

NEW SOUTH WALES

ANIMAL HEALTH SURVEILLANCE

July–September 2015 » Issue 2015/3

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Botulism outbreak in a small beef herd

The Mudgee District Veterinarian investigated a case where eight out of 19 unvaccinated 6-year-old non-lactating adult cattle died over 1 week. Two cows and one bull in the herd were lying down and unable to rise. During the property investigation, the District Vet noticed a sheep carcass (see photo) near a dam where the cattle drank.

The eight cows that had already died could not be necropsied. All had minimal or no signs of struggle before death, and there was no obvious external pathology.

Two of the recumbent cattle were on their sternums with the hindlimbs splayed out; the other animal was lying on its side and near death. The menace response (a blink reflex that occurs in response to an object made to rapidly approach the eye) was decreased in one animal that was tested and absent in one. There was no tongue protrusion, but there was moderate eye protrusion in all animals, giving a 'startled' appearance. This was presumably due to paralysis of the eye muscles and eyelids. All three affected cattle were euthanased because of their poor prognosis and for welfare considerations, and two of them were necropsied.

Both had full rumens: the contents were fibrous with little liquid. The large intestinal contents were 'dry', with small amounts of mucus, and fresh and digested blood, indicating decreased gastrointestinal motility and dehydration.

Samples for laboratory diagnosis were collected from the two cows. Bone, pieces of partly broken down spinal cord, and several maggots were collected from the sheep carcass.

At the State Diagnostic Veterinary Laboratory, fresh kidney samples from each necropsied animal tested negative for lead. Histopathological testing of kidney, liver and intestinal samples revealed nothing specific.

Both the sheep bone and the maggots on the carcass tested positive on an



This cow and seven others died without a struggle from botulism. Photo N. Gillan



The remains of a sheep carcass near the dam were the likely source of the botulism. Photo: N. Gillan

ELISA test for botulinum toxin antigen. A fresh liver sample from a necropsied cow also tested positive for botulinum toxin. This detection of botulinum toxin in both the sheep carcass and the cow liver sample allowed us to definitively diagnose botulism in this case, but note that sometimes this ELISA test has poor sensitivity and a diagnosis has to be made from the clinical signs.

This case highlighted some typical features of the clinical presentation of botulism in cattle, such as eye protrusion and constipation.

The cattle had probably been poisoned from chewing the bones of the dead sheep. After the herd was moved to another paddock and vaccinated there were no more losses.

For further information contact Nigel Gillan, District Veterinarian, Central Tablelands LLS, Mudgee, on (02) 6372 1866.

Another new strain of rabbit calicivirus

A new strain of rabbit calicivirus known as RHDV2 (rabbit haemorrhagic disease virus 2) was confirmed in a wild rabbit in the ACT in July this year. Since then, RHDV2 has been found in isolated locations around the ACT and NSW in pet rabbits, rabbits grown for meat, and wild rabbits.

RHDV2 differs from the variant strain of RHDV first detected in NSW last year and also from the endemic Czech strain of RHDV that has been present in Australia for the last 20 years. In contrast to the Czech strain, RHDV2

affects younger rabbits, including nestlings.

RHDV2 is most closely related to the European RHDV strain, which has spread successfully throughout Europe in recent years. A vaccine specifically against the RHDV2 strain is being developed in Europe, but it is currently not available. The current commercially available vaccine, when given according to the manufacturer's instructions, is believed to provide protection against RHDV2 in most rabbits. Regular booster vaccinations

should be given to domestic pet rabbits to ensure maximum possible protection against this variant.

Virologists at the Elizabeth Macarthur Agricultural Institute (EMAI) are working closely with the Invasive Animals Cooperative Research Centre to identify field strains so that the effects of RHDV2 on rabbit control in Australia can be determined.

For further information contact Sarah Britton, Veterinary Officer, Animal Biosecurity and Welfare Branch, Orange, on (02) 6391 3717.

Pneumonia in steers

This investigation is an example of public and private vets working together to achieve the diagnosis, treatment and management of a disease event. The initial investigation was done by the government vet, whereas ongoing treatment and management of affected stock were handled by the local veterinary practice.

