnant ewes with a 2-week history of being unwell and tailing the mob collapsed and died in the late afternoon near Tamworth after mustering. They normally grazed on improved pastures and did not have a recent history of drenching.

Post mortem examination of one revealed a thoracic cavity filled with yellow-grey turbid and odorous fluid. The left and right cranial lung lobes were dark pink, with diffuse, well circumscribed pale pink raised nodules about 5 to 10 mm in diameter. These felt firm in texture. The caudal lung lobes were a normal pale pink and sharply demarcated in appearance from the cranial and middle lung lobes. The cut surface of the cranial lobes revealed multiple yellowish-white, firm circular areas of suspected necrosis or abscess formation.

Laboratory examination showed extensive multifocal and coalescing

areas of necrosis in the affected areas, flooding of alveoli with inflammatory cells and cellular debris, and thickened pleura with fibrin and inflammation. Each of the large necrotic areas contained a centre of necrosis and colonies of coccobacilli surrounded by a zone of neutrophils, lymphocytes and plasma cells and occasional macrophages.

Streptococcus bovis was isolated from the lung, leading to a diagnosis of *Streptococcus bovis* pneumonia and effective exclusion of the contagious lung tumour called ovine pulmonary adenocarcinoma (OPA) or jaagsiekte (driving sickness) as the cause. OPA is absent from Australia and New Zealand and has been eradicated from Iceland but is otherwise the most common pulmonary tumour of sheep in many countries around the world. The OIE (World Organisation for Animal Health) reports that the disease is caused by a beta-retrovirus that cannot yet be cultured in vitro, but the virus has been cloned and sequenced.



Bronchopneumonia and hydrothorax. Photo courtesy Kylie Hardwicke and Emma Pilkington

For further information contact Bob McKinnon, SDV Central North LHPA Tamworth, on (02) 6762 2900.

Poison peach toxicity in goats

Poison peach toxicity was diagnosed as the cause of death in four dairy goats at Nimbin. Before death the goats had neurological symptoms, including stargazing and stumbling.

The goats were from a CAE (caprine arthritis encephalitis)-free accredited herd and were vaccinated for clostridial infections. They did not respond to intravenous injections of thiamin.

At necropsy the liver was swollen and laboratory examination revealed a severe acute hepatic necrosis (typical of poison peach toxicity) and normal brain tissues, ruling out polioencephalomalacia. Goats had access to scrub at the back of the property, where weeds such as poison peach were present. Although poison peach is commonly reported to be poisonous to goats it is not as common to attend a case and have it well supported by histopathology. Poison peach is also poisonous to sheep and horses.

For further information contact Matthew Ball, North Coast LHPA, on (02) 6621 2317.

Vitamin A deficiency in North Coast poultry

Vitamin A deficiency was diagnosed as the major factor in the death of 50 chickens near Murwillumbah. Deaths had occurred over a 3-month period. Bacterial pneumonia and *Salmonella* and *Capillaria* infection were also associated with the disease outbreak.

Clinical signs reported to the district veterinarian included swollen red and watery eyes, eyelids stuck together, red facial skin, gasping and then death within 2 or 3 days of showing signs. In addition, out of 30 eggs candled to be fertile at 3 or 4 days only six chicks hatched.

A post mortem examination revealed multifocal nodular lesions in the pharynx and oesophagus and yellow caseous exudates at the base of the trachea and in the lungs.

Histopathology demonstrated squamous metaplasia of the oesophageal and tracheal epithelium, multiple abscesses in the lung, extensive perihepatitis, and thickening of the crop epithelium, with numerous

tunnels occupied by operculated eggs of *Capillaria* worms.

The squamous metaplasia is typical of Vitamin A deficiency. *Salmonella typhimurium* type 3 was cultured from the liver.

The diet of the birds was found to be predominantly wheat-based. This is likely to be deficient in vitamin A. Infection with *Capillaria* worms is also reported to decrease effective absorption of Vitamin A. Vitamin A deficiency alone could have caused the ocular signs in these birds. Vitamin A deficiency combined with indoor housing of the birds in a dusty environment would have compromised lung function and predisposed to the bacterial pneumonia.