Bacterial pneumonia was found to be the cause of death in four of 50 mixed-breed, 15-month-old steers near Inverell in northern NSW. The steers were grazing oats and were up to date with their five-in-one vaccinations.

The District Vet was presented with one dead steer, one dying one and one clinically affected one that had fever (39.4°C) and was weak and staggering.

The dying steer had a high fever (40.2°C) and was euthanased. The District Vet necropsied both it and the steer that had already died.

Both steers had acute pneumonia. Areas of reddening and haemorrhage were evident in the smaller ventral lung lobes (see photo).

Pneumonia was diagnosed from the gross pathology and the clinical signs, and the private vet was consulted for treatment options. Although he recommended the antibiotic tulathromycin the owners asked for an alternative, cheaper option (tetracycline) to treat the affected animal. It died overnight and the owner then identified a number of new cases.

The private vet visited the property the next morning. The dead animal was necropsied and clinically affected animals were treated with tulathromycin. One further death was recorded and four clinically affected steers were treated. Unaffected cattle were examined twice daily and moved to another paddock.

Samples processed at the State Veterinary Diagnostic Laboratory confirmed the diagnosis. Histopathology of the lung samples was consistent with *Histophilus* infection. Blood tests showed antibody titres for both pestivirus and bovine respiratory syncytial virus. Viral infection of the lungs along with secondary bacterial infection with *Histophilus* sp. is a common

presentation in feedlots and is a major cost to the industry. In this case, involving paddock grazing, the pathogenesis of disease was similar to that found in feedlots. However, in the absence of strategies such as using trained pen riders daily to identify clinical cases early, deaths occurred before the problem was identified because the stock were only periodically monitored. Graziers in this region have been advised to monitor their cattle more closely during the change of seasons, when it appears that this problem may emerge.

For further information contact Andrew Biddle, District Veterinarian, Northern Tablelands LLS, Inverell, on (02) 6720 8100.



Red and haemorrhagic lung tissue with an accumulation of clear interlobular fluid. Photo: A. Biddle

Water deprivation and sodium ion toxicoses in cattle

District Vets were called to investigate the deaths of cattle in a large, 400-hectare paddock in north-western NSW in August 2015. The mob of 270 seven- to 15-month-old Angus crossbred steers was grazing native pasture—mainly wild turnip, burr medic and galvanized burr. Some of the thick stands of turnip were up to 2 metres high. The cattle had been moved to the paddock 8 days before the deaths. They subsequently lost condition, became lethargic, and had a ‘hollowed out’ appearance.

The six dead cattle were spread throughout the paddock and were presumed to have died over a period of 48 hours since the last check by the caretaker. All had been recently vaccinated with five-in-one clostridial vaccines and drenched. The caretaker called the District Vet to report the initial deaths and then cleaned out the water trough. At this point, the cattle swarmed around the trough, drinking large amounts of water. Three more animals died soon after, near the trough, some showing signs of bleeding from the nose. The caretaker then let the cattle into the adjoining paddock where there was a dam that was fed by the same bore as the trough. The cattle gathered around the dam, several immersing themselves, and a further three animals died in the dam. By the



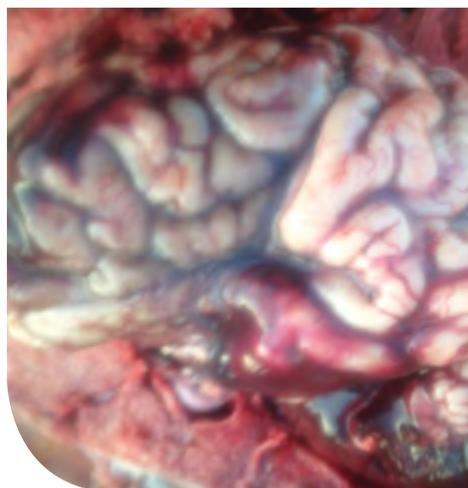
A dead steer with water and sodium ion intoxication, showing bleeding from the nose. Photo: M. Davies

time the District Vet arrived a total of 12 cattle had died.

The District Vet tested the dead cattle for anthrax with an ICT (immunochromatographic) test, but they were negative. A recumbent and severely affected steer was then euthanized and necropsied.

The necropsy revealed a swollen, friable liver that was easily broken with finger pressure and had a zonal pattern on its surface. The kidneys were dark purple, with no clear demarcation between the medulla and cortex. The mucosa of the abomasum was red and congested. The front half of the brain was grey on both sides, and the spinal canal contained a large amount of clear fluid with a jelly-like consistency. The steer was a ‘dark cutter’: in other words, instead of turning cherry red on exposure to air, its muscle tissues remained a dark red or purple.

Histopathological testing of samples sent to EMAI showed vacuolation (cavity formation) in the cortex of the brain, consistent with cerebral oedema (brain swelling). Testing of the aqueous humour (the jelly-like fluid in the eye) and serum from the euthanased steer revealed that both had very high sodium levels (180 and 158 millimoles/litre, respectively; normal range 135 to 152 millimoles/litre). Unfortunately, biochemical analysis of the blood



The front half of the brain (to the left) was grey on both sides. Photo: M. Davies

was not possible as the samples were severely haemolysed on arrival at the laboratory.

Water salinity testing showed that the salinity of the trough water was within recommended safe limits (400 microSiemens/centimetre; recommended value is less than 1600 microSiemens/centimetre).

The District Vet diagnosed ‘water and sodium ion intoxication’ on the basis of the combination of the high blood sodium levels and the brain swelling, along with the history of a sudden intake of large volumes of water.

This condition occurs after a period of water deprivation causes a build-up of extracellular sodium and movement of fluid out of the central nervous system. A sudden influx of water causes fluid to move rapidly back into the central nervous system, causing cerebral oedema. The sudden influx of fluid also causes red cells to burst, causing haemolysis in the blood vessels; this explained the nose bleeds and the haemolysed condition of the blood samples on arrival at the laboratory.

Further discussion with the caretaker of these cattle revealed that they had not been shown the location of the water trough when they were first moved into the paddock. Owing to the paddock’s large size, and the large volume and height of vegetation present, it is likely that the cattle got lost in the paddock and couldn’t find the single water source. Consequently, the early deaths were likely due to water deprivation, whereas the later deaths were a result of sudden overhydration after the water source was discovered by the cattle.

The surviving cattle remained in the paddock with the dam, where they recovered slowly over the next week, with a further two deaths. A total of 15 cattle died during this episode.

For further information contact Megan Davies, District Veterinarian, North West LLS, Narrabri, on (02) 6790 7600.

Prolonged lead residues in cattle over a 21-month period

In 2014, twelve of a mob of 1100 weaned calves running on a 1.5-kilometre-wide and 100-kilometre-long travelling stock reserve in southern NSW became blind, uncoordinated and died. The attending vet found fragments of lead in the rumen of one animal, and assays showed lead levels of 9.2 micromoles/litre (normal, less than 0.20) in whole blood and 276 micromoles/litre (normal, less than 2) in the kidney.

The source of the lead was not discovered, but the cattle were removed to the owner's property. Blood samples from every animal were tested for lead: eight cattle were found to have lead levels higher than the maximum residue limit (MRL, 0.24 micromoles/litre) and were not allowed to enter the human food chain.

The affected animals were detained and tested again 12 months later. Three of the cattle still had lead levels in excess of the MRL.

Nine months later the cattle were tested again, and one still had blood lead levels in excess of the MRL and another two had

detectable levels. The owner chose to have the steer with the blood level greater than the MRL destroyed and its carcass buried. At necropsy, four visible fragments of lead (1–2 x 1–2 x 3–4 millimetres) were seen among the villi (i.e. the finger-like projections) of the lining of the rumen.

Although the initial lead level of the animal with persistent lead levels was quite high, the steer had grown

significantly and was probably twice the weight that it was when it first ingested the lead (i.e. it had grown from 350 to 700 kilograms estimated body weight). The persistent presence of lead in the rumen was a factor in the high levels of lead in that animal.

For further information contact Dan Salmon, Team Leader Murray LLS, Deniliquin, on (03) 5881 1055.