Cyanosis and eye occlusion in vitamin A deficiency. Photo courtesy Matt Ball.

For further information contact Matthew Ball, North Coast LHPA, on 6621 2317.

Plant toxicities in southern NSW

Acute phalaris poisoning caused the death of 20 out of 200 wethers in Hume LHPA in March. The sheep had been introduced to a paddock of short phalaris that had received heavy rain a few days beforehand. Low-level losses from phalaris poisoning were reported in the Albury district in April. Hungry sheep and very short phalaris were typical features of all cases.

Lupinosis was the cause of death in 22 out of 55 cows and two calves in Hume LHPA following introduction to lupin stubble

where sheep deaths from lupinosis had been confirmed earlier in the year. Signs in the affected cows included ill thrift, jaundice, polydipsia, abdominal swelling, recumbency and death. Histopathology indicated that heliotrope was likely to be a complicating factor.

In the Wagga district two cows died and another displayed signs of neurological disturbance, including aggressive behaviour, head rocking and circling, in a paddock where heliotrope was abundant. An affected cow was down and as the district veterinarian lifted its tail to collect a blood sample it sprang to its feet with intent to kill! Fortunately the vet made it back to the car, which the cow proceeded to half demolish in anger. Remarkably, this cow recovered. Lupins had also been fed. High ammonia levels in the aqueous humour of one of the dead cows supported a diagnosis of hepatic encephalopathy.

In the Riverina LHPA four lambs died and seven were affected out of a mob of 150 cross-bred lambs introduced from the Central Tablelands 6 months earlier. The affected lambs lost condition and became blind and jaundiced before death. Post mortem examination revealed an orange, enlarged liver with a lobular pattern and irregular nodules. The kidneys were brown and mottled with radial striations of the cortex. Histopathological changes were consistent with poisoning from *Panicum* spp. or *Tribulus terrestris*.

In Hume LHPA, 35 young Damara-cross sheep died and the remaining 160 were suffering from photosensitisation and ill thrift. The sheep had been introduced from the Western Division a month earlier on agistment and had not been inspected regularly. They had been grazing heliotrope and panic grass. Post mortem findings included jaundice, enlarged fibrous liver, enlarged dark coloured kidneys and dark urine. Histopathology was consistent with ingestion of *Panicum* spp. and *Tribulus*

spp., resulting in steroidal sapogenin crystalloid depositions within the hepatocytes and intrahepatic bile ducts. Mortality in chickens was investigated in the Lachlan LHPA. Out of a flock of 24 400 birds, 850 died and about 10% were sick. Affected birds had breathing difficulties and appeared comatose, with a head twitch when close to death. The droppings were wet and malodorous and contained blood. Post mortem examination revealed marked ascites consistent with a chronic hepatopathy such as that caused by pyrrolizidine alkaloids. Contamination of feed was suspected. A supply of grain from the Liverpool Plains was deemed to be the source of contamination with heliotrope seed. The grain was harvested post-flood, and the suspicion is that this allowed the grain to be at the same height as the heliotrope, thus resulting in contamination. Avian influenza and salmonellosis were excluded.

For further information contact Steve Whittaker, SDV Hume LHPA, on (02) 6040 4210 or Sarah Robinson, NSW DPI, on (02) 6938 1967.

Bovine herpes virus 1 balanoposthitis survey

Further testing of bulls was carried out in the Hume region. In the Gundagai district five out of five Angus bulls with penile lesions tested positive for BHV-1 (serology). Four of the five bulls had been used to join approximately 180 heifers. The pregnancy rate for the heifers was 75%. On another property in the Gundagai district seven out of seven bulls tested positive for BHV-1. Four of the seven had penile lesions. The pregnancy rate in this herd was approximately 75%. On a third property in the district three out of three bulls with penile lesions tested positive for BHV-1 (serology). These bulls were joined to a heifer herd and the pregnancy rate was only 65%.