Lead fragments in the rumen of the affected steer. Photo: D. Salmon

Humpyback near Forbes NSW

Humpyback is a locomotor problem that usually occurs in older sheep. (See *Animal Health Surveillance* issue 2015/2 for another case report.) This case involved what the owner described as 'the best' young ram lambs on a Merino stud in Central West NSW where the condition had not been previously reported.

The owner reported that 30% of a mob of 300 ram lambs were 'stiff-legged', with a 'dropped tail set'. A similar syndrome of low prevalence had occurred sporadically previously and was thought to be arthritis from bacterial infection with *Chlamydomphila pecorum*.

A private vet and the District Vet examined five affected ram lambs. Each had a hunched lower back, with a shortened step in the hindlimbs, and became tired after being moved around a small paddock. When pressured, the most severely affected lambs knuckled over in the hindlimbs and 'dog sat' before

collapsing and lying down upright on their sternums. A badly affected lamb was euthanased. Necropsy showed some mild muscle bruising in the spinal muscles, but no other significant lesions.

At the laboratory, blood samples were negative for *Chlamydia* antibodies. Histopathology revealed mild Wallerian degeneration (a type of disintegration of the nerve axons) of the spinal cord white matter and nerve roots. There was also mild to moderate subacute, multifocal necrosis of the lumbar muscles along the spine associated with a minor increase in levels of the enzyme creatine kinase (383 to 1284 microlitres/litre) in all five blood samples (normal, less than 300 microlitres/litre). Selenium levels in a liver sample were normal, and this excluded white muscle disease.

The plant that has previously been implicated as a possible cause of the disease—'quena', or 'wild tomato' (*Solanum esuriale*)—was found in

reasonable-sized patches, and there was evidence that it had been eaten, despite the fact that the sheep were in a lucerne paddock and had ready access to oaten hay and mineral supplements.

The owner had observed that a disproportionate number of the best ram lambs were severely affected. Because the syndrome is exacerbated by activity, it is possible that the bigger-framed and heavier-woolled rams were under greater heat and exertional stress and therefore showed signs first.

One month on from the ram lambs being moved to 'clean' paddocks and off the quena, they were showing a dramatic improvement.

For further information contact Simon Pain, Lachlan Valley Veterinary Clinic, Forbes, on (02) 6851 1100, and Belinda Edmonstone, District Veterinarian, Central West LLS, Forbes, on (02) 6850 1614.

Eastern grey kangaroo deaths from starvation

During the quarter, a number of agencies received reports from rangers and wildlife carers in NSW and the ACT of more than 300 unwell and dead juvenile eastern grey kangaroos (*Macropus giganteus*). Affected kangaroos were from across a large geographical area around Canberra.

The majority of affected kangaroos were sub-adult juveniles (up to 15 kilograms, 18 to 21 months old). They were thin and weak, with poor coat condition and pale mucous membranes. Individuals brought into rehabilitation died despite supportive care.

Gross post mortem and histological examinations revealed severe and chronic emaciation and variable gastrointestinal parasite burdens. There was no evidence of babesiosis, *Phalaris* toxicity, or toxoplasmosis.

The deaths were caused by starvation from restricted food availability. Contributing factors were overpopulation, cold stress and parasitic burdens. The feed shortage was due to low temperatures and low rainfall over winter. The detrimental health effects of the common gastrointestinal parasites

normally found in kangaroos are exacerbated when food is limited.

Similar seasonal deaths of sub-adult eastern grey kangaroos have been previously observed during the winter months over the past 20 years in the ACT, Victoria and NSW. The deaths have a natural population regulatory effect and serve to protect the population when food resources are restricted.

For further information contact Tiggy Grillo, National Coordinator, Wildlife Health Australia, Sydney, on (02) 9960 7444.

Better field pathologists from training by world experts

Ten NSW Government vets from LLS and NSW DPI have almost completed a high-level training course in the gross pathology of production animals, led by Emeritus Professor Peter Windsor from the University of Sydney and Dr John Glastonbury from Charles Sturt University, with Helen Peam from EMAI and Bruce Watt from LLS assisting.

Participants have been required to achieve excellence in gross necropsy, specimen collection, disease outbreak investigation and disease pathogenesis. The course content was delivered with a combination of case presentations, image modules, hands-on gross pathology and histopathology. One outcome will be more informative gross pathology descriptions in this very newsletter!

Participants were presented with a variety of case material, images and slides and were asked in each case to:

- describe the lesion
- provide a morphologic diagnosis
- provide differential diagnoses and pathogenesis for the cause
- discuss potential other investigations, including laboratory tests (if applicable)

An example is the image of a bovine liver in the box at right. Can you carry out the tasks above?

For further information contact Geoff Campbell, Veterinary Officer, NSW DPI, Orange, on (02) 6391 3534.

Quiz: Can you describe the pathology of this bovine liver?

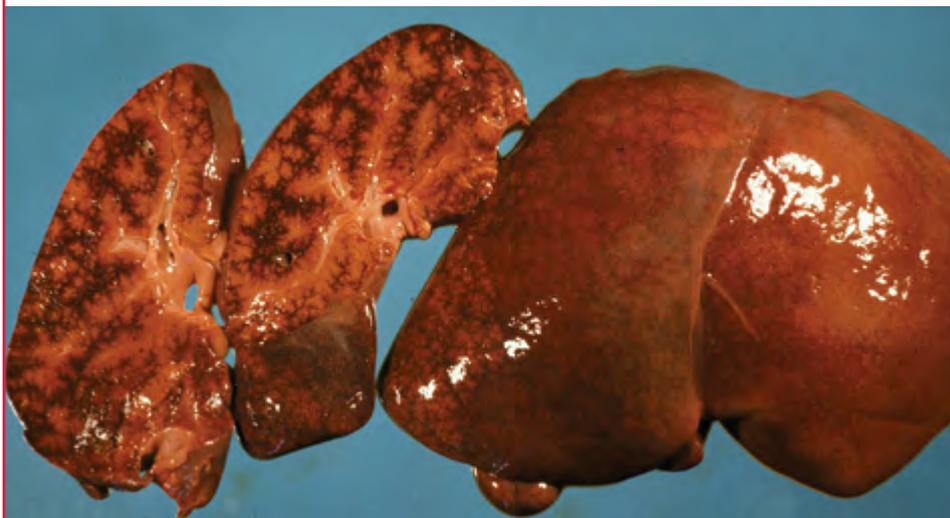


Image kindly provided by the CVO, Victorian Department of Environment, Land, Water & Planning

Lesion description: Cut section of liver with white arborized (tree-branch-like) tissue interspersed throughout dark red hepatic tissue (or 'nutmeg' pattern)

Morphological diagnosis: Severe extensive chronic passive hepatic congestion (nutmeg liver)

Differential diagnoses/pathogenesis: Cardiovascular cause (e.g. cardiotoxic plant, or 'hardware' disease from eating metal objects). Right-sided heart failure leads to increased pressure in the caudal

vena cava, which leads to hepatic vein congestion, which in turn causes the formation of dark red, congested sinusoid regions (irregularly-shaped blood vessels), along with fatty degenerate hepatocytes (liver cells) and fibrosis.

Ancillary investigations, including laboratory tests: Post-mortem examination of the thorax and heart to investigate structural cardiac disease. Assess history and pasture regarding toxic causes.

Sudden cattle death caused by bloat

The owner of a mob of 150 Angus heifers on the Central Tablelands lost three of them in late September 2015. One died before the mob was mustered and handled, and two died after being returned to the paddock. The paddock was not considered a high risk for bloat, consisting of about 2500 kilograms/hectare of 75% grass (cocksfoot, prairie grass, and phalaris) and 25% sub clover. The heifers had been vaccinated with five-in-one at marking in mid-September 2014 and given booster vaccinations 6 weeks later at weaning in February 2015. They were vaccinated again with five-in-one (and Pestigard® vaccine for bovine viral diarrhoea virus) in early June 2015.

Bloat was diagnosed in two of the heifers that were necropsied: the front part of the carcass was very congested and the back part blanched. There was haemorrhage in the conjunctiva of the eye and in the ethmoid turbinates of the nasal sinuses, and a 'bloat line' could be seen on the oesophagus at the level of the first rib. One heifer had marked subcutaneous emphysema (air under the

skin), possibly from air escaping from the lungs because of very high intrathoracic pressure from the bloated rumen or possibly from post mortem autolysis.