In the Albury district bulls on two properties were tested and all were negative for BHV-1 on serology.

For further information contact Steve Whittaker, SDV Hume LHPA, on (02) 6040 4210.

Metabolic disorders in southern NSW

Ketosis and hepatopathy caused losses in a mob of late-pregnant, crossbred ewes in the Narrandera district. The problem occurred when ewes went off their supplementary feed to chase green pick after rain. Thirty ewes died and a further 40 were affected out of 380.

Ketosis was confirmed in seven ewe flocks (and reported by many others) in the Albury district; other incidents were also confirmed at Tarcutta and The Rock. Declining nutrition and colder weather were complicating factors in most of these cases, but many incidents were triggered by recent feed disruption such as missed feeding and movement/handling stress from crutching.

Calcium deficiency caused light losses in ewes with lambs at foot in both the Albury and the Gundagai districts. Recent rain, colder nights and movement stresses were implicated as predisposing factors.

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

Anthrax exclusions

In Lachlan LHPA anthrax was excluded as the cause of sudden death in one out of 109 cattle by PCR test on a property with a previous history of anthrax.

Anthrax was also excluded as the cause of death in 30 out of 300 Merinos on another property.

In the Hume LHPA, anthrax was excluded as the cause of sudden death in two out of five young Suffolk rams in a district where anthrax had occurred

previously. A limited post mortem was performed and enterotoxaemia was suspected as the cause.

For further information contact Belinda Edmonstone, DV Lachlan LHPA, on (02) 6852 1688 or Sarah Robinson, NSW DPI, on (02) 6938 1967.

Cape tulip poisoning of cattle

Six cows died in a mob of 100 that were grazing a dense stand of cape tulip plants in a paddock near Tamworth. Many of the cows developed profuse diarrhoea, which turned mucoid. The cows quickly became recumbent, with mild respiratory difficulty. One animal progressed to lateral recumbency with nystagmus. Clinical pathology showed very high levels of muscle enzymes consistent with damage to the myocardium caused by the cardiac glycoside in cape tulip.

For further information contact Bob McKinnon, SDV Central North LHPA, Tamworth on (02) 6762 2900.

Amazing progress in footrot control

Footrot is a contagious bacterial disease of sheep and goats that is caused by the organism *Dichelobacter nodosus* in association with a number of other bacteria. With full expression, virulent footrot is a severe, debilitating disease that causes significant economic losses from reduced wool growth and quality, poor ewe fertility, poor growth rates, blowfly strike, and reduced value of sale sheep. In infected flocks there are also significant costs associated with the control of the disease.

After 20 years of the Footrot Strategic Plan, NSW is poised to reach a milestone that reflects the tremendous progress made with footrot eradication .

The flock prevalence of footrot in any Livestock Health and Pest Authority is now less than 1%, and any infected flocks are

under quarantine and subject to footrot eradication. Currently there are fewer than 20 flocks with footrot remaining in NSW, compared with over 6000 in 1990. The footrot program in NSW is widely recognised as the most successful disease control program ever undertaken by the sheep industry, and we can now claim that the State's sheep flocks are essentially free of this debilitating disease.



Footrot is getting rare. Photo courtesy of NSW DPI Image Library.

For further information contact John Seaman, NSW DPI, on (02) 6931 3248.

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 NSW DPI's Animal and Plant Biosecurity Branch in Orange on (02) 6391 3237 or fax (02) 6361 9976.

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

This is a report under the Animal Disease Surveillance Operational Plan, Project 3.1, 'Reporting for Animal Disease Status in NSW'.

Prepared by Rory Arthur, Animal and Plant Biosecurity Branch, Orange Agricultural Institute, Orange NSW 2800
Phone 02 6391 3823

E-mail: rory.arthur@dpi.nsw.gov.au

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

Disclaimer

The information contained in this publication is based on knowledge and understanding at the time of writing (June 2009). 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 New South Wales 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 NSW Department of Primary Industries over any equivalent product from another manufacturer.



NSW DEPARTMENT OF
PRIMARY INDUSTRIES