The producer did not anticipate bloat on this pasture, as it had been grazed and was grass dominant. However, the second and third heifers affected had been yarded and held off feed for several hours before being introduced to the pasture. The owner was advised to avoid introducing

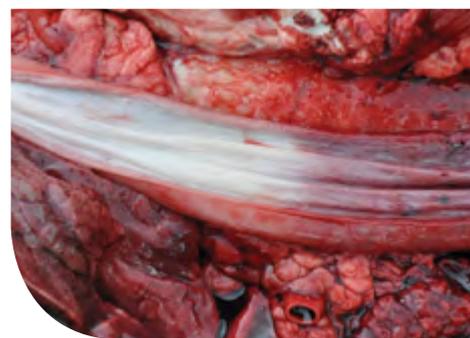


Carcass of a bloated cow, showing subcutaneous emphysema (air under the skin) Photo: B. Watt

hungry cattle to highly palatable pastures (even if they were grass dominant).

Bloat is an important endemic disease costing southern Australian beef producers almost \$80 million dollars a year. Its diagnosis is also important to exclude other causes of sudden death in cattle, such as anthrax.

For further information contact Bruce Watt, Team Leader Central Tablelands, Bathurst on (02) 6331 1377.



'Bloat line', showing congestion at the front of the carcass, haemorrhage of the oesophagus (which has been opened lengthwise) and the trachea at the level of the first rib, and blanching towards the back of the carcass. Photo: B. Watt

Bellinger River snapping turtle deaths: a new virus?

In February 2015, local kayakers reported a severe event of deaths in Bellinger River snapping turtles (*Myuchelys georgesi*) in the Bellinger River in northern NSW. The turtles were described as slow moving and blind.

River levels had been lower than normal in the lead-up to the event, with extremely low water levels before Christmas 2014 and a severe heat episode in early December 2014, as well as elevated water temperatures. Two minor floods had occurred—one in late December 2014 and one in late January 2015.

An estimated 432 snapping turtles were observed to be dead or dying on or after 14 February 2015. Affected turtles had swollen eyes and were emaciated. Many had a slight, clear nasal discharge, and some animals had hindlimb weakness. At necropsy, the animals were thin. Both eyelids were swollen and there was anterior uveitis (inflammation of the pigmented layer inside the eye). There

were also tan-coloured patches on the skin and ventral thighs. Fibrin plaques were observed in the vitreous humour (the clear gel between the eye's lens and retina). There was haemorrhage into the eye, with inflammation extending into the tissues around the eye and possibly into the sinuses. The turtles rely on sight for feeding and therefore were severely affected by the onset of blindness.

A novel virus has recently (July 2015) been detected in the tissues of affected turtles. Extensive testing has shown very high levels of the virus in the tissues in which the most severe lesions were observed, suggesting a major role for this virus. Further work is being done to characterise the virus and work out its significance in the pathogenesis of this disease.

Because of the limited distribution of this turtle, as well as the large numbers affected (at least 25% of the known population) and the case death rate

of up to 100%, the NSW Scientific Committee has recently made a Preliminary Determination to list the species as Critically Endangered under the *Threatened Species Conservation Act 1995*.

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



An affected Bellinger River snapping turtle. Photo: Karrie Rose

Getting information on animal diseases

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

For statewide information, contact the Department of Primary Industries Animal and Plant Biosecurity Branch in Orange on (02) 6391 3237 or fax (02) 6361 9976.

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

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Published by NSW Department of Primary Industries, a part of NSW Department of Industry, Skills and Regional Development

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

www.dpi.nsw.gov.au
www.ils.nsw.gov.au

Disclaimer

The information contained in this publication is based on knowledge and understanding at the time of writing (October 2015). However, because of advances in knowledge, users are reminded of the need to ensure that the information upon which they rely is up to date and to check the currency of the information with the appropriate officer of NSW Department of Industry, Skills and Regional Development or the user's independent adviser.

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